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The EEG in Three Cases of Periodic Catatonia

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As a result of three decades of painstaking work, R. Gjessing (1932-1960) published ten classical *Beiträge zur Somatologie der periodischen Katatonie*. These studies are being continued by L. R. Gjessing (1956, 1958, 1964, 1965) on some of the original patients and on additional subjects.

The final monograph written by R. Gjessing and translated into English is in press. This book contains Gjessing's final conclusions and a detailed summary of his findings. In addition this work includes studies with his co-workers on the ECG (see also Kjaestad, 1942), autopsy findings (see also Cammermeyer, J., and Gjessing, R., 1951), reaction time studies, chronic infection studies, menstrual studies, and electrolyte metabolism in periodic catatonia.

No reports on the EEG studies of Gjessing's patients have been published. However, a number of serial studies were performed on three patients with periodic catatonia and these are of clear scientific and historical value. This paper therefore presents a few of the available findings; in one case including the work which continued after the death of R. Gjessing.

The EEG can probably throw very little light on the fundamental nature of a psychosis, it does however add information about the illness. The clearcut changes in periodic psychoses may also give insight into the nature of the alpha rhythm and the significance of its changes.

The literature on the EEG in periodic psychoses is summarized by Harding, Jeavons, Jenner, Drummond, Sheridan and Howells (1966). A summary of this literature is presented in Table I.

Material

The results are presented from the studies of three patients.

E.Lö. was born in 1912. He has been the subject of intense and repeated metabolic studies (Gjessing, 1964a and b, and 1965). His clinical condition is described in detail in a previous communication (Gjessing, 1964a). It can be summarized as follows: This patient has had very regular periods of catatonic stupor for years. Both the stupor and the interval last about 3–4 weeks. The onset of the stupor is very sudden with an increase of the pulse rate and the basal metabolic rate and with decreased sleeping time. The stupor is most profound in the first third of the catatonic phase, then it gradually disappears over a period of 2–3 weeks.

This patient had never had electroshock treatment nor had he been previously treated with psychotropic drugs. E.Lö. was subjected to repeated EEG studies over three periods, Series 1 from 10.4.56 to 4.6.56 when 32 records were made, Series 2 from 6.5.58 to 7.7.58 when 19 records were collected, and Series 3 from 5.11.62 to 28.3.63 when 62 records were taken. From 1.3.63 to 1.4.63 he received phenelzine sulphate (for days 1 & 2, 3 & 4, 5 & 6, and 7 onwards, 30, 60, 90 and 45 mg. respectively daily). Throughout these studies he was on an artificial and completely controlled diet (H Diet, Gjessing, 1953).

The second patient, B.Gu. was born in 1927; her case has been presented by Gjessing (1958). During the period of study she was amenorrhoeic. Her story can be summarized as follows: Since 1946 she had been ill with periods of catatonic stupor lasting from 13 to 30 days but most often 18 days. These phases were separated by intervals lasting 17 to 23 days. The psychotic phases were independent of the menstrual cycle. Before she was admitted to Dikemark Hospital in August 1952 she had received over a few years 50 electroshock treatments. One series of EEG studies on B.Gu. are presented. They include 13 records taken from 4.11.52 to 2.12.52, while she received the H Diet and no drugs.

The third patient, K.Jo., was born in 1886, and his case has been described by Gjessing (1953).

It can be summarized as follows: This patient had regular phases of periodic catatonic excitement lasting about two weeks, separated by intervals of nearly normal behaviour also lasting approximately two weeks. Maximum excitement occurred in the middle of the psychotic phase. His urinary excretion of 4-hydroxy-3-methoxymandelic acid (VMA) is elevated in each psychotic phase.

This patient had never had electroshock treatment or psychotropic drugs before he was given reserpine. One series of EEGs was taken from 6.4.56 to 24.5.56; the reserpine 5 mg. a day was started 7.4.56 and continued throughout the study.

In each series in this study the EEG was taken at first during an interval phase and throughout the ensuing stupor or excitement phase. In the third series of E.Lö. the serial recordings were continued after the stupor phase and through a period of loading with the monoaminoxidase inhibitor. In the series of K.Jo. all EEG recordings except the first were taken when the patient was given 5 mg. of reserpine a day.

EEG Technique

The recordings which were available were bipolar studies made on a six-channel Grass machine (1948). Small button electrodes were used, using one of the earlier Maudsley patterns. The EEG recording procedures had been so standardized that records were easily comparable. Each series of recordings was examined visually by one of us but without any knowledge of the patient's mental state or the concomitant biochemical findings. The visual assessment was of the slow rhythms (theta amplitude in μVolt or classified abundance on a five-point scale), alpha rhythm, its frequency, amplitude and location, the beta rhythm (14–18 c/s, beta classified abundance on a three-point scale), low-voltage fast activity (20–30 c/s classified abundance on a five-point scale), and the response to overbreathing and to photic stimulation (classified flicker burst response). The units used were arbitrary except for alpha frequency and amplitude and theta amplitude.

