



**NATIONAL UNIVERSITY OF SCIENCE AND TECHNOLOGY
POLITEHNICA BUCUREȘTI**

DOCTORAL SCHOOL OF ELECTRICAL ENGINEERING



EXTENDED SUMMARY DOCTORAL THESIS

CONTRIBUTIONS TO THE ANALYSIS OF BIOELECTRICAL SIGNALS IN NEUROCORTICAL ACTIVITY, IN ORDER TO IMPROVE USEFUL INFORMATION IN MEDICAL DIAGNOSIS

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Chapter 1 – Introduction

The work is based on an original experiment of analysis of neuro-cortical signals, using the analysis of EEG electroencephalographic signals and the visual evoked potential of PEV, to support the diagnosis in neurological and neuropsychiatric pathologies. The relationships between the parameters of neuronal activity and the processing of visual information are investigated, by comparing normal and pathological data.

1.1 Presentation of the doctoral thesis

Experimental data (EEG and PEV: amplitudes, frequencies, latencies, power spectra, etc.) are obtained in the laboratory and processed with specialized instruments. The results are presented through graphs, EEG maps and PEV representations, being analyzed to highlight the correlations between normal and pathological brain activity. The main objective is to identify new relationships and types of brain electrical activity relevant to diagnosis and treatment.

1.2 Purpose of the doctoral thesis

The theme is based on the author's experience in neurophysiology. The work aims to develop innovative EEG and PEV analysis methods to improve the diagnosis and highlight neurological and neuropsychiatric pathologies. For this purpose, technical methods of electrical signal processing and analysis are used.

The theoretical foundations of electrical signals and statistical methods applied to experimental data are presented. The results demonstrate the potential of these methods in identifying brain pathologies and developing useful applications in medical practice.

1.3 Content of the doctoral thesis

The paper analyzes the generation of the electrical signal under the action of a stimulus, its transmission and processing in the neural structures involved, with emphasis on the visual system.

Phenomena recorded at rest and in flash visual stimulation are described, presented by statistical parameters, tables, 2D/3D graphs, cortical maps and correlation diagrams.

Images of the experimental process and graphs highlighting new pathology indicators developed by the author are included.

The paper contains chapters dedicated to the neurophysiological correlative activity for the categories "Normal" and "Pathological-Clinical", presenting links between the observed electrical phenomena and the indicators of classification in various pathologies. The study targets a selected set of neurological and neuropsychiatric pathologies with a major impact on medical practice.

Chapter 2 – The Electrical Phenomenon – The Basis of Neuronal Activity (Synthesized Version)

Neocortical physiological activity is based on the electrical processes of the neuronal system, the foundation of central information processing. This is generated by the unequal distribution of loads at the level of the neuronal membrane and the excitability property, manifested by depolarization and electrogenesis. Electrogenesis produces the neuronal electric field, propagated on the cell membrane and described by theoretical models that highlight the complexity of electrical processing.

2.1 Introduction to the study of neocortical electrical activity

Cortical electrical activity comes from the electric fields generated by the reverberation of excitation between the cortical layers, especially II–IV of the visual cortex. It is described by the zonal dipole model "Source Dipole" and the "Central Source Dipole" (C.S.D.) model. [25] Keith H. Chiappa.

Electrical activity has a dominant role over biochemical activity because:

- can activate structures at a distance through the electric field;
- initiates propagable signals;
- generates local or distant biochemical changes;
- supports the integration of information into association and memorization circuits;
- it takes place much faster than biochemical processes;
- can induce excitation, deexcitation or cortical inhibition;
- transforms statistical biochemical phenomena into deterministic neuronal processes;
- Globally, it remains statistical due to the large number of neurons involved.

The electrical phenomenon is present at all biological levels, from receptors and axons to associative multisensory structures, which integrate signals from different analyzers.

