

CHANGES OF THE COMPUTERIZED EEG MAP IN VARIOUS FORMS OF EPILEPSY AFTER ADEQUATE THERAPY

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Introduction

In some previous papers (Popoviciu et al., 1981, 1982, 1983, 1984; Poliac, Popoviciu et al., 1981) we presented the original methods by which our research group had carried out for the first time in the world the computerized electroencephalographic map, a noninvasive method of high fidelity in collecting, processing, complying and temporo-spatially representing the electric cerebral activity in assembly images. In another work (Popoviciu et al., 1984) we reported the comparative results of the computerized EEG maps in 125 cases of epilepsy with various electro-clinical forms. In the present work, using this method which supplies a tridimensional spatial representation by means of a mathematical model built in a Felix C-256 Universal Computer or a M-118 micro-computer we proposed to study, in various forms of focal epilepsy and Petit Mal epilepsy, the dimensions of the epileptic foci, as well as their changes under the influence of the applied antiepileptic treatment.

Material and Method

By the values collected from the Siemens-Elma Mingograph provided with a Quantifizier System 400, we studied the changes obtained under the influence of the anti-epileptic treatment upon one of Hjorth's three parameters, namely upon Activity (A), i.e. the amplitude of the cortical electric activity in a group of patients selected clinically and EEG-ally to have various forms of focal epilepsy and Petit Mal epilepsy.

The values of the amplitude of the cerebral bioelectric activity have been introduced, by means of a mathematical model, first in a Felix C-256 Universal Computer and then in the M-118 micro-computer in order to elaborate the computerized electroencephalographic maps of the above mentioned forms of epilepsy.

The EEG map of activity reflected the spatial setting-out of the amplitude of the EEG signal at the level of the cortex and evidenced by means of the contour levels both the active zones and their level of activity as well.

In all the examined cases we used 12 levels of resolution in order to assure a finer comparative analysis. There has been changed only the value in μV of the variation interval of a resolution level because it depends on the maximum and minimum values of activity in the given computerized electro-encephalographical recordings.

For each studied case there were carried out three electro-encephalographic maps for each recording corresponding to the three montages

used to collect the data: a bipolar longitudinal montage for Routine 1 and two unipolar montages of the focal type ("source derivation") for Routine 7 and 8, according to the method introduced by Berglund and Hjorth (1976) and improved by Popoviciu et al. (1982), (Fig. 1).

Results and Discussions

In the cases with temporal epileptic attacks a bidrug therapy (with Carbamazepine and Diphenylhydantoin) was used, for those with fronto-central focal attacks a bidrug therapy with Phenobarbital and Diphenylhydantoin was used, while in the cases with polymorphous attacks initially Carbamazepine and Diphenylhydantoin were administered, and where there were no results the therapy was changed associating Carbamazepine with Primidone. In the cases with Petit Mal attacks succinimides (Ethosuccinimide or Methsuccinimide) were administered, depending on the electroclinical form.

The obtained results are exemplified with some of the cases:

1) M.P. case, 17 years old, diagnosis. Temporal epilepsy. There were

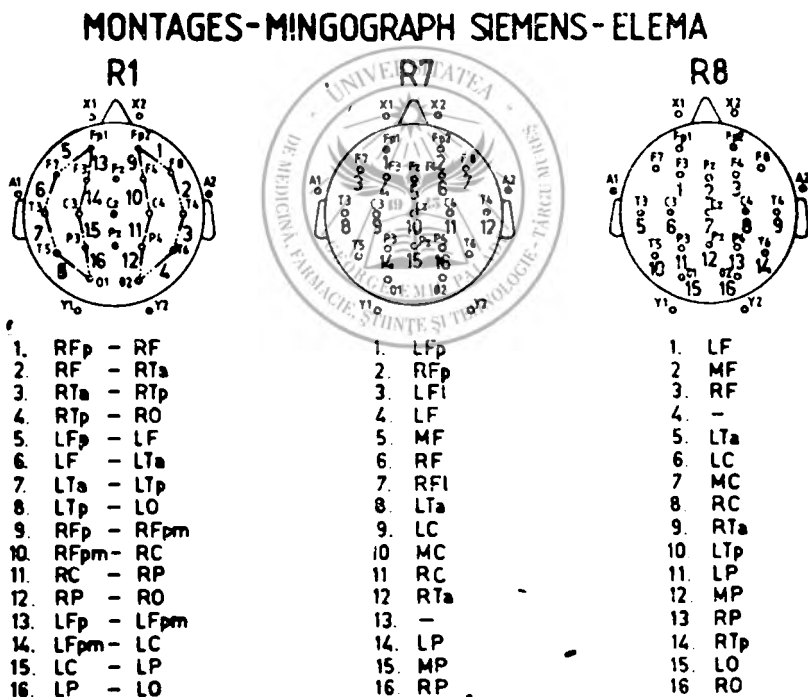


Fig. 1: The montages used on the Siemens Elema Mingograph. Routine 1: bipolar longitudinal montage; Routine 7: anterior unipolar montage; Routine 8: posterior unipolar montage