One of the other authors, quite independently and also without knowledge of the clinical state or biochemical results, carried out the following simple quantitative techniques. Measurements were made of the activity in right parieto-occipital derivation of the parasagittal montage. The starting point was 15 cm. (5 seconds) after the first eye closure. Scores were derived from the subsequent 30 cm. (10 seconds) of the record. On six records the above was not realistic, as the patient opened his eyes, or the artefact at the time was such as to make quantification quite meaningless. In these circumstances the last ten seconds of the montage was studied.

The following scores were derived from the above periods.

(1) The Mean Dominant Frequency. The number of cycles in the ten-second period were counted and divided by ten.

(2) The Amplitude Count. The number of peaks or troughs falling outside a 50 μVolt (peak to peak) band during the first two seconds of the ten-second period. The band used was the band best fitted to the data.

(3) Eye Movements. The number of eye movement potentials occurring in the frontal derivation during the ten-second period were counted. It should be noted that this was done when the eyes were closed. This was studied as a possibly useful behaviour measure correlating with clinical descriptions of the patient staring vacantly ahead.

The response to photic stimulation was assessed. It was noted that as part of the study short bursts of flashes at approximately 14 c/s had been given for periods of half a second every second. The degree to which this produced a slow wave response of 2 c/s was rated on an arbitrary four-point scale. The study was limited and continues into the period of medication. The results are, however, presented, as they are part of the study and suggest a different response in stupor and during the interval.

Biochemical Methods

The phenolic amines were studied by the method of Kakimoto and Armstrong (1962), phenolic acids by the method of Armstrong, Shaw and Wall (1956).

Abbreviations: VMA: 4-hydroxy-3-methoxy

mandelic acid; NMN: normetadrenaline; MN: metadrenaline; MAOI: monoaminoxidase inhibitor; BMR: basal metabolic rate.

Results

Figs. 1 and 2 show some of the primary EEG records from which the studies were made. Fig. 1 contains six records from Series 3 of E.Lö. and shows the changes from the interval to the stupor phase. Fig. 2 shows two records each from the three patients, B.Gu., K.Jo. and E.Lö. and contrasts their interval and reaction phases.

Figs. 3 and 4 show the results of the EEG studies and the psychic state, BMR and pulse rate changes studied simultaneously in E.Lö. The very clear correlation between the changes in all the factors studied can be seen. In particular in the stupor phase the alpha frequency, the mean dominant frequency, the beta classified abundance, the low voltage fast activity, the BMR and the pulse rate increase as the alpha amplitude, the amplitude count and the theta abundance drop. Figure 4 also includes the excretion of VMA. It can be seen that the shape of the curve for VMA excretion is like that of the alpha rhythm changes, it rises smoothly to a peak in the middle of the fully developed stupor. The pulse rate and BMR jump quickly to their highest values at the onset of the stupor and gradually decrease thereafter. Fig. 5 shows the MN, the NMN and VMA excretion together with the alpha frequency and the mean dominant frequency. It can be clearly seen that the curve for MN is more like that of the BMR and pulse rate than is the curve of NMN excretion. The latter correlates more strikingly with the changing depth of the stupor and the alpha rhythm. This quantitative relationship may be of special significance.

The eye movements count during the study of E.Lö. shows the efficiency of this score as a behavioural measure of the patient's state.

The alpha location is presented, but this is probably another score of alpha amplitude. When the alpha amplitude is low, that which can usually be recognized in the centro-parietal derivations cannot be seen visually.

In Series 3 of E.Lö. (Fig. 4) it should be specially noted that for some reason the interval phase was longer than usual, and after the stupor phase the alpha frequency did not quite return to his usual interval level before he was given the monoaminoxidase inhibitor. It is particularly interesting to note that during this period with monoaminoxidase inhibitor the alpha scores and mean dominant frequency scores also remain somewhat elevated. The large rise in the mean dominant frequency on 1 March, 1963 is in addition very clearly related in time to the onset of the administration of the drug. Nevertheless, the onset of the next stupor phase is again clearly seen in the EEG changes at the end of this series of recordings.

Fig. 4 also includes the results of the study of the responses to bursts of light flashes when this was performed, and though the results are from too short a period they are similar to those reported by Jenner and Merskey (1963) in another periodic psychotic patient.

Fig. 6 shows the results of the studies on B.Gu. and K.Jo. The results are less striking than those from E.Lö. but again in B.Gu. the alpha frequency change can be seen to change with the mood. This was first noted by N. B. Johannessen in 1952 but not reported. In this study (B.Gu.) the changes of the mean dominant frequency, beta abundance, and low voltage fast activity seem to precede the mood changes. The study is, however, too short to assess the significance of such changes.