2.2 The phenomenon of electric signal propagation at the level of the axonal membrane

2.2.1 Local electrical potentials

Local potentials are transient changes in transmembrane tension generated by a depolarizing agent. They are extinguished if the stimulus does not exceed the threshold of electrical depolarization of the cell membrane, and the electrotonic transmembrane voltage decreases exponentially, being characterized by the spatial constant (λ) and the time constant (τ).

Main properties of local potentials:

- they do not produce complete depolarization;
- se transmit decremental;
- have graduated amplitude;
- has latency;
- allow spatial and temporal integration;
- depend on membrane conductances and capacities;
- they can trigger the action potential if they exceed the threshold;
- are influenced by metabolism and membrane condition.

Mathematical relationships describe the propagation of membrane tension, the exponential decrease of electrotonic potential, and the constants λ and τ , which characterize spatial and temporal attenuation.

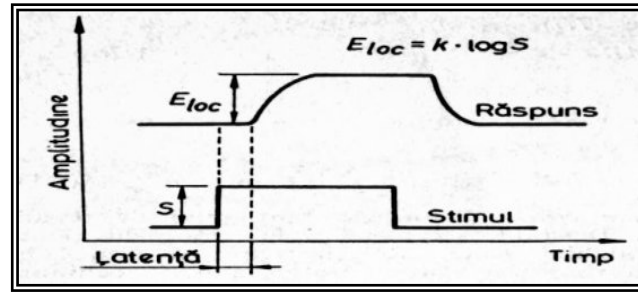


Fig 2.1 Characteristics of the local membrane potential caused by a rectangular electrical stimulus according to D.Mărgineanu and

The partial derivative equation showing the distribution in space and time of the UM transmembrane tension for the excitable cell-neuron, highlighting the constant of space (ec.2.4) and time (ec.2.8) is [1],[2]:

$$-\lambda^2 \frac{\partial^2 U_M}{\partial x^2} + \tau \frac{\partial U_M}{\partial t} + U_M = 0 \quad (2.1)$$

The simple solution of membrane tension is

$$U_M(x) = U_M(0)e^{-\frac{x}{\lambda}} \quad (2.3)$$

The electrotonic potential will decrease exponentially by e times over a distance equal to:

$$\lambda = \sqrt{R_M / (R_i + R_e)} \quad (2.4)$$

λ is a coefficient called *the spatial constant or length constant*, or spatial attenuation

$$\lambda \approx \sqrt{\frac{R_M}{R_i}} = \sqrt{\frac{r_M d}{4r_i}} \quad (2.7)$$

The term

$$\tau = R_M C_M \quad (2.8)$$

represents the time constant of the membrane, i.e. the time after the application of the stimulus at which the electrotonic potential decreases e times compared to the maximum value.

2.2.2 Action potential or depolarization impulse

The action potential is an electrical signal resulting from the depolarization and repolarization of the neuronal membrane, arising from the application of a supraliminal stimulus. It unfolds rapidly, with distinct phases of ascension and descent, and constitutes the fundamental unit of the transmission of nervous information.

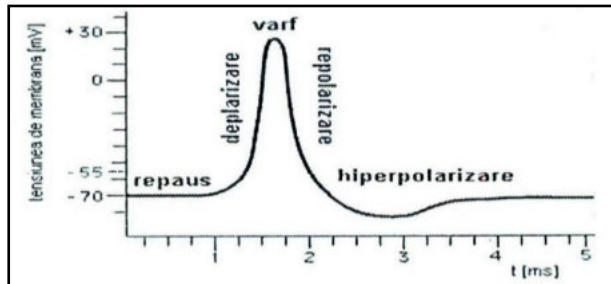


Fig 2.2 Depolarization Impulse or Action Potential - [5] Figure shows the stages of depolarization, repolarization, and electrical hyperpolarization of the excitable membrane after the stimulus is applied.