performed two EEG recordings at an interval of 3 months used to carry out the EEG maps. The first recording is that of February 2, 1982.

In R_1 : left posterior temporal focus with $A = 248 \mu\text{V}$ (in C8). Besides this there were evidenced: a left frontal focus ($A = 208 \mu\text{V}$) and another one, right posterior temporal ($A = 188 \mu\text{V}$), (Fig. 2).

In R_7 : In the same case the computerized EEG evidences a bilateral frontal focus, ampler on the left ($A = 300 \mu\text{V}$).

In R_8 : In the same case and in the same recording one can see a left posterior temporal focus ($A = 392 \mu\text{V}$) and a right posterior temporal one ($A = 296 \mu\text{V}$).

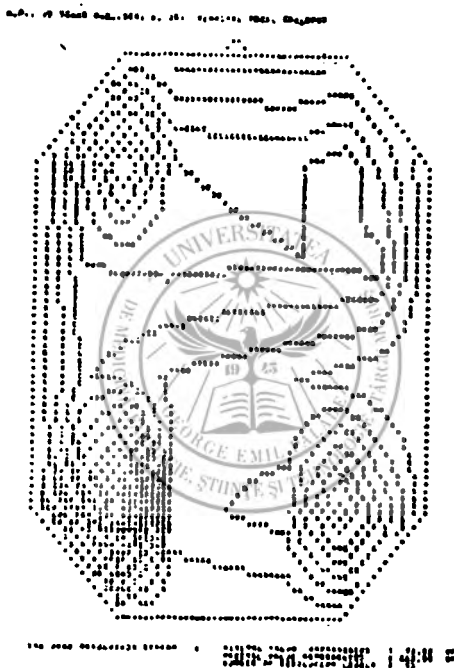


Fig. 2: M.P. case, 17 years old, Dg.: temporalepilepsy. The computerized EEG map in R_1 . Left posterior temporal focus with $A = 234 \mu\text{V}$. First recording.

The second recording after three months, on May 26, 1982: In R_1 : the highest value of Activity was of $148 \mu\text{V}$ in the right anterior temporal (Fig. 3).

There was noted a reduction of the amplitudes of the cortical electric activity under the influence of the applied treatment. (with Carbamazepine and Diphenyldantoin).

2) M.M. case, 19 years old; diagnosis: Polymorphous epileptic attacks (false absences, psychomotor seizures).

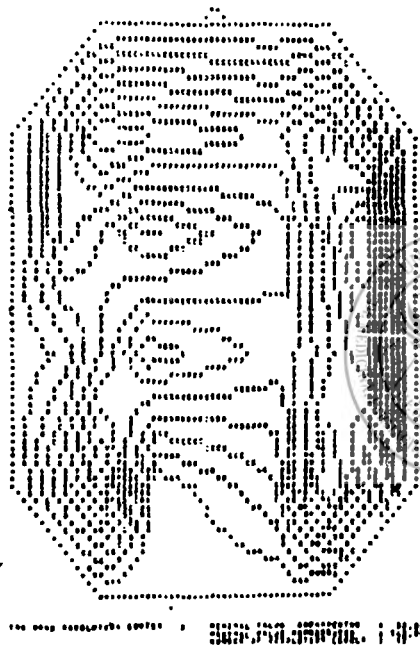


Fig. 3: The same case. The second recording after three months. The computerized EEG map in R. There is evidenced a very extended right fronto-central temporal focus