The changes in K.Jo. (see Fig. 6) are even less striking than those from B.Gu. This is possibly due to the fact that the patient received reserpine, which partially blocks the sympathetic nervous system. The mood change was also very slight.

Discussion

Table I shows a summary of the literature on the EEG changes found in studies of periodic psychoses. That changes occur in the alpha rhythm and other factors in the EEG and that these correlate well with the changes in mental state of an individual is clear. It is, however, more difficult to predict the type of change. The problem is complicated by diagnostic fashions and the terminology to be used for the various phases. If a patient has a clear interval phase of apparent health and a psychotic phase (reaction phase) contrasting with this, it might seem

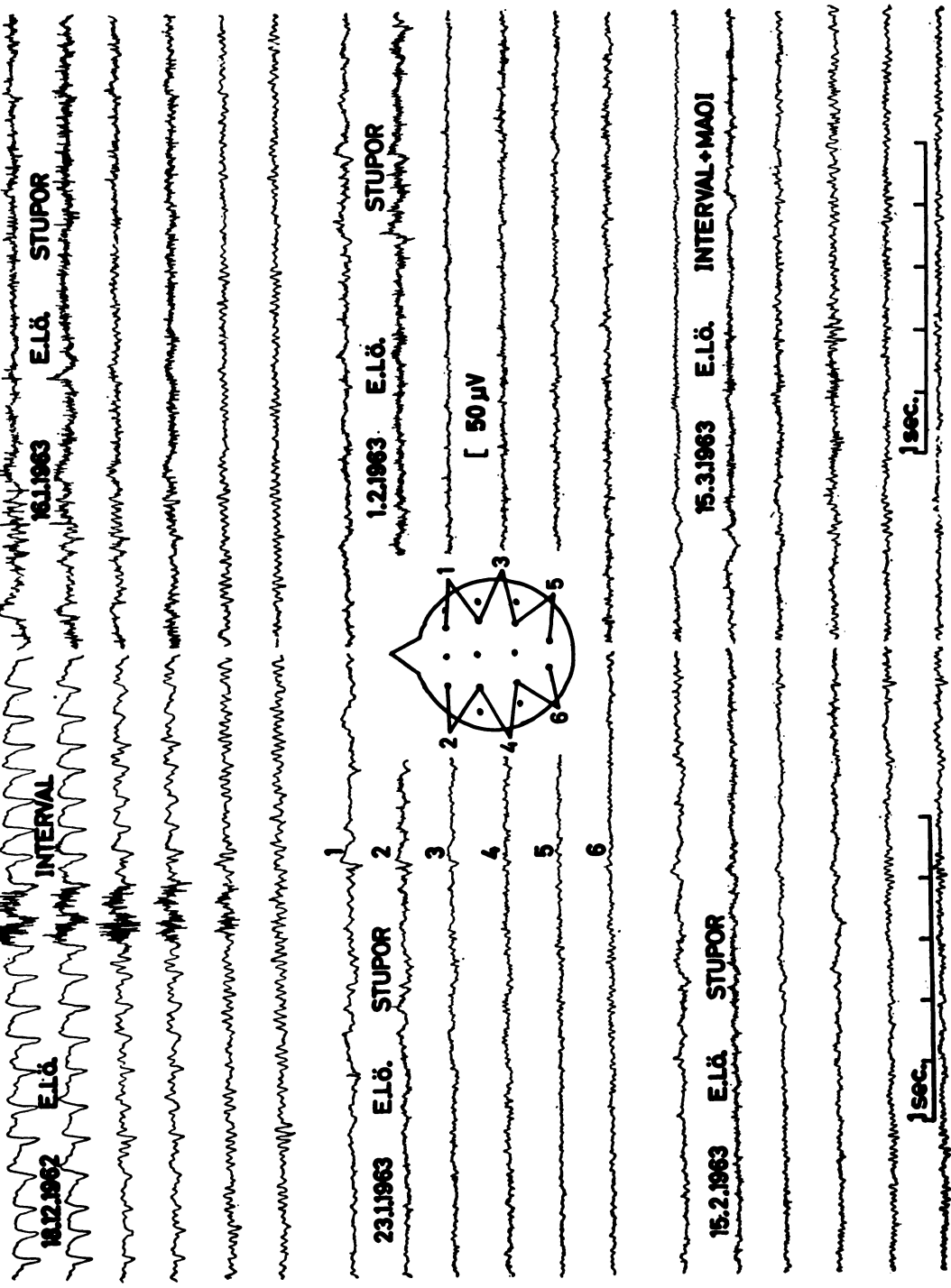

FIG. 1.—EEG recordings from E.L.Ö. 1962-1963. 18 December is in an interval. 16 and 23 January and 1 and 15 February are during a stupor (see Fig. 5). 15 March is in the subsequent interval, but when a monoaminoxidase inhibitor was being given.

