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Epilepsy in Lagos, Nigeria

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Summary. An epidemiological study of epilepsy in Nigeria is presented. The incidence of this symptom-complex is assessed at between 8 and 13 per 1000.

A male preponderance is noted in the series of 117 patients studied and in other African series. It is likely that this is the result of unnatural selection since African men seem to seek medical attention more frequently than their women folk. Two peaks of age of onset are observed in the 13 months to 5 years and 11–20 years age groups.

The late presentation at orthodox medical centres reflects the strong belief of evil spirit or 'juju' as the cause of epilepsy and accordingly it is usually believed that the unorthodox medical practitioners ('native doctors') are best able to treat epilepsy.

A family history of epilepsy is present in 12·8% of the 117 cases. Among the centrencephalic cases 16·1% had a family history of epilepsy whilst such a history was present in 9·1% of the symptomatic cases.

Of the clinical types of centrencephalic epilepsies encountered in the sixty-two patients, grand mal accounted for sixty cases (51·2%). There was only one case of petit mal and one case of infantile spasm. Of the fifty-five cases of symptomatic epilepsy seventeen patients (30·9%) had focal (excluding temporal lobe) epilepsy: there were six patients (10·9%) with Jacksonian epilepsy, eleven (20%) with temporal lobe epilepsy, eleven (20%) with hemiconvulsion–hemiplegia–epilepsy syndrome and ten (18·1%) with secondary 'grand mal' epilepsies. The symptomatic cases accounted for 47% of the total series of 117 patients.

It was possible to arrive at a diagnosis in thirty-three of the fifty-five cases (60% of symptomatic epilepsies.

Extensive investigation into the role played by parasitic disease in the pathogenesis of this series of epileptic patients failed to produce a single case of parasitic epilepsy. The higher incidence of positive toxocara skin test in a number of the patients tested is noted but its significance is still unknown.

Résumé. Il s'agit d'une étude épidémiologique de l'épilepsie au Nigéria. La fréquence de ce complexe de symptomes est estimée entre 8 et 13 pour mille. On remarque que les hommes prédominent dans la série de 117 patients étudiés aussi bien que dans d'autres séries africaines. Il est probable que la prédominance des hommes résulte d'une sélection arbitraire, puisque parmi les Africains les hommes semblent consulter les services hospitalier plus souvent que les femmes.

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On a observé deux sommets quant à l'âge de début de la maladie. L'un se trouve entre 13 mois et 5 ans et l'autre entre 11 et 20 ans.

La consultation tardive des services hospitaliers orthodoxes s'explique par la croyance répandue aux mauvais esprits ou au 'grigri' comme cause de l'épilepsie. Donc selon l'opinion populaire ce sont les médecins non-orthodoxes qui sauraient le mieux soigner les épileptiques.

On a constaté une histoire d'épilepsie dans la famille de 12,8% des 117 cas. Parmi les cas centrencéphaliques 16,1% avaient une histoire d'épilepsie dans la famille tandis que cette histoire était présente dans 9,1% des cas symptomatiques.

Parmi les types cliniques d'épilepsies centrencéphaliques recontrés chez soixante-deux patients, l'épilepsie Grand Mal se manifestait dans soixante cas (51,2% de la série totale). Il n'y avait qu'un seul cas de spasme infantile. Parmi les cinquante-cinq cas d'épilepsie symptomatique, dix-sept patients (30,9%) souffraient de l'épilepsie focale (à l'exclusion de l'épilepsie temporale). Il y avait six patients (10,9%) présentant l'épilepsie jacksonienne, onze patients (20%) l'épilepsie temporale, onze patients (20%) le syndrome de l'épilepsie hémiconvulsive-hémiplégique et dix patients (18,1%) le Grand Mal secondaire. Quarante-sept pour cent de la série totale de 117 patients étaient des cas symptomatiques.

Dans trente-trois des cinquante-cinq cas (60%) d'épilepsies symptomatiques, il a été impossible d'arriver à un diagnostic.

Une enquête soigneuse sur le rôle joué par les maladies parasitiques dans la pathogénie de cette série de patients épileptiques n'a pu démontrer aucun cas d'épilepsie parasitique. Une fréquence plus grande de cuté-réaction positive au toxocara chez un nombre de patients examinés a attriré notre attention sans que nous en ayons pu expliquer la signification jusqu'à présent.

Epilepsy is one of the most common neurological conditions encountered in Nigeria. This symptom-complex forms about 10% of all neurological admissions to adult medical wards at the Lagos University Teaching Hospital (Table 1).

TABLE 1. The Nigerian neurological profile, Lagos University Teaching Hospital, 1962–67

Total adult neurological admissions	1220	
Tetanus	450	(36·0%)
Cerebrovascular accidents*	205	(16·8%)
Epilepsy	124	(10·2%)
Paraplegia	109	(9·0%)
Meningitis	102	(8·3%)
Polyneuropathy	51	(4·1%)
Tropical neuropathic syndrome	39	(3·2%)
Parkinson's syndrome	20	(1·6%)
Neurosyphilis	15	(1·2%)
Encephalopathies	15	(1·2%)
Poliomyelitis	8	(0·6%)

* Excluding trauma and venous sinus thrombosis.

INCIDENCE OF EPILEPSY IN LAGOS

The determination of true incidence of any disease considered as a social slur or insanity is usually difficult. The incidence of epilepsy in Nigeria has not been determined. Since the incidence of any condition or disorder derived from clinic or hospital records is unreliable because of pre-selection of the sample, it was decided to include a study of the incidence of epilepsy.

Methodology

The Department of Community Health, College of Medicine, University of Lagos, maintains a sample of some 14 000 persons, or approximately 1% of the urban population, located in thirty blocks of 400–500 persons each spaced throughout the metropolitan area in accordance with population density, as revealed in the 1963 census. A random sample from five of these blocks selected at random was investigated for their knowledge of and attitude to epilepsy. The total number of respondents in this 'attitude sample' was 381. In view of the extensive nature of the enquiry on the attitude to epilepsy, this group was merely asked if any member of the family or household suffered from epilepsy, the names and ages of the epileptic persons and the relationship to the respondent. To go into details of the size of the family and household would have considerably lengthened the time of a difficult interview on a delicate subject.

Accordingly, all the remaining twenty-five blocks in the urban Lagos sample area were interviewed with a separate questionnaire designed to assess the incidence of epilepsy. About 15 adult persons were randomly selected from each block, making a total of 381 respondents for this incidence survey. The questionnaire was designed to determine the total number of persons (alive or dead) in the *immediate* family and in the household—including all the children and adults. They were then asked whether any member of the immediate family and/or household suffered from epilepsy. Other questions covered the treatment received by the epileptic person (none, unorthodox 'native' orthodox or both) and whether the epileptic patient was still alive or dead.

Precautions taken to ensure reliability of the answers included the following:

1. That the interview was conducted by trained and experienced interviewers. Each interviewer trained at the Department of Community Health, College of Medicine, University of Lagos, was assigned to an area in each block for all epidemiological studies conducted by the College of Medicine. Thus, the interviewer was fully conversant with his or her area and the residents knew him or her well. This ensured that the respondents were less likely to be unco-operative with such an interviewer dealing with as delicate a probe as this.

2. That the respondents knew what was meant by 'epilepsy'. No confusion with other convulsive disorders was likely, since they were all recognized by different names.