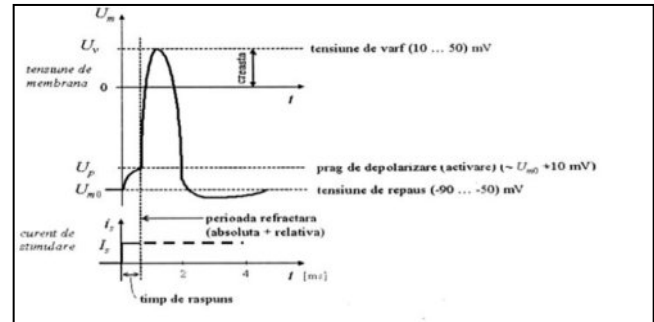


Fig 2.3 Depolarization Impulse or Action Potential at the Neuron – [5],[2]

The main characteristics of PA are:

1. It is triggered only at supraliminal stimuli, exceeding the depolarization threshold.
2. Comply with the "all or nothing" law: once initiated, the PA has a constant shape and amplitude.
3. It is fast and self-accelerated, with latency inversely proportional to the intensity of the stimulus.
4. It is irreversible and transient, returning to the resting value after a phase of hyperpolarization.
5. It has a short duration (1–3 ms) – for the neuronal axon.
6. It propagates over long distances, without decrement.
7. It is regenerative, the axon becoming fit again for a new BP after a short time.

2.2.4 Mathematical modeling of PA propagation

Action potential propagation (PA) can be described by the Hodgkin–Huxley equation, a differential equation expressing the variation of membrane tension in space and time. It is deduced from the model of independent transmembrane ionic currents and through its solutions, faithfully reproduces the experimentally observed PA. The differential equation of the variation of membrane tension in space and time is a relationship called **the Hodgkin–Huxley equation**

$$\frac{1}{v^2(R_i + R_e)} \cdot \frac{d^2 U_M}{dt^2} = C_M \cdot \frac{dU_M}{dt} + (E_K - U_M) \bar{g}_K n^4 + (E_{Na} - U_M) \bar{g}_{Na} m^3 h + (E_L - U_M) g_L \quad (2.1)$$

The differential equation of the variation of the membrane tension in space and time is deduced from the model of the transmembrane independent ionic currents, under conditions of fixed potential, but it describes very well by the calculated solution the experimentally recorded PA v.fig 2.5.

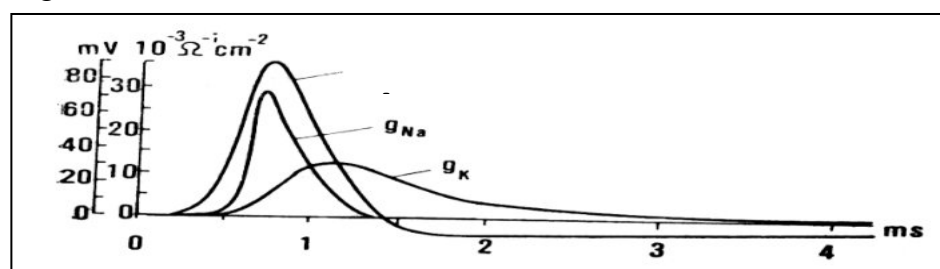


Fig 2.5 The solution of the Hodgkin-Huxley equation and the temporal dependence of the membrane potential and ion channel conductances of Na⁺ and K⁺ according to V Vasilescu & D Măroineanu. 1979. [5]

