NEURO-PSYCHIATRIC DISORDERS IN THE BANTU

1. CONVULSIVE DISORDERS — A PILOT STUDY WITH SPECIAL REFERENCE TO GENETIC FACTORS

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This article is the first report of a long-term investigation which is being carried out at Meadowlands Clinic into neuro-psychiatric disorders occurring among the Bantu in this part of the Witwatersrand area.

The question of the comparative spectrum of neuro-psychiatric disease, both as regards relative incidence and variable pattern of symptoms in different ethnic groups, is but part of the current world-wide movement for the study of the epidemiology of disease in general. In defining the field as a whole one of us[1] drew attention to the work already done at Baragwanath Hospital (the parent hospital of the Meadowlands Clinic) by Reef, Lipschitz and Block[2] demonstrating a difference in distribution of neurological disease (including disseminated sclerosis) in the Bantu as compared with Whites. Moreover, our clinical impression suggests that at Baragwanath Hospital there is a high incidence of gross hysterical states comparable to that characteristic of a less sophisticated era in Europe. The rich prospects for research held out by such observations has led to our embarking on our present series of studies.

The main objects of this study, which is our first investigation in this field, are as follows:

1. A longitudinal study of the incidence of convulsive disorders, mental disorder, and mental deficiency with reference to the ethnic variations of these conditions.
2. An investigation into the genetics of these disorders.
3. A study of congenital neuro-psychiatric diseases in relation to the abnormalities of pregnancy.
4. The assessment of aetiological factors such as nutritional status, alcohol consumption, helminth infestation, primitive beliefs, and socio-economic causes, and the integration of the patient in the family and community milieu.

Phase 1 of this investigation is limited to a study of convulsive disorders.

METHODS OF INVESTIGATION

This project was commenced on 1 September 1959, and the preliminary results assessed on 1 March 1961.

Meadowlands Clinic is a polyclinic where all types of illnesses are investigated. The clinical records are filed in a cohort system of age and ethnic groups. All neuro-

psychiatric diseases are indicated by a system of signals which facilitates abstraction of these from the various filing cohorts. The clinic has been established for 3½ years and, from the issue of cards, it appears that there is nearly full coverage of the population who have attended the clinic at one time or another for various complaints.

All the recorded patients and new patients have been examined at the clinic during the period of this survey. If the diagnosis could not be determined the patients have been admitted to Baragwanath Hospital for further investigations. The homes of all patients were visited by a trained Bantu nurse to obtain additional information on family backgrounds.

Environment and Demography

Meadowlands is a Bantu residential area with a population of close on 60,000. There are 8 administrative zones with separate ethnic-group demarcation, viz. Nguni, Sotho, Shangaan, and Venda races.

TABLE I. ETHNIC COMPOSITION OF MEADOWLANDS TOWNSHIP

Nguni	*Sotho*	*Venda/Shangaan*	*Total*
15,300	29,500	11,500	56,300

Intermarriage does occur among the groups, but the Shangaans and Vendas still maintain many of their primitive customs. The women wear leather skirts and metal rings on their feet. In some households food is imported from the Northern Transvaal and corn is ground in wooden mortars. Many of the children are sent back to the tribal areas for long periods, where they are subject to such diseases as malaria and bilharziasis.

We observe a transition stage between a primitive rural life and the complexities of modern civilization. The mental adaptation to these changes has yet to be assessed. The tribal protective authority has broken down to be replaced by European laws and customs with totally different social disciplines. A complex economic environment has replaced the simple economy of the tribe. Rising costs make it necessary for both parents to work. This is evident in the Nguni and Sotho households where lack of parental care tends to produce malnutrition among infants, and delinquency and a high rate of illegitimacy among the adolescent population. There is marked disintegration of the family unit. This is in contrast to the Venda and Shangaan women, who have maintained their tribal outlook and who, for the most part, do not work in the European sphere of influence. They remain culturally primitive while their menfolk have adopted the urban way of life.

An important cause of neuro-psychiatric disorders is the presence of single or combined nutritional/alcohol factors. In these areas, potent alcoholic beverages of bizarre composition are consumed. The long-term ingestion of such drinks, with an inadequate diet of maize, produces physical and psychological pathology which is not seen in European communities.