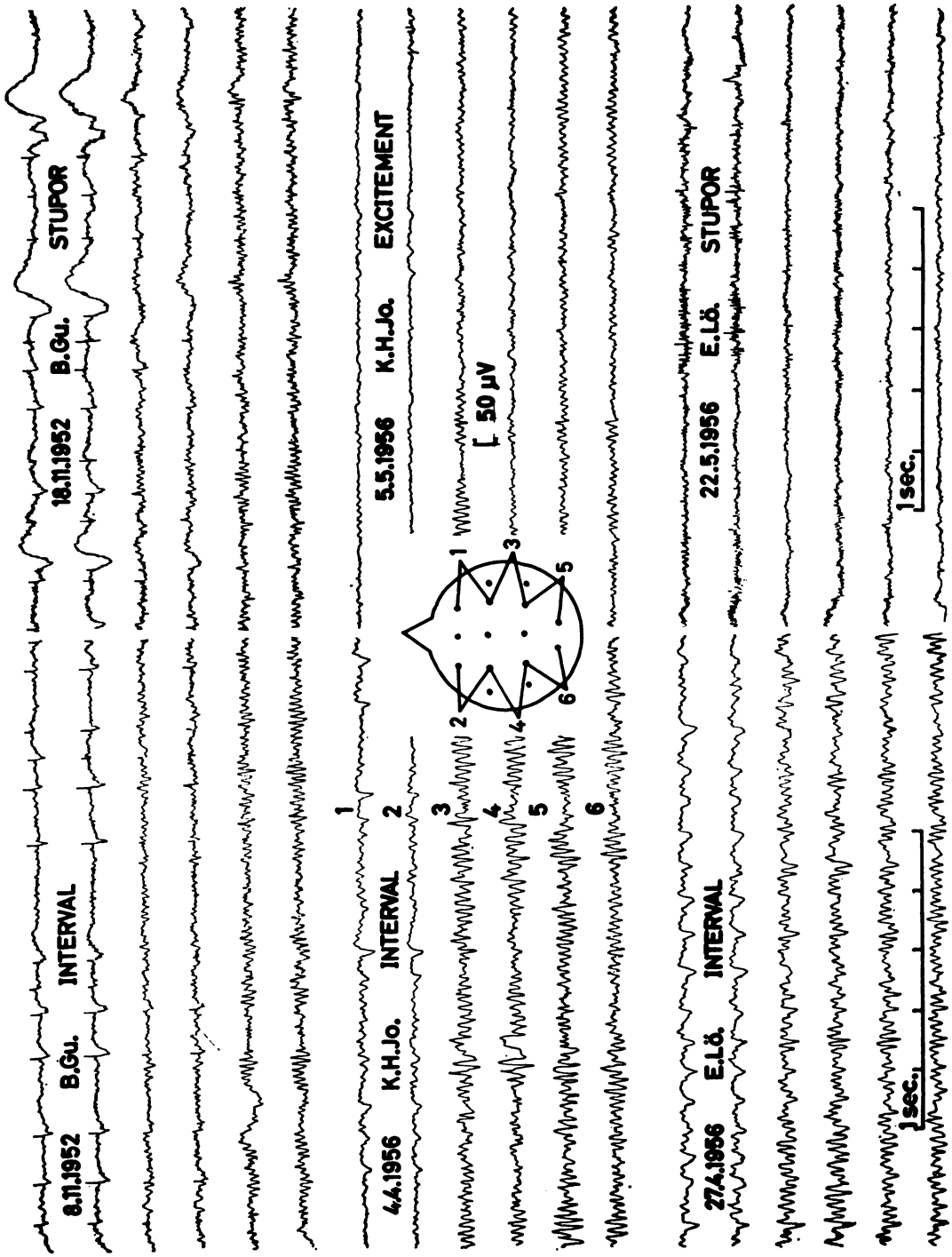

FIG. 2.—EEG recordings for all three patients from the interval and reaction phases.