3. That what was meant by *immediate* family—comprised father, mother and the full siblings (brothers and sisters) whether they were alive or dead. Nigerians tend to be generous with the embodiment of many unrelated persons into their family, this may occasionally include long-stay lodgers, servants and servants' children let alone those who get incorporated into the 'family' by sheer proximity of residence of themselves or their parents in days gone by. The existence of widely practised polygamy complicates matters much further. Where the respondent was an unmarried male or female, he or she was asked about the total number comprising father, mother and full siblings, alive or dead. A married male respondent was

asked to enumerate the number of his wives and the number of children borne to him by only these wives, alive or dead, since he may have acquired some of the wives by 'inheritance' or by marrying a widow or a previously unmarried mother who would add their own children to the family pool. Similarly, a married woman was asked to enumerate her current husband and all the children she had borne for him—alive or dead.

4. Members of the household were taken to mean all residents in the household, whether they were related or not.

5. That the respondents must be adults—as in the attitude survey—to ensure correct information on the questions.

TABLE 2. Incidence survey

Area code	Total no. of family of respondents	No. of epileptic persons in area	Total no. persons in household	No. of epileptic persons in household
01	68	1	43	1
02	95	1	37	1
04	128	0	51	0
05	134	0	65	0
06	121	0	41	0
07	135	0	87	0
08	113	1	81	0
10	135	0	73	0
11	117	0	61	0
12	86	0	58	0
13	97	0	87	0
14	91	1	72	1
16	63	1	33	0
17	64	0	30	0
18	106	0	97	0
19	107	1	85	1
21	96	0	38	0
22	106	0	82	0
23	91	0	63	0
24	106	0	95	0
25	84	0	41	0
26	114	1	55	0
28	119	1	89	1
29	110	0	61	0
30	106	0	72	0
Total no. of areas = 25	2592	8	1597	5

Incidence = 3·08 per 1000.

Results. Table 2 shows the results of the Incidence Survey in twenty-five 'blocks' of urban population of Lagos.

The total number of randomly selected respondents in the twenty-five blocks were 381 who had an *immediate family* numbering 2592 persons. There were eight persons with

epilepsy in this group of 2592 immediate family members, giving an incidence of 0·31% or 3·1 per 1000. This figure may not necessarily represent the true incidence of epilepsy in urban Lagos.

In view of the social slur and psychological complex surrounding epilepsy, it is highly probable that a number of cases were concealed. Although the types of epilepsy were not identified either by the respondents or the non-medical interviewers, but it is very likely that some of the eight persons might have suffered from centrencephalic epilepsy. According to the questionnaire, four of these eight persons started having these attacks when young and these four were still alive—whilst the other four persons started having their attacks in adult life and all these four were now dead. It is probable that the four early-onset cases of epilepsy were centrencephalic in type which may well have been subjected to some genetic influences. The present review of 117 cases of epilepsy showed the presence of family history in 12·8% of the cases, being 16·1% for centrencephalic epilepsy (sixty-two cases) and 9·1% for the symptomatic series (fifty-five cases). If this genetic influence is assumed to operate on these four patients, it may account for more cases of epilepsy in their families (average immediate family size being eight persons) than revealed in the survey.

Febrile infantile convulsion is not as benign as formerly thought, but in fact it may be responsible for a few other cases of epilepsy in the survey since febrile convulsion is particularly rife. Other potent precipitating factors in the genesis of epilepsy are birth trauma and central nervous system infections which are common locally. It may well be that a number of the persons affected by these factors never live long enough to develop epilepsy. The role played by parasitic diseases and lower standard of health needs to be considered as well.

Furthermore, certain non-convulsive forms of epilepsy may be unrecognized as epilepsy, for example psychomotor epilepsy or 'epileptic equivalents'. Some attacks may be so brief as to escape observation and detection by the lay population, for example petit mal. This is one of the limitations of a questionnaire survey since the lay population would only recognize the convulsive forms of epilepsy.

In all the twenty-five blocks sampled, with a total number of 1597 people in the 381 households, there were five epileptic persons encountered, these being members of the immediate family of the households. The other three epileptics were not living with their immediate family. No unrelated person with epilepsy was discovered in any of the 381 households. This is not surprising since any lodger who has epilepsy is ejected from the household and completely ostracized because epilepsy is thought to be infectious. Known epileptics do not find it easy to get accommodation.

An approximate incidence of epilepsy was also determined from among the 381 respondents in the five 'blocks' in urban population of Lagos who were examined for their attitude towards epilepsy. To the question, 'Does anyone in your family or household suffer from epilepsy?' ten respondents (2·6%) answered in the affirmative.

The average household size in Lagos is about 3·3 persons. All the 381 respondents answered this question. The total number of people in the 381 households is about 1257.

Assuming that the epileptic patient mentioned was not listed by more than one respondent in the enquiry, then the expected incidence of epilepsy in Lagos will be about 8 per 1000. This figure is probably closer to the lower level of the true incidence which is likely to be about 13–15 per 1000 when we take into consideration the factors discussed above, namely, concealment of epileptic cases, failure to recognize forms of epilepsy other than the tonic-clonic types.

Comment

The incidence of epilepsy in different parts of Africa shows some remarkable similarity. Piraux (1960), in a medical census of a community of 15 000 Congolese people, found an incidence of 0·4%. Smartt (1959) found an incidence of 0·013% of the population in Central Province of Tanganyika from the attendance records of all the epileptic patients in Government and Mission Hospitals and Native Dispensaries. Similarly, Hurst, Reef & Sachs (1961) showed an incidence of 0·09% among the clinic attendances in the Meadowlands Township, near Johannesburg. For many reasons, few African epileptic patients ever get to an orthodox medical centre for treatment. Epilepsy is held to be due to evil spirits or the effect of black magic ('juju') (Levy *et al.*, 1964; Dada, 1968) and accordingly only the unorthodox medicine man is capable of unravelling its cause and treating it, as pointed out by Smartt (1959) himself. 'Fits affecting women and children seem to be regarded by patient and parents alike as a misfortune to be accepted and there must be very large numbers of rural sufferers who have never sought medical help' (Levy *et al.*, 1964).

Bird, Heinz & Klintworth (1962) found an incidence of 3·67 per 1000 among adult Bantu male mine-workers, aged between 18 and 55 years. The selection of this special group of people indicates that the incidence among the Bantu is probably higher than this figure, when women, children and all the men are taken into consideration. Levy *et al.* (1964), on the other hand, discovered an incidence of 0·74% from among the Semokwe reserve, the majority of whom were women and children, the men having migrated to the townships for their employment. Thus, this figure might well be higher if the men were included. Pernot, Tridan & Penquin (1961) quoted Pequinot & Rosch who found an incidence of 3·4 per 1000 among male North Africans attending the hospital as in- and out-patients and 4·9 per 1000 of hospitalized males. Haddock (1967) in an inquiry on in-patients and out-patients 'about their personal and family history of epilepsy during their examination and treatment in Korle Bu Hospital' Accra, Ghana, found an incidence of 0·46% in the males, 0·2% in the females and an overall incidence of 0·33%. There are a number of factors which detract from the real value of these figures, for example the selection is somewhat ambiguous. Thus, epileptic patients with 'only fits occurring in the patients themselves and their full siblings were used in estimating the incidence of epilepsy', but excluded from the study were infantile convulsions and 'anybody seen whose attendance appeared to have anything to do with epilepsy'. The determination of the incidence of epilepsy from among the patients attending 'a general medical unit with a bias towards neurology' and in a teaching hospital at that cannot be said to be representative of the community.