2.2.5 Propagation of action potential by non-myelinated axons

In non-myelinated axons, depolarization is transmitted by local electrotonic currents, which decrease exponentially with distance from the stimulation point. If the depolarization exceeds the excitability threshold, it extends to neighboring areas, generating action potential (PA) propagation. The propagation is unidirectional due to the existence of a refractory zone in the already depolarized region. Local currents have a maximum value at the stimulation site and decrease exponentially: \hat{I} is maximum

$$i(x) = Ie^{(-ax)} \quad (2.16)$$

The propagation speed depends on the axonal diameter and is low (1–2 m/s), insufficient for rapid transmission of information in large organisms. Non-myelinated axons are found in cortical gray matter, deep nuclei, and prechiasmatic segments of the optic nerve, prior to myelination. If the intensity of the action current (Hermann, see fig. 2.6) exceeds the reobase, it acts as an excitant for the neighboring region, causing the depolarization zone to shift by electric diffusion. PA propagation occurs in the sense that the membrane is at rest; in the opposite direction, the area is refractory by post-PA hyperpolarization (see fig. 2.7).

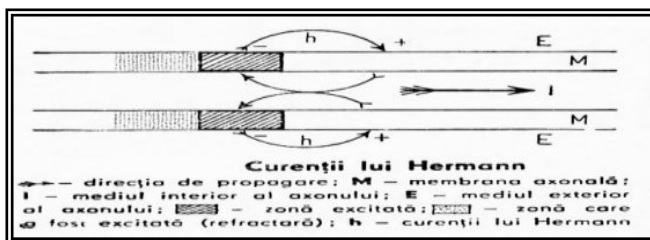


Fig 2.6 Hermann membrane currents - after Edmond Nicolau and Constantin Bălăceanu, 1967,[8]

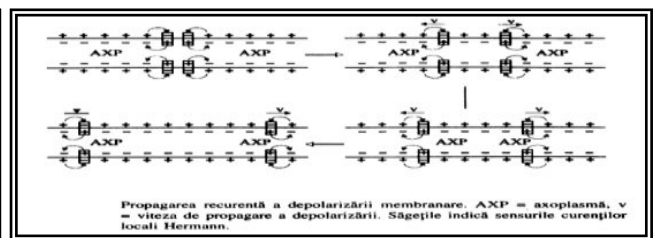


Fig 2.7 The meaning of the propagation of PA - according to A Popescu, 1994, [1].

For non-myelinated axons between the rate of propagation of PA per axon and the axonal diameter d :

$$v(t) \sim \sqrt{d} \quad (2.17)$$

The potential variation moves steadily along the axon, while on the somatodendritic membrane it is transmitted with decrement. In long dendrites there may be specialized regions that, when crossed by a PA, generate depolarizations of high amplitude and high propagation speed. Non-myelinated axons are found in the cortical gray matter, deep nuclei and in certain segments of the optic nerve, before myelin is formed.

2.2.6 Propagation of action potential by myelinated axons. The Frankenhauser and Huxley model

Most axons are myelinated, having an insulating sheath made up of lipid and protein layers produced by Schwann cells. The structure is segmented by the alternation of myelinated internodes and Ranvier nodes. PA transmission is achieved by jumping conduction: depolarization "jumps" from one Ranvier node to another, if the stimulation threshold is exceeded. Longitudinal currents (Stampfli) facilitate the rapid and efficient propagation of the signal, ensuring a much higher speed compared to non-myelinated axons.

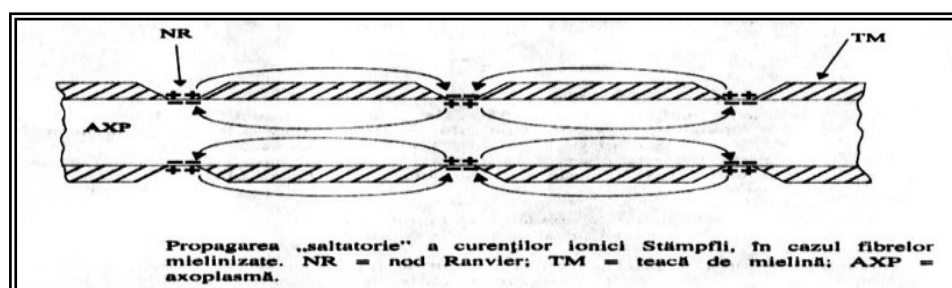
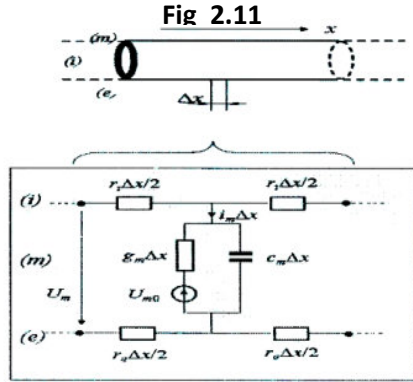