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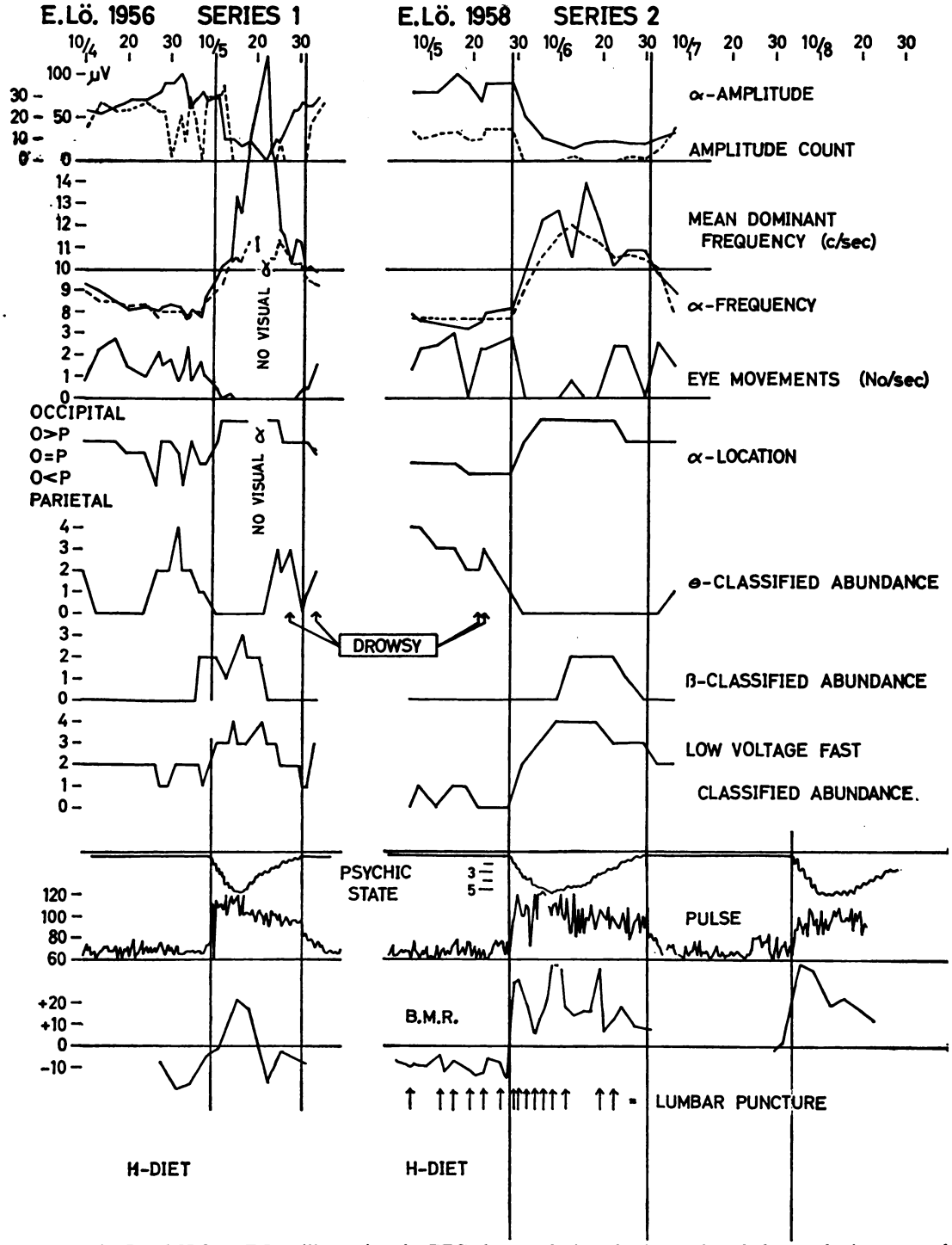

FIG. 3.—Series I and II from E.Lö., illustrating the EEG changes during the interval and the psychotic stupor, the mental state (1 to 5, 5 deep stupor) pulse rate and B.M.R. Alpha amplitude and mean dominant frequency ——, amplitude count and alpha frequency c/sec. – – – – .