It will be noted that the general incidence of these convulsive disorders in this area is approximately 1 per 1,000. This figure is lower than the incidence of 4 per 1,000 quoted in a recent survey by the members of the College of General Practitioners in Great Britain.[3] Every effort has been made to trace these patients in Meadowlands. The dramatic and urgent nature of an attack makes attendance for therapy more likely. Nevertheless, one cannot be certain that patients have not short-circuited the clinic service. However, the impression remains that the incidence is low, and until a comprehensive survey is made, the actual figure must remain uncertain. An interesting feature is the very low incidence among the Shangaan and Venda residents. These people are far more primitive in their outlook and way of life than the Sotho and Nguni races. There is no present evidence that among the more primitive tribes the incidence of epilepsy is lower than among more civilized peoples.

TABLE II. ANALYSIS OF CASES

1. *Age/sex distribution of convulsive seizures*

	0 - 9	10 - 19	20 - 29	30 - 39	40 - 49	50 *plus*	*Total*
M.	7	7	4	1	0	2	21
F.	4	10	7	4	3	1	29
	11	17	11	5	3	3	50

2. *Ethnic variations*

	Nguni	*Sotho*	*Venda/Shangaan*	*Total*
Cases	34	13	3	50
Population	15,300	29,500	11,500	56,300
Cases per 10,000 ..	22·2	4·4	2·6	

Clinical Description of Material

The age/sex distribution of our material is depicted in Table II. This follows the general pattern of the age distribution of Meadowlands residents. Of the patients 21 were males and 29 females.

TABLE III. POSSIBLE AETIOLOGICAL FACTORS

Difficult labour, birth trauma ..	7
Head injury	5
Cysticercosis	1
Cardiovascular accident	1

Possible aetiological factors elicited from the history were obtained in 14 patients and these are shown in Table III. The 5 patients with head injuries were all adults and the injuries severe ones.

TABLE IV. TYPE OF ATTACK

Grand mal	41
Focal	8
Petit mal	2

With the aid of interpreters and relatives it proved possible to ascertain the nature of the attack in all patients except one. This information is recorded in Table IV. Twenty-three patients gave a positive history of warning auras before the attacks — a rather high incidence. The nature of the attacks is shown in Table IV. The 8 patients who gave an aura of dizziness found it very difficult to describe the true nature of their experiences. One patient volunteered that his attacks were precipitated by singing, especially hymns, which produced a feeling of elation — 'Doctor, I feel like a king' — and soon afterwards he would have a *Grand mal* attack. Two patients gave rather unusual auras: one felt as if he could not close his eyes and the other felt as if his eyes were full of tears just before an

attack. There were no epigastric auras (Table V). Forty-one patients had *Grand mal* attacks, 8 had focal attacks, and 2 *Petit mal* attacks. One patient had both *Grand mal* and *Petit mal* attacks.

TABLE V. INCIDENCE AND TYPE OF AURA

Dizziness	8
Visual disturbances	4
Tinnitus	2
Feeling of fear	1
Inability to close eyes	1
Crying	1
Eyes full of tears	1
Palpitations	1
Aphasia	1
Confusion	1
Weakness of right hand	1
Feeling of elation—attacks precipitated by singing	1

TABLE VI. INCIDENCE OF ASSOCIATED PSYCHIATRIC FEATURES

Mental defect	12
Psychosis	1
Behaviour disorder	3

Of the 50 patients seen, 12 were regarded as being mentally defective both on history and personal examination. The degree of the defect in all of these was sufficiently severe to justify institutional treatment. One additional child showed severe behaviour problems, and one adult was frankly psychotic. These patients comprise 28% of our total and are recorded in Table VI. This very high percentage is unusual and may be accounted for by the fact that it is for this sort of complaint that the African is most likely to seek the aid of a doctor. These patients are a great burden to their relatives and, since no adequate facilities exist for their care, they have to remain at home and often prevent an adult member of the family from working.