Fig 2.8 Jumping propagation of Stampfli currents - after A Popescu, 1994, [1]

2.2.7 Cylindrical Cell Model – Coaxial Cable Model

The axonal model is built on the basis of an equivalent circuit and a mathematical model that describes the displacement of depolarization along the membrane. The axon is characterized by the conductivities of the component media: intracellular ($\sigma_i \approx 1\text{--}2\text{ S/m}$), extracellular ($\sigma_e \approx 10\text{--}20\text{ S/m}$, about ten times greater than σ_i) and membrane (variable σ_m , depending on membrane selectivity). The model's assumptions include:

- axial symmetry and reduction of the problem to one dimension (1D);
- homogeneous but non-isotropic internal and external environments;
- equivalent circuit with distributed elements;
- the lack of inductive elements, which leads to an electric diffusion phenomenon, not to propagation in the form of waves. The model's assumptions include:



Thus, the transmission of depolarization is described as a process of successive diffusion along the axon. The electrical diagram of the membrane differs for the polarized and depolarized state, being associated with distinct mathematical expressions for the transmembrane current [2].

2.2.8 Equation of spatio-temporal distribution of membrane tension

The model uses transmembrane current and leads to partial diffusion-type differential equations [2].

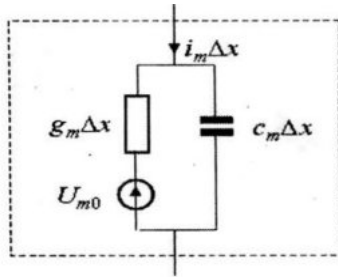


Fig 2.12 a
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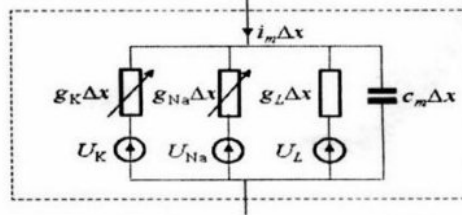


Fig 2.12 b
Modul
transversal
membrana
depolarizata

For the case of the electrically depolarized membrane v.fig.2.12.b, the diagram of the electrical circuit and the transmembrane current given by the expression b) are used.

- for the polarized membrane – linear equation;

$$a) i_m = (U_m - U_{m0})g_m + c_m \frac{\partial U_m}{\partial t} = u_m g_m + c_m \frac{\partial U_m}{\partial t} \text{ for polarized membrane -} \quad (2.22)$$

- for the depolarized membrane – a nonlinear equation with variable coefficients, numerically solvable by the Hodgkin–Huxley model.

$$b) i_m = (U_m - U_{Na})g_{Na} + (U_m - U_K)g_K + (U_m - U_L)g_L + c_m \frac{\partial U_m}{\partial t} \text{ for depolarized membrane} \quad (2.23)$$

Note: The models shown (made on an axon of squid or African frog, non-yelinated) cannot be applied directly to the human visual system, where the axons are in a high percentage myelinated. The necessary adjustments are made in relation to the Frankenhauser–Huxley model, applicable to myelinated axons, especially at the level of the Ranvier nodes. The models can be used in the non-yelinated axons in the prechiasmatic area, in the deep nuclei of the RF and in the complex neuronal structure of the hippocampus.