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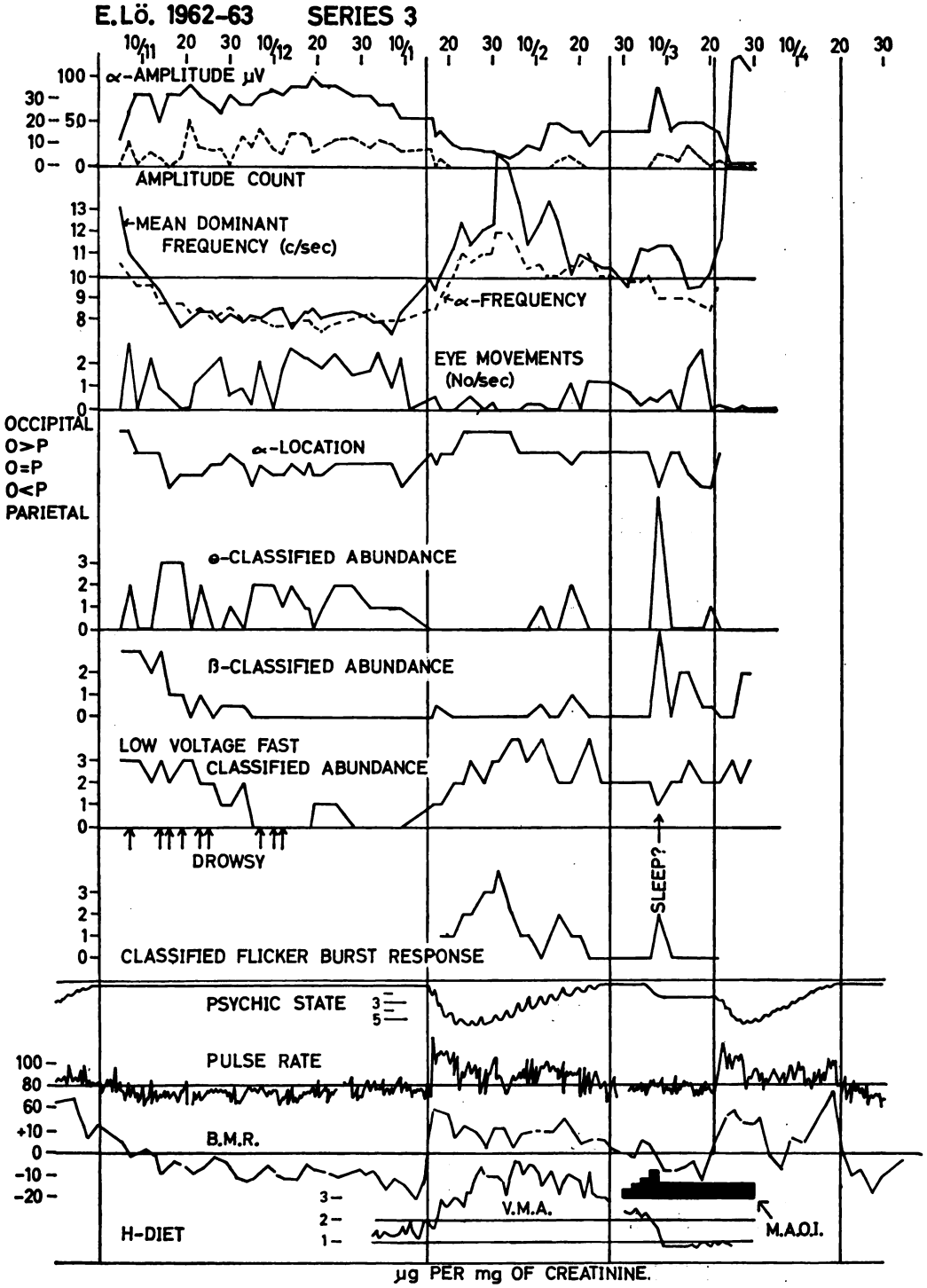

FIG. 4.—Series III from E.Lö. showing the EEG findings, the mental state, pulse rate, BMR and VMA during the interval and the stupor, and after a monoaminoxidase inhibitor. Alpha amplitude, and mean dominant frequency ———, amplitude count and alpha frequency– – – – –.