TABLE VII. FREQUENCY OF ATTACKS: GRAND MAL AND FOCAL EPILEPSY

2 or more per week	6
1 per week	14
1 per month	12
1 attack per 3 months	3
Less than 1 a year	4
Bouts of attacks	4

All the patients' records were carefully scrutinized, the frequency of attacks assessed as recorded in Table VII, and the end-results of treatments were assessed. Despite the presence of adequate facilities for the treatment of their disease, not one patient was fully controlled and not one patient attended regularly for treatment as instructed. All patients and relatives were questioned on this aspect and the following facts emerged: Patients attend at the clinic usually soon after they have had an attack and expect to be given medicines which would produce an immediate cure. When the prescribed drugs failed to stop the attacks, or if the attacks recurred when the drugs were finished, some of them lost faith and did not attend again or they began to attend very irregularly afterwards. The idea of continuous suppressive therapy over a period of many years was completely foreign to them. Even after detailed explanation few of them were prepared to accept this form of treatment.

Fourteen of the patients admitted to having consulted witch-doctors. Thirteen were told that they had offended their ancestors and one that he had been bewitched by a neighbour. Suitable treatment was prescribed for all at a cost varying from £1—£5 (R2—R10). No patient benefited from this treatment and no patient returned for a second course.

The attitude of relatives and friends to our patients was ascertained by members of the nursing staff and ourselves. In no case did we find that relatives were ashamed of having epilepsy in their family. They were not afraid of the disease and did not associate it with evil spirits or demons. They regarded epilepsy as they would any other disease with no special stigma attached to it.

Genetic Aspects

The Bantu clinical material at the Meadowlands Clinic holds out the prospect, because of the large family groups in contrast to the smaller family groups of Whites (in Europe and America) upon which our present knowledge is based, of throwing light on the status of genetic factors in epilepsy. The average size of sibship in the 46 families* upon which the present pilot study was conducted is 5·8 with a range of 1—16.

New light on this question is urgently needed because of the radically conflicting evidence of, on the one hand Conrad [4] (Germany) and Lennox and the Gibbses[5,6] (USA) stressing the prominence of the hereditary factor, and on the other, Alström's Scandinavian figures,[7] which reduce the rôle of genetics in this sphere to the barest minimum, a finding which may in a measure correspond with the incursions into the sphere of idiopathic or cryptogenic epilepsy by the neuropathological findings of our neurosurgical age.

What then are the points at issue within the camp of the geneticists in the sphere of epilepsy which require further work in clarification?

The work stressing the importance of the genetic factor comes, as has already been indicated, from two groups. In Conrad's comprehensive pioneer study[4] the expectancy figures in consanguineous groups of cases diagnosed as idiopathic epilepsy were 4·0% for siblings, 4·3% for two-egg twins and 86·0% for one-egg twins. The similarity of the figure for siblings and two-egg twins, categories which may be equated as to hereditary equipment, and the extremely high concordance rate for one-egg twins with identical hereditary equipment as between co-twins, are eloquent and cogent testimony to the validity of the genetic factor. Kallmann, in reflecting on Conrad's work, taken in conjunction with general considerations concerning the ubiquity of the epileptic mechanism in man under certain conditions of release and its prevalence throughout the animal kingdom, is inclined to the view that the genetic mechanism is polygenic. Lennox and the Gibbses,[5] using dysrhythmia in the EEG as their criterion of epilepsy, record the remarkable finding of 100% concordance in one-egg twins and 25% concordance in those of the two-egg variety — the ideal figure for a fully penetrant single dominant gene. Our two groups of workers are thus in full agreement on the importance of

* The discrepancy from the figure of 50 recorded earlier in this article is apparent, not real — being based on the fact that in 4 cases siblings other than the index cases are affected.

the rôle of genetic factors in epilepsy, differing only in the details of the genetic mechanism involved.

Then, in 1950, came the publication of work by Alström,[7] based on a study of epileptic patients admitted during the years 1925 - 1940 to the neurological clinic of the Caroline Institute of the Serefimer Hospital, the only university clinic for neurology in Sweden at that time. Alström remarks that the patients came from all over the country, but that the urban population, especially that from the capital, was over-represented. He claims at the same time that this sample was otherwise probably a more representative one for patients suffering from convulsive disorders than a sample taken from hospitals for the insane or from institutions for epileptics with their selection of mentally affected patients. The investigation of his 897 index cases with their blood relations began in 1945 and ended in 1950.