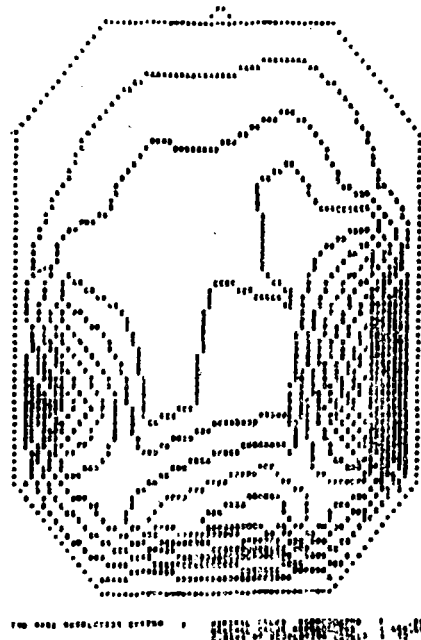
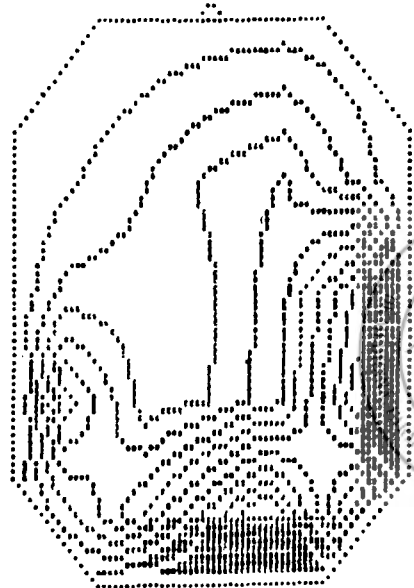


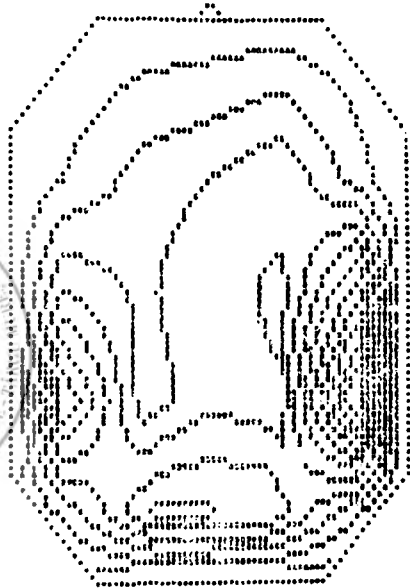
Fig. 4: M.M. case, aged 19 years, Dg.: polymorphous epileptic attacks. The computerized EEG map in R. First recording. A high amplitude right posterior temporal focus of $A = 488 \mu V$





THE EEG AMPLITUDE SCALE IN μ V. 100

Fig. 5: The same case. Second recording. The computerized EEG map of activity in R_3 . A high activity right parieto-temporal focus of $A = 856 \mu V$



THE EEG AMPLITUDE SCALE IN μ V. 100

Fig. 6: T.I. case, 28 years old, Dg.: frontal and temporal epilepsy. Second recording. The computerized EEG map of amplitude in R_3 . There is evidenced a clear cut right frontal temporal focus



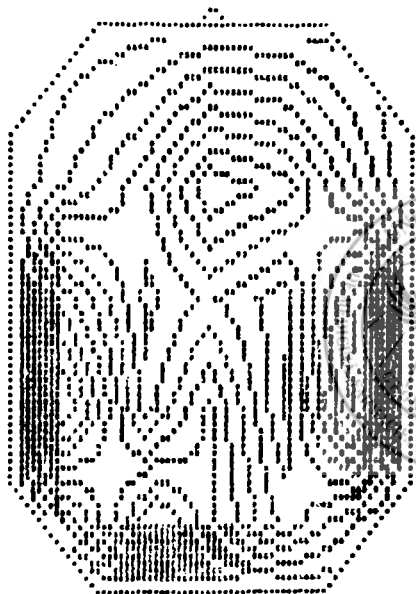


FIG. 7: D.A. case, 19 years old, Dg.: Petit Mal absence and myoclonic P.M. The computerized EEG map of Activity in R₃. Right posterior temporal which is extended mirrorlike on the left posterior temporal, too.

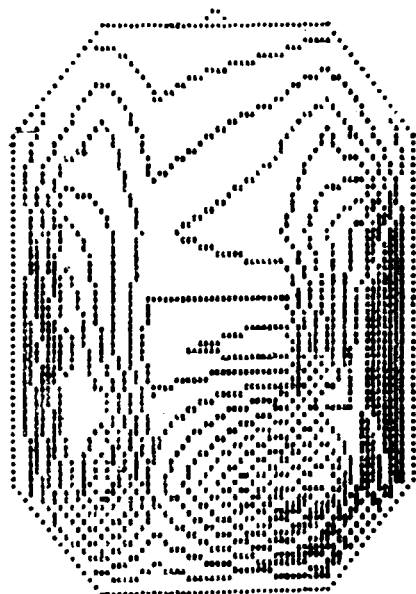


FIG. 8: The same case. The eighth recording. The computerized EEG map of Activity in R₁. There is evidenced a clear cut right posterior temporal focus, extended enough but of small amplitude: 88 μV

Fig. 7: D.A. case, 19 years old, Dg.: Petit Mal absence and myoclonic P.M. The computerized EEG map of Activity in R₃. Right posterior temporal which is extended mirrorlike on the left posterior temporal, too.