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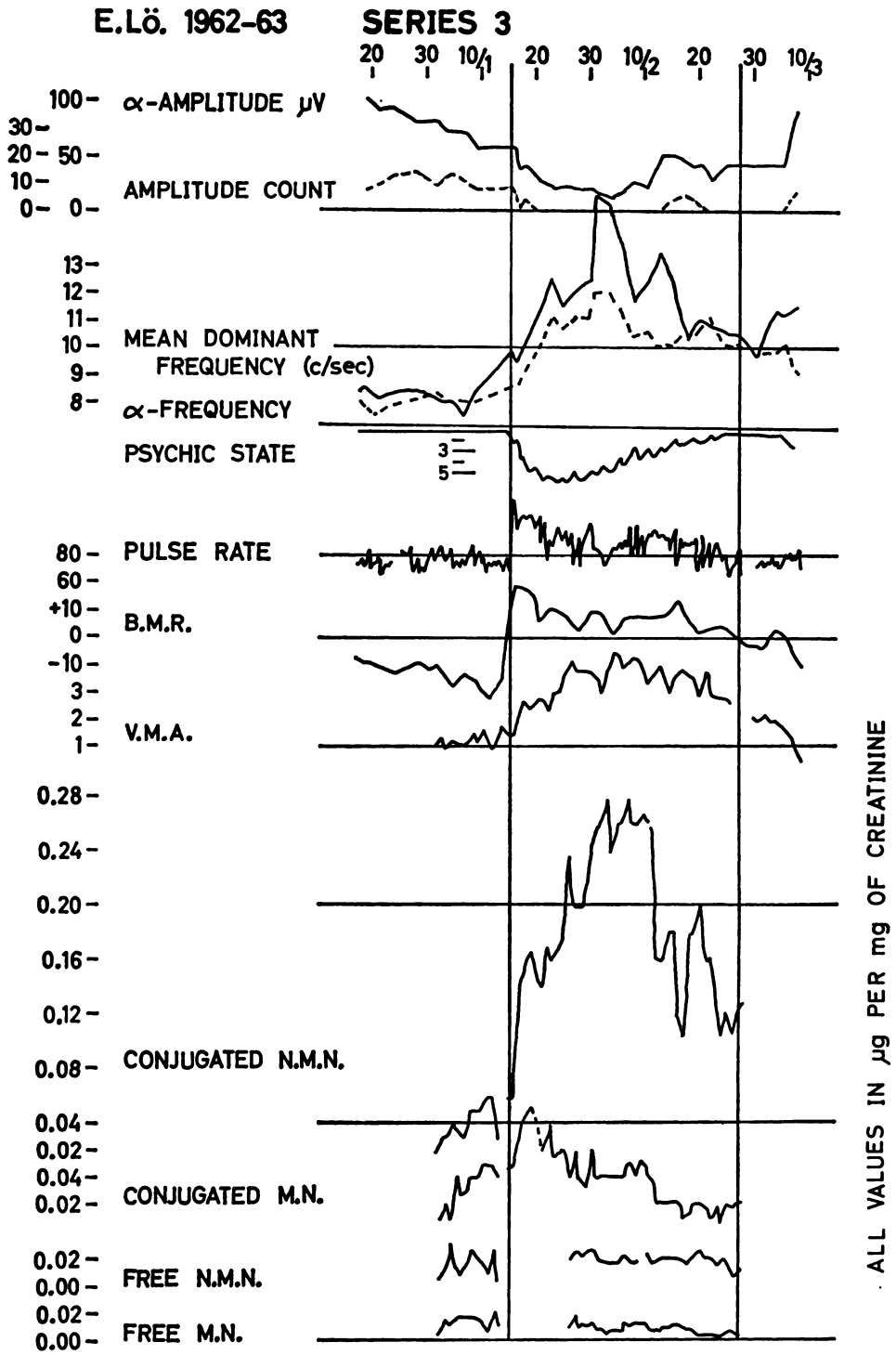

FIG. 5.—These curves illustrate the similarity between the mean dominant frequency on the one hand and excretion of VMA and NMN on the other hand, whereas the curve of the pulse and the BMR are more like the MN curve. Alpha amplitude, and mean dominant frequency ———, amplitude count and alpha frequency – – – – – –.

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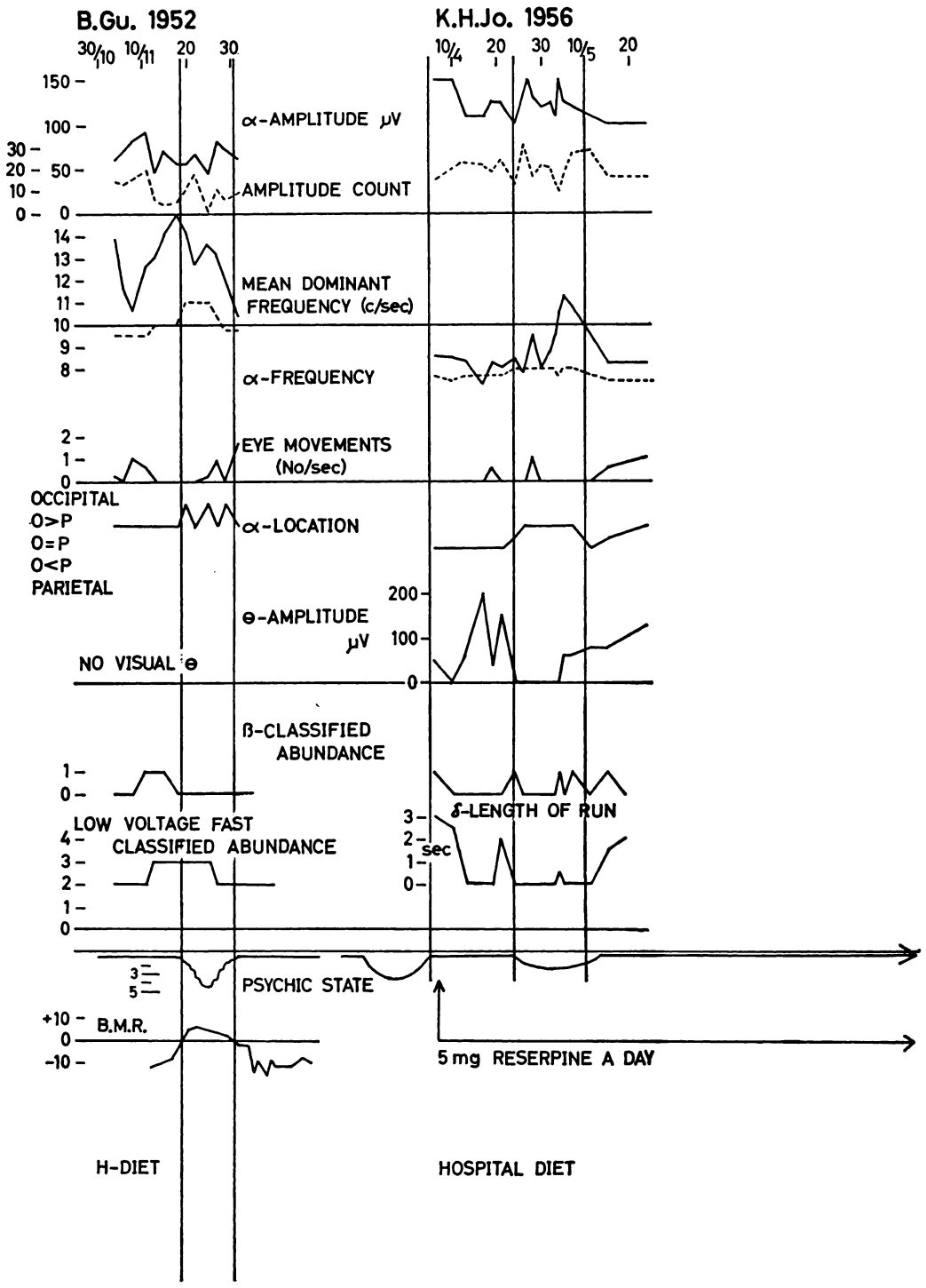