Questionnaire inquiry surveys in Africa produce low and incorrect figures in view of the public attitude to epilepsy, and unless public education and understanding of epilepsy is improved, we may have to 'collaborate' with the 'medicine' men (native doctors) who see a large number of epileptics. Apart from Levy's figures of 0·74% (as mentioned earlier, this figure would be higher if the men were included), the incidence found by various workers in Africa seemed to lie between 3 and 4 per 1000. This must be regarded as the lowest level of incidence and it appeared that the same degree of concealment probably exists in many parts of Africa. The incidence of epilepsy in Africa may be between 3 per 1000 (lower limit) and probably more than 7·4 per 1000 (upper limit). 'From what is known of aetiology, it is probably that the numbers would be higher in under-developed countries with frequent childhood infections involving the brain and less adequate obstetric services' (W.H.O., 1957).

TABLE 3. Studies on incidence of epilepsy: methods and results

Method	Investigator	Population sampled	Incidence per 1000	Remarks
Questionnaire inquiry	Stein (1933)	Relatives of hospital staff, U.S.A.	6·6	May not be representative
	Lennox (1960)	Medical students, nurses, non-epileptic patients, U.S.A.	7·0	May not be representative
	Haddock (1968)	In- and out-patients, Accra, Ghana	3·3	Very selective sample
	Levy *et al.* (1964)	General population—Semokwe Reserve, Rhodesia	7·4	Not representative—majority women and children
	Dada (1968)	General population, Lagos	3·1	Small urban sample
Hospital/clinic records	Kurland (1959)	Mayo Clinic patients, Rochester	2·98	May not be representative
	Pond *et al.* (1960)	Patients in fourteen General Practices, S.E. England	6·2	Large sample—no bias
Rejection/discharge rates in occupation	Edwards *et al.* (1943)	Registering draftees, U.S.A.	3·7	Not representative
	Bailey *et al.* (1929)	Draftee rejection, U.S.A.	5·15	Not representative
	Bird (1964)	Mine workers—Rejection and discharge, S. Africa	3·67	Not representative

EPIDEMIOLOGY OF EPILEPSY IN LAGOS AS REVEALED BY A STUDY OF 117 PATIENTS

These patients were seen at the Lagos University Teaching Hospital (L.U.T.H.) from 1962. A number of them were studied by my colleagues but the majority of them were under my care from 1965. The main criteria for inclusion in this review were as follows:

1. Each patient should have had more than one attack of convulsive disorder presumed to be epileptic. Isolated fits were excluded.
2. A good history should have been recorded from the patient's relatives.
3. Adequate record of the findings of both the neurological and general examinations should be available.
4. Certain basic investigations should have been carried out. These were haemogram and urine analysis, blood sugar, serum calcium, liver function tests, serological tests for syphilis, chest and skull radiographs, cerebrospinal fluid examination; electro-encephalographic investigations were only carried out on the patients in the past one year. Specialized neuro-radiological investigations were not generally available until 1965 and then these were carried out when a radiologist with a bias towards neuroradiology was available. The lack of a neuro-surgeon in Lagos meant that a number of these patients had to be sent to another centre not only for neuro-surgical treatment but also for more definitive investigations carried out by the neuro-surgeon.

Thus, there was a large element of selection in this series and any conclusion drawn must necessarily take this into consideration.

In order to obtain all the required data from the patients' case records and to be able to subject the data to computer analysis, a questionnaire was drawn up to cover various aspects of history, clinical presentation, investigations, diagnosis and treatment, including prognosis.

Results

Sex incidence

The sex distribution of the 117 patients is given in Table 4.

TABLE 4. Sex distribution in 117 cases of epilepsy

	Male	Female
Both groups (117 cases)	69 (59·0%)	48 (41·0%)
Centrencephalic group (62 cases)	41 (66·1%)	21 (33·9%)
Symptomatic group (55 cases)	28 (50·9%)	27 (49·1%)

Region of origin

Most of the patients (eighty-seven cases, 74·4%) were from Lagos and the Western regions. There were seventeen patients (14·5%) from the Eastern region, ten (8·5%) from the Mid-Western region and three (2·6%) from the North.

Age of onset

One hundred and two of the 117 patients (95·7%) had their attacks before the age of 40

years. Forty-three cases (45·2%) had their first attack in the first decade of life but about half of these were aged between 13 months and 5 years. There were thirty cases (25·7%) in the second decade, sixteen (13·7%) in the third decade, thirteen (11·1%) in the fourth decade, four (3·4%) in the fifth decade and only one patient (0·9%) in the sixth decade. Thus 102 patients (95·7%) had their first attacks before the age of 40 years.

Age and sex distribution, 117 cases

Although there was an element of selection in these cases, the age and sex distribution was studied with a view to comparing this with the age and sex distribution in the general population (Table 5).

TABLE 5. Age and sex distribution (117 cases compared with the general population*)

Age of onset	117 cases		General population	
	Total no. of cases	Male/female ratio	Total no. of sample	Male/female ratio
Up to 1 year	10	2·3 : 1	2265	1·05 : 1
13 months to 5 years	28	1·5 : 1	2139	0·98 : 1
6–10 years	15	0·6 : 1	1658	0·96 : 1
11–20 years	30	1·5 : 1	2506	1 : 1
21–30 years	16	2·2 : 1	2748	1·3 : 1
Over 30 years	18	1·3 : 1	3400	1·2 : 1
Total—all ages	117	1·4 : 1	12451	1·1 : 1

* As compiled in the demographic data supplied by the Department of Community Health, College of Medicine, University of Lagos, Medical Statistics. The present series were selected as described above. Accordingly, it was not possible to determine whether the preponderance of one sex over the other was the result of this selection or not.

COMMENTS ON THE CLINICAL PRESENTATION OF EPILEPSIES

Sex distribution of epileptic patients

The present series of 117 patients was very selective. If consecutive cases had been studied, it would be possible to determine whether the epilepsies show some peculiar sex distribution or not. However, in this series, there was a male preponderance (59%) in the total sample of 117 patients. In the centrencephalic group this male incidence was exactly twice (66·1%) that of the female. The symptomatic group of epilepsies appeared to be evenly distributed among the two sexes (50·9% male and 49·1% female).

In an earlier paper, Dada & Odeku (1966) observed a male incidence of 64·1% in a group of 234 epileptic patients (combined centrencephalic and symptomatic epilepsies). Of the 167 centrencephalic epilepsies, the male incidence was 61·6% whilst in the symptomatic group of sixty-seven patients it was 70·0%. This series of 234 patients suffered the same draw-back of selection.

Gelfand (1957a) could not assess the sex distribution in his series of seventy-five patients seen 'in and around Salisbury' which had a predominant male population of workers. Piraux (1960) quoted a population survey in Usumbura of about 15 000 people among whom

were sixty-eight known epileptics, forty-five male and twenty-three female. He agreed that his series of 209 Central African patients (151 male and 58 female) was probably artificially weighted as the women were unable to remain in hospital long enough for all the studies to be carried out due to family responsibilities. This paper by Piraux was similar to our present series of 117 patients in that similar criteria for selection were used in both studies. Levy *et al.* (1964) also observed 'marked sex difference' in their 100 epileptic patients 'who appeared spontaneously at the Harare hospital'—eighty-eight of them being males compared with

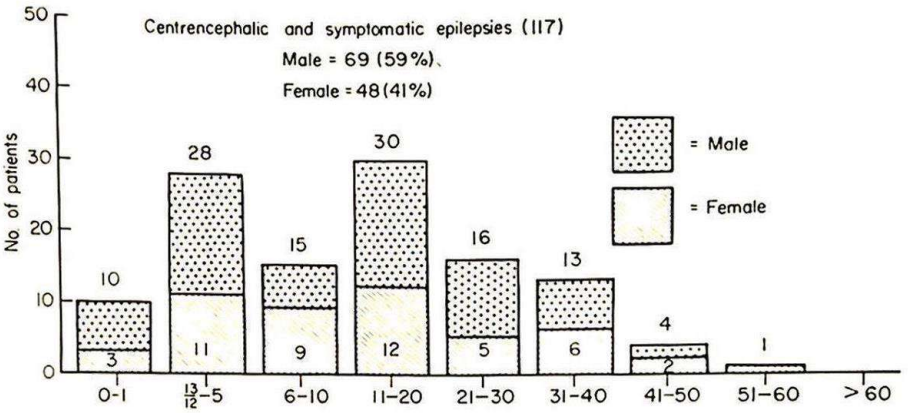

FIG. 1. Age of onset and sex incidence in 117 epileptic patients.