Fig. 8: The same case. The eighth recording. The computerized EEG map of Activity in R₁. There is evidenced a clear cut right posterior temporal focus, extended enough but of small amplitude: 88 μV

There have been performed two EEG recordings at an interval of approximately six months and the respective EEG maps as well.

The first recording on July 16, 1982

In R₁: a right posterior temporal focus ($A = 352 \mu\text{V}$) is evidenced;

In R₇: a right lateral frontal focus ($A = 248 \mu\text{V}$) is evidenced;

In R₈: one can see a right posterior temporal focus with a high amplitude ($A = 488 \mu\text{V}$), (Fig. 4).

The second recording on December 6, 1982: *In R₁*: a large right parieto-temporal focus is evidenced with $A = 600\text{--}520 \mu\text{V}$, as well as a left posterior temporal one of $400 \mu\text{V}$, while *in R₈* one can see the same right parieto-temporal focus extending towards the right occipital zones and having an $A = 856 \mu\text{V}$ (Fig. 5).

This case presented neither clinically nor electroencephalographically any improvement under the treatment with Carbamazepine and Diphenylhydantoin, on the contrary it got worse. After the second recording the treatment with Finlepsin was changed to Primidone.

3) *T. I. case*, aged 28 and diagnosis of focal temporal and frontal epilepsy. There were made 5 EEG recordings followed by the fulfilment of the respective EEG maps. The first recording made on April 5, 1982. *In R₁* there was a right posterior temporal focus of low amplitude, $A = 152 \mu\text{V}$.

At the second recording on May 31, 1982 there is also a right posterior temporal focus *in R₁*, as well as a right frontocentral one, $A = 104 \mu\text{V}$. At hyperpnoea, *in R₈* the right posterior temporal focus became more evident: $A = 284 \mu\text{V}$ (Fig. 6).

At the fourth recording on September 13, 1982 (during treatment with Carbamazepine and Diphenylhydantoin), at hyperpnoea there are *in R₈* a focus also in the right posterior temporal and having an $A = 124 \mu\text{V}$, as well as a mirrored one on the left posterior temporal region. The frontal focus could be evidenced only on one of the 5 recordings by the means of the EEG map, the maximum value at hyperpnoea being of $128 \mu\text{V}$ on the left fronto-polar region. Otherwise, clinically also, the focal temporal attacks were more frequent than those focal frontal.

4) *D. A. case*, 19 years old, diagnosis: Myoclonic Petit Mal and Petit Mal Absence. There were performed 8 recordings followed by the fulfilment of the respective EEG maps. In the first recording, there could be found on the EEG tracing discharges of spike and waves and poly-spikes and waves of 3/sec which were bilateral, synchronous and quasi-synchronous. The EEG map in *R₈* evidenced a left posterior temporal focus of $A = 280 \mu\text{V}$ and at hyperpnoea a right one of $A = 268 \mu\text{V}$ (Fig. 7).

For the other recordings the EEG tracing and the EEG maps changed only slightly.

The last recording made after 6 months from the first one (during treatment with Methsuccinimide) the EEG tracing is greatly improved, slightly slower and having more frequent theta waves while the EEG map in *R₁* evidenced a right posterior temporal focus of $A = 88 \mu\text{V}$ (Fig. 8).

Conclusions

The dynamic studies of the epileptic foci in various formes of epilepsy investigated by us have evidenced the presence of multiple epileptogenic foci which cannot be detected on the standard EEG recordings demonstrating the usefulness of the computerized EEG map as method of electro-clinical diagnosis in epilepsy.

We recommend the fulfilment of the EEG maps in the cases of resistant to the applied therapy in order to be able to detect the fitted treatment.

The EEG map provides an immediate image about the way in which the applied anti-epileptic treatment influences the epileptic foci by reducing the maximum values of Activity (amplitude) in them towards normal values of cerebral bioelectric activity. Then there is no such influence the computerized EEG map translates the stability of the pre-exitent image.

References

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