Salient findings of this study were as follows. In the first place the expectancy figures for parents, $1{\cdot}3 \pm 0{\cdot}27\%$; for siblings, $1{\cdot}5 \pm 0{\cdot}25\%$; and for children $3{\cdot}0 \pm 0{\cdot}93\%$, were not significantly higher than those in the general population. Secondly, families with epilepsy in members other than in the index case are lacking in the majority, i.e. 92%, of cases. Thirdly, among the 16 pairs of twins of this study, two of which pairs were monozygotic, there was not a single case of concordance as to epilepsy. In this connection Conrad[4] discusses a small sample of 4 unselected single-egg Scandinavian twin pairs (the index case, but not the co-twin, suffering from epilepsy in every case) drawing attention to the lack of agreement with Conrad's and Lennox's series,[5] and pinpointing the fact that 'the probability of getting such a sample at random is less than 0·002 if the "degree of manifestation" were to be the same as in Conrad's and Lennox's series'.

Despite Alström's figures quoted above, which reveal no genetic factor in epilepsy, the examination of individual pedigrees in his series discloses, according to Alström's own admission, a genetic factor — in fact a single dominant mechanism — in approximately 1% (11 index cases belonging to 8 families in his sample of 897 index cases and their families). This is the type of genetic mechanism, it will be recollected, that Lennox and the Gibbses postulated as being operative in their series, but present throughout instead of in only 1% of cases. It is, however, impossible to arrive at a final explanation of the discrepant evidence of the two groups of advocates on the available evidence. That is why our present study has been undertaken with a view to clarifying the issue on new material with the peculiarly favourable feature of large family size. Before proceeding to a description of our genetical pilot study, we should point to possible general sources of the discrepancies between the views of Conrad[4] and the Gibbses and Lennox[5,6] on the one hand, and Alström[7] on the other. The following thoughts come to mind:

1. Accepting the possibility that the epilepsies may be divided into two groups, (*a*) genetic, and (*b*) non-genetic, it is readily conceivable that different samples may contain variable loadings of the two varieties, owing to either (*i*) some selective process in the collection of the sample, as might well be brought about by a clinic, in contrast to a mental hospital, constituting the source of the material, or (*ii*) by the more or less plentiful introduction into a specific country or area of the genetic variant of the disease. (Thus, a generally rare genetic condition such as Huntington's chorea or porphyria may, by the chance of introduction into an isolated area, assume disproportionate dimensions.)

2. Alström's work gives the impression of deficient recording of ages of all categories of blood-relationship, resulting in our often having to be content with nett figures which lack universality in the sense of applicability in all samples, and are thus often misleading as compared with expectancy figures.

Let us now turn to the findings of our own study with the pointers it gives for a more comprehensive investigation.

The 46 epileptic patients have an average sibship figure of 5·8 with a range of 1 - 16. Side by side with the advantage for genetic purposes of working with these large family groups, it should be recorded that in this pilot study it has not been possible to ascertain the ages of all the groups of blood relationship investigated, which include siblings, half-siblings, children, nephews and nieces, cousins, parents, uncles and aunts, and grandparents and their siblings. Such ascertainment of age is essential for expectancy studies, and it is proposed to include this in the full investigation which, according to our plans, will follow the current pilot study. This will entail careful investigation by the social worker attached to the proposed team at house-to-house visits, and in view of the cultural limitations of the groups investigated, even in an urban area, we may have to be content with ages assessed as falling within a defined age range rather than the precise age.

Despite this limitation, our preliminary pilot study is adequate to produce evidence along the following two lines:

1. The percentage of families showing one or more members exhibiting epilepsy in addition to the index case, for comparison with Alström's low figure cited above.
2. The types of genetic mechanism suggested in different pedigrees contained in our material.

With regard to the first point, our material shows an incidence of 13 out of 46 families, i.e. a figure of 28·3%* in contrast to Alström's 0·8%. Statistical computation shows this difference to be significant at the 0·1 per cent level. Thus, even at this early stage, our study has afforded evidence on the side of Conrad and the Gibbses and Lennox on the importance of the genetic factor in epilepsy.