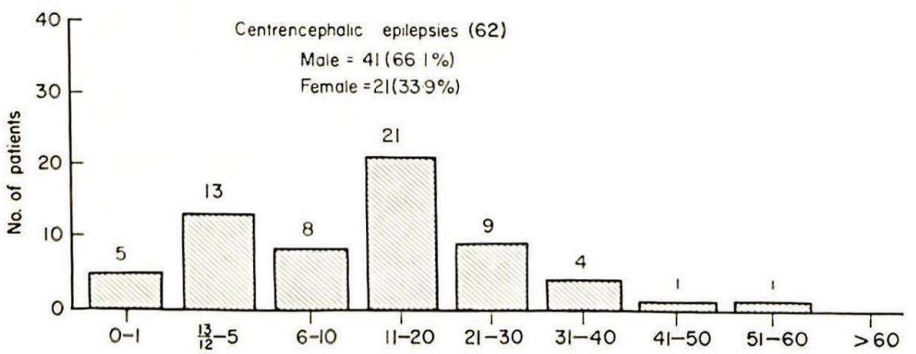

FIG. 2. Age of onset and sex incidence in sixty-two patients with 'centrencephalic epilepsies'.

twelve females. They ascribed the sex difference to the migration of the men to towns in search of work. Male epileptic patients probably attended the hospital for treatment 'because the continuance or appearance of their fits constituted a threat to their livelihood'. In the Semokwe Reserve, of 130 epileptic patients, seventy-three were male and fifty-seven female. They concluded: 'We doubt whether so great a sex difference actually exists, but are unable to account for the discrepancy'. They quoted Chitiyo who had more male epileptic patients in Port Herald Hospital, Nyasaland. In contrast to the higher male incidence of epilepsy, Hurst *et al.* (1961) found twenty-one males and twenty-nine females.

In conclusion, it might be observed that for any epidemiological data to be reliable, the

sample should be representative of the general population and any selection exercised should be weighted only for the criteria of the disorder under analysis. Thus, the higher male incidence observed in Africa might well be due to unnecessary and artificial selection of the various samples.

Age of onset of attacks

In the present series (centrencephalic and symptomatic cases) there appeared to be a double 'rise' in the incidence of the epilepsies related to age of onset. Up to the age of 1 year, there were ten patients. Between 13 months and 5 years of age, the number of epileptics had risen to twenty-eight. This was followed by a fall to fifteen cases between 6 and 10 years of age. Between 11 and 20 years of age, the number of patients had risen once more to twenty-nine. This was similarly followed by a fall to sixteen in the 21–30 years age group—and it continued to fall in the older age groups. The first 'rise' was produced equally by both the centrencephalic and symptomatic groups but the second 'rise' at 11–20 years age group was largely due to the centrencephalic epilepsies.

This observation was not noted in an earlier paper (Dada & Odeku, 1966) but retrospective examinations of the report showed that this double 'rise' was also present then. Similar observation was made by Piraux (1960). Kaushik (1960) reporting an earlier experience in Ibadan did not observe this phenomenon.

Most of the epileptic patients had their first attack before the age of 20 years. Levy *et al.* (1964), reported 56% of the Semokwe Reserve patients had their first seizure whilst under 20 years of age but in the European group this figure was 72%. In our present series 70% of the cases were under 20 years of age when they had their first seizure.

Sex related to age of onset

Apart from a higher female incidence at 6–10 years of age, the male preponderance was maintained throughout all the other age groups. Unfortunately, our selected series could not be compared with the sex distribution in the general population.

Duration of symptoms prior to first consultation at the Lagos University Teaching Hospital

Seventy-eight of the 117 patients (66·7%) had their symptoms for over 52 weeks before being seen at the Hospital (Table 6). There are a number of reasons for this. As will be shown later in the attitude survey, it is popularly believed that since epilepsy is due to supernatural powers of evil spirit or 'juju', the 'native doctor' is the best person to cure it. Thus, forty-seven of the 117 patients (40·2%) had received 'native treatment' alone whilst a further twenty-five (21%) had combined both orthodox and unorthodox ('native') treatment (Table 8). With the failure of unorthodox treatment, the patients turned to orthodox management. Ninety-one of the 117 patients (77·8%) had experienced four attacks or more before their first consultation at the Hospital (Table 7). These late consultations tended to produce very gross neurological or psychiatric abnormalities in the patients. Brain tumours tended to be massive in size. The late consultation and delayed treatment occasionally affected the prognosis. This unfortunately strengthened the belief of those advocating 'unorthodox' treatment. All the ten patients who had a solitary attack before their first consultation either had other attacks subsequently or had abnormal neurological findings to qualify them for inclusion in this series.

TABLE 6. Duration of symptoms prior to first consultation at L.U.T.H.

	117 cases	% of 117
Within a few hours to 6 days	17	14·5
1–4 weeks	4	3·4
5–12 weeks	3	2·6
13–24 weeks	8	6·8
25–52 weeks	7	6·0
Over 52 weeks	78	66·7

TABLE 7. Number of attacks before first consultation at L.U.T.H.

	117 cases	% of 117
One attack	10	8·5
Two attacks	8	6·8
Three attacks	8	6·8
Four or more attacks	91	77·8

TABLE 8. Previous medication

	117 cases	% of 117 cases
None	23	19·7
Orthodox	22	18·8
Unorthodox ('native')	47	40·2
Orthodox and unorthodox ('native')	25	21·4

Previous medical history

There is probably no other disorder that previous medical history is as important as in the epilepsies. Whilst the relationship between this symptom-complex and various previous episodes in the patient's life may not be immediately apparent, the data collected from such information may in time prove very valuable.

The incidence of the previous illnesses or 'accidents' was compared in the three groups, namely, the whole series of 117 cases, the centrencephalic group (sixty-two cases) and the symptomatic group (fifty-five cases) (Table 9).

A large number of the patients could not supply the information required on their previous illnesses or accidents. Statistical analyses of the available data did not show any significant differences between the centrencephalic and symptomatic groups. However, when the incidence of these predisposing factors (previous illnesses, Tables 10–15) was compared with a group of 117 non-epileptic controls (who were matched age for age and sex for sex) significant differences occurred in all the factors except for prolonged jaundice, the figures being too small for statistical analysis.

TABLE 9. Previous medical history

	117 cases	Centrencephalic (62 cases)	Symptomatic (55 cases)
Difficult labour	9 (7·7%)	4 (6·5%)	5 (9·1%)
Prolonged jaundice	3 (2·6%)	2 (3·2%)	1 (1·8%)
Febrile infantile convulsions	26 (22·2%)	11 (17·7%)	15 (27·3%)
Meningitis/encephalitis	5 (4·3%)	1 (1·6%)	4 (7·3%)
Head injury	11 (9·4%)	6 (9·7%)	5 (9·1%)
Severe headaches	19 (16·2%)	8 (12·9%)	11 (20%)
Failing vision	5 (4·3%)	1 (1·6%)	4 (7·3%)
Family history of epilepsy	15 (12·8%)	10 (16·1%)	5 (9·1%)

TABLE 10. Difficult labour

	Patients	Matched controls
Yes	9	6
No	89	111

$\chi^2 y = 0{\cdot}7988$.
$P = 0{\cdot}30$–$0{\cdot}50$, not statistically significant.