FIG. 6.—This figure gives the EEG findings from B.Gu. and K.Jo. with the mental state, and B.Gu.'s BMR. Alpha amplitude, and mean dominant frequency ———. Amplitude count and alpha frequency – – – –.

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appropriate to classify the EEG response in terms of these changes, whether the psychotic phase is one of excitement or of stupor. However, even if this method is used, Bonkalo *et al.* (1955) show that one of their patients has a decreased alpha rhythm frequency in catatonic stupor. When the diagnosis is nearer that of manic depressive psychoses, there can either be three (manic, depressed and normal phase), or only two phases in the cycle. It is then difficult to see how to compare their changes with the periodic catatonic, especially as Harding *et al.* (1966) also showed that one in three of their patients had the opposite changes from the other two. The subject clearly requires further clarification.

Quantification of the EEG in these and other patients is not always easy, and in their studies Rowntree and Kay (1952) and Hes (1960) do not comment on the alpha rhythm. Harding *et al.* (1966) show that in one of the three patients whom they reported it is impossible to detect the alpha changes visually, but quite clear from studies by the computer technique of Bailey and Harding (1966) that changes are occurring with the same period as those of the mental state. In this study more information might have been obtained had such methods been available for B.Gu. and K.Jo.

In the case of E.Lö. the results are so striking that the technique used may well be giving us most of the information available in the data.

Summary

One hundred and forty electroencephalograms from three periodic catatonic patients have been studied. They were taken by N. B. Johannessen during the metabolic studies of R. and L. Gjessing. A quantitative correlation

Table I

Authors	No. of Pts.	Diagnosis	Alpha	Other Comments
Anderson *et al.*, 1964	1	Manic depressive	9 cps α abundance decreased in mania	
Bonkalo *et al.*, 1955	1	Periodic excitement	α frequency increased and α amplitude decreased in excitement	
	2	Periodic stupor	1 α frequency increased. 1 α frequency decreased. 2 α amplitudes decreased in stupor	
Gunne and Holmberg, 1951	1	Periodic catatonia	α frequency increased. α amplitude decreased in attacks	Irregular slow in attacks
Hes, 1960	1	Manic depressive	No comments	Slow waves in mania
Harding *et al.*, 1966	3	Periodic psychosis	In 1 α frequency decreased when hyperactive. In 2 decreased α amplitude in mania with increased frequency	In 1 slow waves in depression
Rowntree and Kay, 1952	2	Recurrent schizophrenia	No comments	Increase in amount and decrease of frequency of slow in attack
Winnik and Assael, 1966	3	Manic depressives with recurrent depression	In one patient increased synchronization in depression. On the others no comment is made	In all three patients abnormal slow wave activity during depression

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between the mental state, the excretion of catecholamine metabolites, changes in the alpha rhythm and other factors in the EEG are presented and discussed. In particular the alpha frequency increases and its amplitude decreases during the recurring psychotic phases. The graph of the alpha frequency and mean dominant frequency has the same shape as the excretion of 4-hydroxy-3-methoxy-mandelic acid (VMA) and of normetadrenaline suggesting a possible relationship with sympathetic nervous system changes.

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