2.2.9 Adrian Edgar Douglas' Law

Adrian Edgar Douglas' law, formulated in 1923, describes the relationship between the intensity of nerve stimulation and the frequency of action potential (PA) discharges. According to this law, the frequency of impulses increases with the intensity of the stimulus, and at the threshold intensity (I_0) a minimum frequency of discharge is obtained. Experimental data show that if stimulation remains constant after a gradual increase, the frequency of impulses decreases exponentially over time. This phenomenon, called adaptation, reflects the restoration of the state of polarization of the axonal membrane towards the resting level.

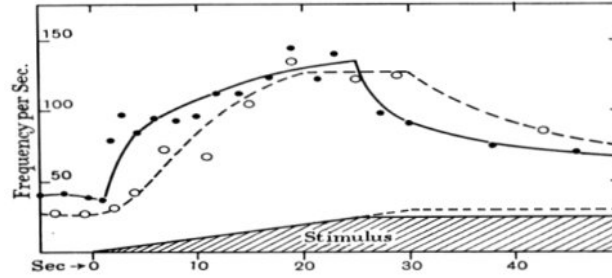


Fig 2.13 Gradual increase in the frequency of the electrical response when the needle is inserted into the skin. Adrian

The relationship is put in the form indicated by the experimental curve : $\nu = A \cdot \ln \frac{I}{I_0} + \nu_0$ or

$\nu - \nu_0 = A \cdot \ln \frac{I}{I_0}$, where I_0 and ν_0 are the intensity and frequency of PA discharges at the

threshold of stimulation, where $A = \frac{\nu_0}{\ln t_c}$. Relationship $\nu = A \cdot \ln \frac{I}{I_0} + \nu_0$ it is Adrian E,D 's

law.[9], (2.32)

Chapter 3 Experimental methods and techniques in obtaining the database for signal analysis and the stages of processing VEP and EEG signals

3.1 Purpose of conducting experiments

The experiments aim to collect and analyze EEG and VEP data for:

- obtaining databases in the state of rest and flash stimulation, both for normal and pathological cases;
- highlighting EEG and VEP activation patterns under normal and pathological conditions;
- analysis of EEG correlations between cortical areas and between activity frequencies;
- analysis of the correlations of the VEP between the components of the signal, at rest and in stimulation;
- identification of pathological patterns and obtaining specific EEG and VEP markers;
- Modeling of VEP activity under various flash stimulation conditions.

3.2 Electrical equipment involved in obtaining VEP and EEG

The assembly of devices forms a unitary system intended for visual stimulation, data collection, processing and storage for further analysis. The system includes:

- signal collection and flash stimulation device;
- electroencephalograph for primary signal pickup and processing;
- Digital Data Analysis System.

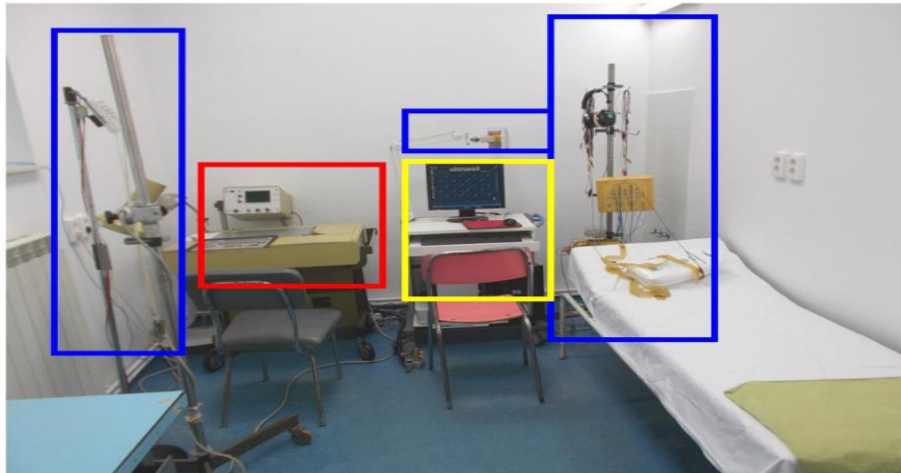


Fig 3.1 Electrical equipment involved in obtaining the EEG electrical activity and the PEV signal (with blue border - the system for collecting electrical signals and flash stimulation; with a red border - the electroencephalograph system; with yellow border – digital signal processing system – PC station) - Nicolae Stelian Stanciu. Neurophysiology Laboratory 2018