TABLE 11. Febrile infantile convulsion

	Patients	Matched controls
Yes	26	5
No	73	112

$\chi^2 y = 18{\cdot}76$ with 1 degree of freedom.
$P < 0{\cdot}001$.

TABLE 12. Prolonged jaundice

	Patients	Matched controls
Yes	3	5
No	94	102

TABLE 13. Meningitis/encephalitis

	Patients	Matched controls
Yes	5	0
No	104	114

TABLE 14. Head injury

	Patients	Matched controls
Yes	11	5
No	101	112

$\chi^2 y = 1{\cdot}92$ with 1 degree of freedom.
$P = <0{\cdot}1\text{-}0{\cdot}2$, not statistically significant.

TABLE 15. Family history of epilepsy

	Patients	Matched controls
Yes	15	0
No	102	115

PREVIOUS MEDICAL HISTORY

'The past events of a person's life may be not a string of pearls, but a series of unfortunate happenings. Which of these may be blamed for a late-arriving epilepsy?' (Lennox, 1960).

The significant role played by difficult labour, febrile infantile convulsion, meningitis and encephalitis, and family history of epilepsy, have been shown in the statistical data presented by comparison of the 117 epileptic patients with 117 controls who were matched age for age and sex for sex. The data available for prolonged jaundice at birth and head injuries were too small to be of statistical significance.

In Africa, poor antenatal and obstetric care is responsible for a high percentage of birth injuries. Hurst *et al.* (1961) had an incidence of 14% of their fifty epileptic patients who gave a history of birth trauma. If the number of people failing to supply information of their birth (the 'don't-knows' and 'not recorded') are removed from the present series of 117 patients, then nine out of ninety-eight patients (9·1%) had prolonged labour. The role played by birth trauma in the pathogenesis of epilepsy may be by producing sclerosis of Ammon's horn, or infantile hemiplegia from temporary carotid occlusion produced by badly applied forceps or by haematoma—extradural or intradural.

The other form of head injury—that is post-natal—did not feature prominently in the present series largely because cases of head injuries usually remain under the care of the surgeon except for the consultation request for neurological opinion. Nevertheless, very few of the patients gave a convincing history of antecedent head injuries in this series. Head injury as a factor in the pathogenesis of epilepsy is likely to assume a more important role in the future in Nigeria with the vast increase in vehicular traffic and recklessness of drivers as well as a result of war wounds.

Febrile infantile convulsion is another important predisposing factor in epilepsy. It is a very common condition in Nigeria (Table 16 and Fig. 3).

The incidence of febrile infantile convulsion shows two peaks, one during the rainy season (April–September) and the other during the dry season (October–March) (Fig. 3).

Malaria and gastroenteritis have their peak incidence during the rainy season and most of the cases of febrile infantile convulsion are due to either of these infections. The exact pathogenesis of simple febrile infantile convulsion (that is, due to extracranial disorder) remains obscure—but probably hyper-natremia associated with the dehydration of gastro-enteritis may be responsible for the convulsion. Cerebral malaria could cause convulsion on its own accord. During the dry season, upper respiratory infections and meningitis are common. These can and do give rise to convulsion in infants. Ounsted, Lindsay & Norman (1966) have shown the significance of both birth injuries and febrile convulsions in the

TABLE 16. Febrile infantile convulsion

	Seen	Dead
Total no. of paediatric emergencies	17087	589 (3·5%)
Total no. of febrile infantile convulsion	952	100 (10·5%)

Febrile infantile convulsion accounted for 17% of all deaths.

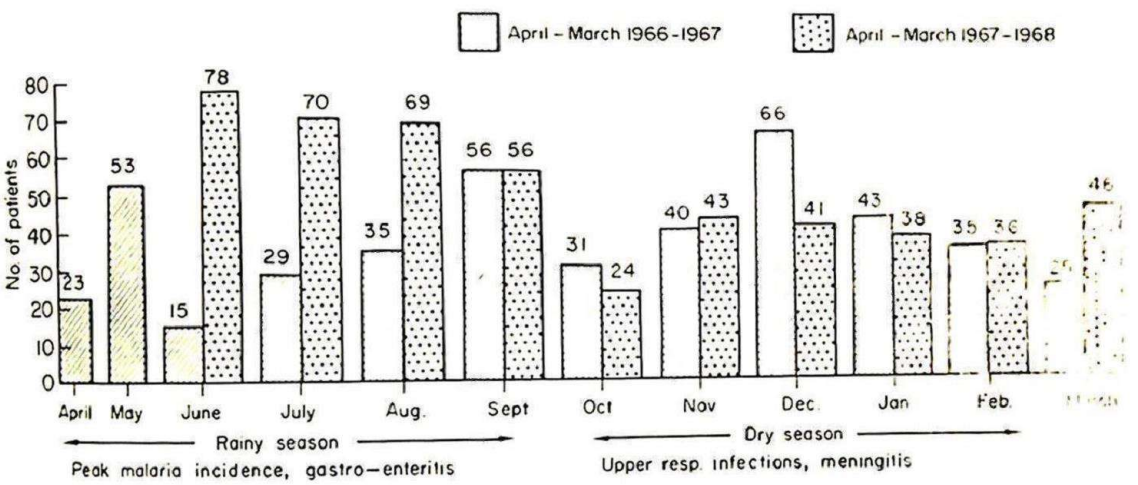

FIG. 3. Febrile infantile convulsion seen in casualty of Lagos University Teaching Hospital between April 1966 and March 1968.

pathogenesis of temporal lobe epilepsy. In the present series, the condition accounted for 22·2% of the 117 epileptic patients. When those who could not answer the question on early febrile convulsion are removed, then twenty-six of the ninety-nine remaining epileptic patients (i.e. 26%) had febrile convulsion.

Thus, 'the prognosis of febrile convulsion is almost certainly not as good as has been thought' (W.H.O., 1957).

Previous meningitis and encephalitis occasionally give rise to epilepsy as a result of scar formation. There were five such cases of a total of 109 patients (4·5%) who could give the answer in the enquiry in previous medical history. Levy *et al.* (1964) reported 6% of their 100 patients had meningitis and encephalitis previously.

Since the causation of the epilepsies may be due to birth trauma, early childhood infec-

tions (febrile convulsion, meningitis and encephalitis) and since all these disorders are indicative of the standard of antenatal and obstetrical care, as well as the general public health, then incidence of epilepsy in underdeveloped or developing countries should be higher than in the more developed countries. This should be more so when the role of parasitic diseases and poor nutrition is considered. That the incidence of epilepsy in Africa approximates the established incidence in more developed countries must mean that many cases of epilepsy in the African never get to the hospitals as well as a high level of concealment when public enquiries are made into the incidence of epilepsy.

It is also expected that genetic influence would play its part in the predisposition of the African to epilepsy. Fifteen of the present series of 117 patients (12·8%) gave a family history of epilepsy. Levy *et al.* (1964) found a higher familial tendency, 36% in their 100 Semokwe Reserve patients. This is rather high and it may well be that the African interpretation of 'family' is responsible. The figures given by Hurst *et al.* (1961) from among the Bantu of thirteen out of forty-six families (28·3%) may be considered high for the same reason.

In the present series, 16·1% of patients with centrencephalic epilepsies as opposed to 9·1% of the patients with symptomatic epilepsies have a positive family history of seizures.

Frequency of attacks

The frequency of seizures in the 117 patients is given in Table 17.