Turning to the second point, analysis of the 13 positive pedigrees (of the 46) shows that 3 of these are strongly suggestive of a penetrant single dominant mechanism and 1 of irregular dominance, while the remaining 9 are equally compatible with irregular dominance, or recessiveness, as indicated in Table VIII. A portion at least, therefore, of these results is in line with the thesis of single dominance of Lennox and the Gibbses, appearing also in that of 0·8% of Alström's cases where a genetic mechanism was conceded by him.

As a conclusion to this pilot genetic investigation, it may be said that the positive findings are sufficiently

* This figure is probably conservative, since cases 10 and 39 in Table VIII each had a relative whose psychosis may well have been epileptic in nature.

encouraging to warrant our proceeding to the more comprehensive survey already alluded to.

(At an impressionistic estimate the anticipation that the rate of questionable paternity in our Bantu group would be unduly high was not realized. This point, however, will be one of the special terms of reference of our proposed extended enquiry.)

TABLE VIII. GENETIC FEATURES

Identification	*No. of sibs.*	*Relatives affected*	*Probable type of genetic mechanism*
1 J.Ma.	4	Negative	
2 L.K.	15	Negative	
3 S.D.	0	Negative. Inadequate history	
4 D.S.	2	Negative	
5 L.M.	2	Negative	
6 J.Q.	3	One of five children Two siblings and mother	Penetrant single dominant
7 P.D.	3	Negative	
8 T.L.	3	Negative	
9 I.M.	4	Negative	
10 M.M.	4	Negative. Paternal uncle mentally disordered	
11 M.N.	4	Negative	
12 A.M.	6	Negative	
13 J.Mo.	6	Maternal great-aunt on her maternal side. Maternal cousin on her paternal side	Irregular dominant
14 N.M.	3	Negative	
15 S.B.	4	Nephew (1 of 2 sons of an elder brother)	Irregular dominant or recessive
16 B.K.	3	Negative	
17 M.F.	7	1 sibling	Recessive or irregular dominant
18 M.P.	4	1 uncle and 1 of 5 paternal siblings	Recessive or irregular dominant
19 M.Z.	3	1 of 3 other male siblings	Recessive or irregular dominant
20 G.D.	7	Negative	
21 Ja.Mo.	7	Negative	
22 V.K.	7	Negative	
23 M.N.	3	Negative	
24 E.M.	2	Negative	
25 F.N.	5	Negative	
26 P.M.	5	Paternal grandmother	Recessive or irregular dominant
27 M.Z.	3	Mother, 1 of 4 sibs. and only female child	Penetrant single dominant
28 C.N.	7	Maternal aunt and 1 of 4 siblings	Recessive or irregular dominant
29 W.N.	5	Negative	
30 M.Nd.	6	Paternal uncle and 1 of 3 siblings	Recessive or irregular dominant
31 Jo.Mo.	4	Negative	
32 J.Mak.	4	Negative	
33 L.T.	9	Negative	
34 M.H.	4	Negative	
35 El.M.	0	Negative	
36 P.T.	8	Negative	
37 Ru.M.	6	Sister and father	Penetrant single dominant
38 S.D.	0	Negative. Poor history	
39 B.M.	0	Negative. Paternal grandfather psychotic	
40 S.J.	8 (1 twin pair)	Negative	
41 M.Ng.	7	Negative	
42 D.G.	4	Negative	
43 G.N.	12	1 sister and another sister with confusional episodes	Recessive or irregular dominant
44 D.M.	4	Negative	
45 Jo.Mo.	4	1 sibling	Recessive or irregular dominant
46 P.M.	6	Negative	

SUMMARY AND CONCLUSIONS

1. In this article a series of 50 Bantu patients suffering from convulsive disorders, examined at the Meadowlands Clinic in the Witwatersrand area between 1 September 1959 and 1 March 1961, is reported.

2. A description of special features of an ethnic-cultural type that have presented themselves to date in the clinical and social aspects of our patients, is given.

3. A tentative attempt at aetiological analysis is made.

4. The large family size of our Bantu patients, as compared with the modal White family of our time, has permitted a preliminary assessment, substantiating the correctness of the view of the schools that attribute significance to genetic factors in a considerable proportion of epileptics.

5. The purely pilot nature of this study is stressed.

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