TABLE 17. Frequency of fits related to types of epilepsy

	117 patients		Idiopathic		Symptomatic	
	No.	%	No.	%	No.	%
Once only	7	5·9	5	8·1	2	3·6
Once daily (about 360 attacks per annum)	3	2·5	3	4·8	0	0
Many times daily (more than 360 attacks per annum)	12	10·2	5	8·1	7	12·7
Once weekly (about 50 attacks per annum)	5	4·2	4	6·5	1	1·8
Many times weekly (more than 50 attacks per annum)	18	15·3	8	12·9	10	18·2
Once monthly (about 10 attacks per annum)	17	14·5	6	9·7	11	20·0
Many times monthly (more than 10 attacks per annum)	21	17·9	11	17·7	10	18·2
Once every 3 months (about 4 attacks per annum)	7	5·9	4	6·5	3	5·5
Once every 4–6 months (about 2–3 attacks per annum)	10	8·5	6	9·7	4	7·3
Once every 7–12 months (less than 2 attacks per annum)	7	5·9	5	8·1	2	3·6
Once over 12 months (about 1 attack per annum)	10	8·5	5	8·1	5	9·1

Sixty-one of the 117 patients (52·1%) had from ten to over fifty (but less than 360) attacks per annum and this was reflected in the two groups of epilepsies as well. These sixty-one patients constituted the moderately severe group.

Fifteen patients (12·7%) were troubled by daily attacks. Eight of these were centrencephalic and seven were symptomatic. These fifteen patients constituted the severest group.

The least severe group comprised forty-one of the 117 patients (35·1%). They had from one attack in one year or more to about four attacks per annum.

Frequency of a fit is an important criterion for prognostication and assessing the result of

treatment. For admission into the study on prognosis, patients with three or more reported fits per annum and who had been on regular treatment for at least 1 year were included.

The frequency of the fits is related to sex, age of onset and diagnosis in Table 18.

Levy *et al.* (1964) found 11% of their 130 patients were in the severest group, 72% in the moderately severe and 14% in the least severe group. Three cases were indefinite as to the frequency of their fits. Bird (1964), in his study of epilepsy among the Bantu mine-workers, did not specify the frequency of fits in his report which only mentioned the number of fits. 27% had more than twenty fits whilst 63% had up to five fits. Piraux (1960) observed that 12% of his patients had less than four attacks per annum whilst 32% had more than four attacks per week, 35·5% had less than four attacks per month and 20·5% experienced less than four attacks per week.

TABLE 18. Frequency of fits related to sex, age and severity of epilepsy

		Very severe (15 patients; 12·7%)	Moderate (61 patients; 52·1%)	Mild (41 patients; 35·1%)
Sex	Male	8	42	19
	Female	7	19	22
Age of onset	Up to 1 year	0	9	1
	13 months to 5 years	5	15	8
	6–10 years	3	7	3
	11–20 years	3	12	15
	21–30 years	1	6	9
	31–40 years	1	10	2
	41–50 years	0	1	3
	51–60 years	0	1	0
	Over 60 years	0	0	0
Diagnosis	Centrencephalic	8	29	27
	Symptomatic	7	32	14

Differentiation into centrencephalic and symptomatic epilepsies

The series of 117 patients comprised sixty-two cases of centrencephalic epilepsies and fifty-five cases of symptomatic epilepsies. The presence of an aura, localization of the attacks, retention of consciousness, and the presence of neurological abnormalities on examination indicated the possibility of symptomatic epilepsy. The following tables show that this differentiation of symptomatic from centrencephalic epilepsies was significant.

Aura

The presence of an aura distinguished between the two groups of epilepsies (Table 19). Five of the cases of presumed centrencephalic epilepsies described aura before their attacks. After excluding the possibility of symptomatic epilepsies in these patients, it was felt that their psychological reaction to the attacks was probably responsible for the presence of the aura. It was interesting to observe that all these five patients had seen more than two doctors before being seen in our clinic. It was therefore possible that an aura might have been suggested to them by their previous medical attendants during 'direct questioning'. These were also the five patients who described localization of their attacks (Table 20).

Most of the vivid auras were described with psychomotor epilepsy. These included sudden fear from an approaching force, visual hallucinations (dancing gnomes), epigastric sensations (rising epigastrium or 'tiredness' from within the abdomen) and dizziness. No patient described a 'déjà vu' experience. Also encountered were sensory auras—usually a feeling of insects crawling on part of the body.

TABLE 19. Aura

	Centrencephalic (62)	Symptomatic (55)
Aura present	5	28
Aura absent	56	27
Not recorded	1	0

$\chi^2y = 23{\cdot}866$ with 1 degree of freedom.
$P < 0{\cdot}001$.

TABLE 20. Localizing features of attacks

	Centrencephalic (62)	Symptomatic (55)
Present	5	34
Absent	56	21
Not recorded	1	0

$\chi^2y = 34{\cdot}899$ with 1 degree of freedom.
$P < 0{\cdot}001$.

Localizing features of attacks

The commonest localizing feature was involuntary movement of limbs of one side of the body (twenty-two cases), of only one limb (three cases). Adversive movements of the head, eyes and rotation of the neck were seen in ten cases. Psychomotor automations were observed in three cases. Aphasic arrest followed by loss of consciousness was described by three patients.

Neurological examination

Details of the neurological examination are given in Table 21.

TABLE 21. Neurological examination

	Centrencephalic	Symptomatic
Normal	54	10
Abnormal: localizing	0	39
Abnormal: not localizing	6	1
Cerebral palsy	2	5

$\chi^2y = 53{\cdot}4$ with 1 degree of freedom.
$P < 0{\cdot}001$.

CLINICAL TYPES OF EPILEPSIES IN 117 CASES

On the history of the patient's symptoms, the description of the seizure, past medical and family history and physical examination, it was possible to arrive at a provisional diagnosis of the clinical type of epilepsy as shown below.

Centrencephalic epilepsies	
Grand mal	63 cases
Petit mal	1 case
Infantile spasm	1 case
Total	65 cases

Symptomatic epilepsies	
Focal (not temporal lobe)	20 cases
Jacksonian epilepsies	6 cases
Psychomotor epilepsies	12 cases
Hemiconvulsion—hemiplegia epilepsies (H.H.E.)	11 cases
Total	49 cases
Status epilepticus	3 cases

Following investigations with or without fresh information on the patient's symptoms, past medical and family history and reviews of physical examination, the final diagnosis of the clinical types of epilepsies is as follows:

Centrencephalic epilepsies	
Grand mal	60 cases (51·2%)
Petit mal	1 case (0·8%)
Infantile spasm (salaam fit)	1 case (0·8%)
Total	62 cases (53·0%)

Symptomatic epilepsies	
Focal (not temporal lobe) epilepsies	17 cases (14·5%)
Jacksonian epilepsies	6 cases (5·1%)
Psychomotor epilepsies	11 cases (9·4%)
Hemiconvulsion–hemiplegia epilepsies	11 cases (9·4%)
Secondary 'grand mal' epilepsies	10 cases (8·5%)
Total	55 cases (47%)

Thus, the symptomatic epilepsies accounted for 47% of the total series of 117 patients.

Clinical types of epilepsies in 117 cases

Dada & Odeku (1966) in a similar series of 234 epileptic patients had shown the symptomatic epilepsies to be only 26·7%. Levy *et al.* (1964) grouped their epilepsies into generalized, focalized, petit mal, amyotonic and unspecified. Clearly the 'focalized' group represented symptomatic epilepsies some of which might be in the 'generalized' group as well. Their 'focalized' group was 12% in the Harare Hospital series, none in the Semokwe Reserve series and 15% in the European group. Piraux (1960) found that 158 (75%) of his 209 epileptic cases were symptomatic. 'Splanchnopsychic' attacks (temporal lobe epilepsies) accounted for eighty-six cases (41·1% of the total). Gelfand (1957b) observed that idiopathic epilepsy is the most common form of epilepsy in the African but no figures were given.

With reference to the clinical types encountered in centrencephalic and symptomatic epilepsies, sixty of the 117 patients (51·2%) had grand mal, focal (excluding temporal lobe) epilepsies accounted for seventeen cases (14·5%). There were eleven cases of psychomotor epilepsies (9·4%), of hemi-convulsion–hemiplegia epilepsy (H.H.E. syndrome) ten cases (8·5%) presented with secondary 'grand mal'. There were six cases (5·1%) of Jacksonian epilepsies. Only one case of petit mal (0·8%) was encountered in the present series. There was only one case of infantile spasm (salaam fit).

Piraux (1960) had thirty-one cases of idiopathic epilepsies from among 209 epileptic patients in the Congo, a percentage frequency of only 18·1. There were 158 cases (75·5%) of partial or symptomatic epilepsies. The remaining twenty cases could not be classified. Hurst *et al.* (1961) reported forty-one of fifty-one epileptic patients (80·3%) had grand mal, two had petit mal (3·9%) and the remaining eight patients (15·7%) had focal epilepsy. Levy *et al.* (1964) found eighty-six of their 100 patients in the Harare Hospital group and 91% of the Semokwe Reserve group had 'generalized' seizures which probably comprised both centrencephalic and symptomatic types. This is because twenty-seven of the eighty-six patients had premonitory symptoms. There were only 4·5% who suffered from petit mal in the Semokwe Reserve group; no petit mal was recorded in the Harare Hospital group. Dada & Odeku (1966) found 167 cases of centrencephalic epilepsies in their 234 epileptic patients. These consisted of thirteen cases of petit mal, eleven of petit mal–grand mal, and 143 cases of grand mal epilepsy. There were sixty-seven cases thought clinically to be due to a focal pathology, twelve of these were post-traumatic epilepsy; of the remaining fifty-five cases. the cause of the seizures was proved in twenty-four cases. Twelve of these twenty-four presented with Jacksonian epilepsies, eleven were motor and one was sensory. There was only one case of psychomotor epilepsy in the twenty-four cases. The remaining eleven cases showed other forms of focal epilepsy. The data on the clinical types of the thirty-three cases without proved aetiology were not available.

Experience in Africa shows that centrencephalic epilepsies account for the majority of the cases. The relative proportions of centrencephalic to symptomatic epilepsies depends on the interest and experience of the clinician backed by adequate facilities for investigations.

General medical examination

Only five of the patients (all in the symptomatic group) had some abnormalities on general medical examination. These were considered related or contributory to the seizures in three cases (pulmonary tuberculosis and tuberculoma—one case; facial naevus in two cases of Sturge-Weber Syndrome). Of two cases with unrelated abnormalities in the general medical examination, one had infective hepatitis and the other had chronic cystitis.

Evidence of native treatment of epilepsy

Unorthodox 'native' treatment of epilepsy consisted of a mixture of cow's urine, tobacco leaves and other ingredients. This mixture, which is taken by mouth as well as rubbed on the skin, was administered during or shortly after a seizure; its odour lasts for several hours after administration. Thus, its use cannot be easily disguised although several patients and their relatives denied that the medicine was ever used. This same mixture is used in all forms of convulsive disorders. Since most of these cases were not seen during or shortly after an attack, in only seven cases could any evidence of native medical treatment be found.

Unusual presentation of epilepsy

Four of the patients (young epileptics) presented with enuresis. Hypoglycaemia was excluded as a possible cause of these nocturnal epilepsies.

Tetanus

One patient was admitted to the hospital with tetanus which had developed from the native surgical procedures he received.

Associated conditions

Pregnancy. Five of the patients had their first relapse of epilepsy since childhood, during pregnancy. These seizures occurred from the third month of pregnancy onwards. None of them had eclampsia. Two of the patients also had seizures soon after parturition.

Migraine. There was one case with migraine in the series.

Hospitalization

Ninety-five of the 117 patients (81·2%) were hospitalized for investigations and treatment. Three arrived in status epilepticus, one of whom died. The autopsy showed fronto-parietal meningioma.

DISCUSSION ON AETIOLOGY OF SYMPTOMATIC CASES

There were fifty-five patients presumed to have symptomatic epilepsy on the basis of the clinical history including a record of an eye-witness account of the seizure, neurological findings and results of investigations.

The sex distribution was about equal in this group, there being twenty-eight males and twenty-seven females. The age of onset and sex distribution is shown in Fig. 4.

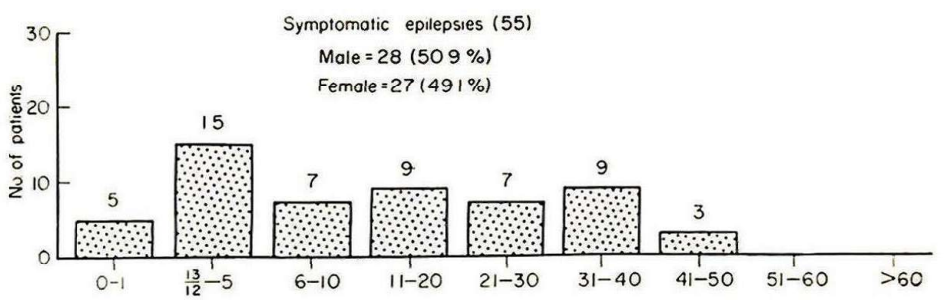

FIG. 4. Age of onset and sex incidence in fifty-five patients with symptomatic epilepsies.

It was possible to arrive at a diagnosis in thirty-three of the fifty-five cases (60%), although in the three patients with mental retardation who were presumed to be due to inborn errors of metabolism, the biochemical lesion could not be identified due to lack of technical facilities. In the remaining twelve patients (40%) there were ten defaulters and two patients

were awaiting more definitive investigations although cerebral tumour was highly probable in each of these two cases.

The analysis of the thirty-three patients was as follows:

Hemiconvulsion–hemiplegia epilepsy syndrome (H.H.E.)	11 cases
Cerebral palsy	7 cases
Meningo-encephalitic scars (three post-traumatic epilepsy and two post-infective)	5 cases
Meningioma	3 cases
Mental retardation (in-born errors of metabolism)	3 cases
Tuberculoma	2 cases
Sturge-Weber syndrome	2 cases

PARASITIC DISEASE AND EPILEPSY

The high incidence of parasitic infestation in underdeveloped countries may be expected to give rise to large numbers of epileptic patients. Cysticercosis is perhaps the most frequent parasitic disease associated with epilepsy (MacArthur, 1934; Ray, 1941; Dixon & Hargreaves, 1944; Acha & Aguilar, 1964; and several others. Dixon & Hargreaves (1944), reviewing relevant literature, found ninety-nine cases in 52 years. They added 185 of their cases bringing the total to 284 in 1944. The figure has probably increased to 400 or so by now, i.e. during 76 years, an average of about five cases per annum. Earlier reports by Hulshoff (1937) incriminating malaria in the pathogenesis of epilepsy had not been confirmed by other workers. However, malaria can produce epilepsy by causing febrile infantile convulsion which may give rise to post-convulsive encephalopathy.

Another parasite thought to be involved in the pathogenesis of epilepsy is *Ascaris* (Sprent, 1955; Brown, 1965a).

Schistosomiasis of the brain had also been held responsible in some cases of epilepsy. Kane & Most (1948) found fifty-one cases in literature up to May 1946, twenty-seven of these presented with convulsions.

Similarly, a number of reports have incriminated *Filaria* in the pathogenesis of epilepsy (Carayou, Collomb & Sankale, 1959).

More recently, *Toxocara* has been suggested as a possible epileptogenic parasite (Woodruff, Biseru & Bowe, 1966).

Production of epilepsy by parasite may occur in a number of ways, notably by:

1. Actual migration of the parasite into the brain (Sprent, 1955).
2. Production of a toxic, convulsant substance by the parasite (Deschiens & Poirier, 1949).
3. Transportation of virus to the brain by the parasite with subsequent encephalitis and epilepsy.
4. A conditioned metabolic deficiency which predisposes to convulsion. Chance & Dirnhuber (1948) examining the vitamin composition of some worms and their hosts found *Ascaris* contained more pyridoxine than liver of the host. Brown (1965a) suggested a conditioned pyridoxine deficiency may be found in patients infested with *Ascaris*.
5. Production of allergic manifestations which may include convulsion (Sprent, 1951).
6. Febrile convulsion in infants which predisposes to epilepsy (Ounsted *et al.*, 1966).

In view of these reports the patients with epilepsy were screened for evidence of parasitic

disease. The blood was examined for high eosinophilia, malaria and filarial parasite latter requiring examination of mid-day and mid-night specimens. The terminal urine patients was examined for schistosome ova. Skin test for toxocariasis was carried o skin snips were examined for *Onchocerca volvulus*. The stool was examined by m Stoll count method for ova of all the parasites. Evidence of pyridoxine deficienc investigated by the Tryptophan load test. Apart from routine skull and chest radiog soft tissue radiographs were carried out to exclude calcified parasites.

Before incriminating any parasitic disease in the pathogenesis of epilepsy, it is nec to assess the incidence of the various parasitic diseases in the community. Malaria course, prevalent. Schistosomiasis exists in Southern Nigeria especially in the creek ar

Okpala's (1961) survey on the 'normal' intestinal parasitic flora (as revealed by examination) in symptomless Nigerians, showed 71·5% had *Ascaris*, 66·6% had *Tr trichiuria*, 58·3% had hookworm ova. Gilles (1964) in an environmental study of a Ni village community also found the incidence of hookworm (*Ankylostoma duodena Necator americanus*) 71%, ascariasis 70% and *Trichiuris trichiura* 45%.

Cysticercosis appears to be rare in West Africa (Bowesman, 1952). More recently O ten & Laing (1967) reported a case from Ghana. Gilles (1964) did not find a case village population sample he studied. In contrast to its rarity in West Africa, cysticerc common in South Africa (Heinz & Macnab, 1965) and in Southern Rhodesia (G 1957b) where calcified cysts of taenia solium were found in radiographs of eight seventy-five cases of apparently idiopathic epilepsy.

The following methods were used for the investigations:

Routine investigations. Regardless of the type of epilepsy presented, all the patient investigated as follows: (1) white cell count—differential and total; (2) stool examinati parasites and ova; (3) skull, chest and soft tissue radiographs; (4) urine (terminal, possible) for schistosome ova.

Further investigations were carried out on the patients with high eosinophilia or more in the absence of other causes of high eosinophilia, notably allergic or hyperse states. There were forty-two such patients (3·59%). Also included in this group were patients who had 'many *Ascaris* ova' in their stool specimens. These fifty-four were further investigated as follows: (1) ova count in stool by a modified Stoll (2) blood examinations at mid-day and mid-night for microfilaria; (3) skin snip fo *cerca volvulus*; (4) *Toxocara* skin test; (5) tryptophane load test for pyridoxine d

Space does not permit a full report on the results of these investigations which presented elsewhere (Dada, 1969). In general, apart from a higher incidence of *Toxocara* skin test (13·0%) among the epileptic patients (instead of a 'normal' inci 2·1%) no parasitic disease could be incriminated in the present series of 117 patie high incidence of positive *Toxocara* skin test may indicate toxocariasis as the epilepsy in some, if not all, of these cases as suggested by Woodruff *et al.* (1966). before any firm conclusions can be made concerning the role of toxocariasis in genesis of epilepsy, it is necessary: (a) to test a larger number of epileptic pati employ other confirmatory tests—for example, serological investigations; and (c strate the existence of the toxocara, within the brain.

It is of interest to note that Haddock (1968), working in Ghana, has foun higher incidence of positive toxocara skin tests among his epileptic patients. He, maintains that the test is not sufficiently sensitive.

THE SIGNIFICANCE OF HAEMOGLOBINOPATHY IN THE PATHOGENESIS OF EPILEPSY IN NIGERIA

Convulsions feature prominently in the neurological manifestations of sickle cell disease. Quite often these convulsions occur during an acute cerebrovascular insult and may not necessarily be epileptic. Similarly in younger children, febrile seizures which occur during 'crisis' may not necessarily be epileptic. Hughes, Diggs & Gillespie (1940), in an extensive review of literature on the involvement of the nervous system in sickle cell anaemia, found convulsions in eight of the thirty-one patients. Greer & Schotland (1962), reviewing 400 cases of patients with abnormal haemoglobin, found thirty-two patients (8%) had convulsions. Twenty-four were less than 14 years old and only four were more than 33 years old at the time of the first seizure. One patient had psychomotor seizures only. The others had generalized motor seizures; five patients with cerebral vascular syndrome had focal motor seizures as well. Of fifteen patients with recurrent seizures, five, including the patient with psychomotor spells, never had more specific symptoms of haemoglobin disease. Precipitating factors thought to be related to the abnormal haemoglobin were present in twenty-two patients. These factors include surgery and anaesthesia pregnancy parturition, transfusion, severe dehydration, fever and infection and a variety of hypoxic states.

Baird *et al.* (1964), in an attempt at clinico-pathological correlation in five autopsy cases, recognized two types of pathological lesions—vascular capillary occlusion and dilatation of the precapillary arterioles, deposition of iron pigment in the free state and within the macrophages on the one hand and parenchymal changes of small infarcts and neuronal loss on the other. Three of these five patients presented with convulsions, lethargy in one case, grand mal type seizures, transient unresponsiveness in another case, whilst a third had transient episodes of disorientation, transient weakness of the extremities. There was, however, no convincing clinico-pathological correlation as to the site of the lesions suffered by these patients.

Rowland (1952) had seven patients with seizures in his series of twenty-seven cases of sickle cell anaemia with neurological manifestations. In five of these patients, the seizures occurred only once and in the presence of complicating acute states. He sounded a word of warning: 'While sickle cell disease can cause a variety of acute and chronic neurologic syndromes whether or not anaemia is present, the co-existence of the sickling tendency and neurological disorder is not always etiological and other causes must be excluded.'

Sickle cell disease may be associated with epilepsy (distinct from non-epileptic convulsion) in a number of ways. Repeated convulsions may give rise to post-convulsive epileptogenic scars (Norman, 1962). Epilepsy may supervene a cerebro-vascular accident induced during a sickling crisis, hence the interest in the hemiconvulsion–hemiplegia epilepsy (H.H.E.) syndrome or post-hemiplegic epilepsy in these series. However, in spite of the prevalence of sickle cell disease in Africa, there has been no report linking sickle cell disease with epilepsy.

The results of genotype determination, carried out in 93 of the 117 cases, show that haemoglobinopathy does not appear to play any significant role in the pathogenesis of epilepsy in Nigeria but much larger series of epileptic patients will have to be studied before any firm conclusions could be made. The generalized convulsions occurring during crisis are probably not epileptic since in a large proportion of the cases these convulsions represent isolated events, as pointed out by Rowland (1952).