3.3 Visual Receiver Flash Stimulation System

Visual stimulation is performed with a white flash system (strobe), with standardized energy (0.3 J; 0.6 J; 1.2 J). The distance from the visual receiver is 25–30 cm. Scotopic stimulation is initially applied, followed by intermittent photopic stimulation. For the repetition of the VEP test, a break interval of 0.5–1 min is observed to eliminate visual fatigue. The frequency of the stimulus is 1 Hz. The lamp used has low inertia and matte emissive surface, ensuring uniform distribution of light intensity.

3.4 Ways to collect the electrical signal at the VEP

The VEP signal is recorded with electrodes placed on the scalp in the cortical visual areas:

- Fz – median frontal area;
- O1, O2 – bilateral occipital areas, corresponding to the Brodmann visual area 17.

The electrodes, of the silver "mushroom" type, are fixed with electrolyte gel, after degreasing the scalp with medicinal alcohol. The contact resistance should be $< 10 \text{ k}\Omega$ to avoid artifacts. Each electrode is connected to a recording channel, part of the data collection system. v fig 3.2 and fig 3.3

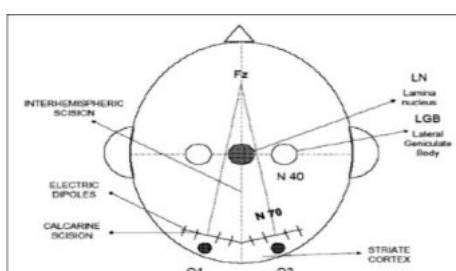


Fig. 3.2 Collection of the PEV signal [10] modified by Stanciu Nicolae

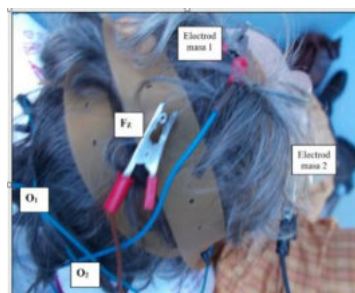


Fig. 3.3. Technique of collecting the VEP electrical signal. O1, O2 occipital areas of registration, left O1 and right O2 respectively. Fz is the reference electrode. E1, E2 ground electrodes. Vertical view from above.. Neurology Clinic. Neurophysiology Laboratory 2018. Nicolae Stelian Stanciu – [20]

3.5 Ways to collect the electrical signal at EEG

The recording of spontaneous EEG activity is done according to the internationally standardized "10–20" system, which is widely used. It involves placing 21 electrodes on the surface of the scalp, with landmarks at the Nasion (upper base of the nose) and Inion (cranial base, midline). The electrodes are distributed as a percentage over the Nasion–Inion distance (10%, 20%, 20%, 20%, 10%). The "lead-source" (QA) model displays the electric potential at each point relative to neighboring electrodes, using the weights method. v fig 3.4 and fig 3.6

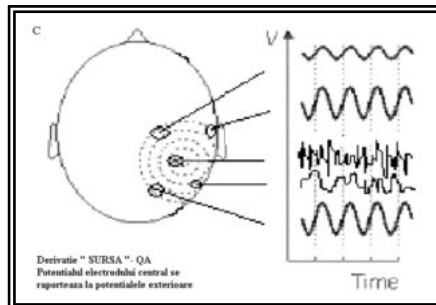


Fig 3.4.
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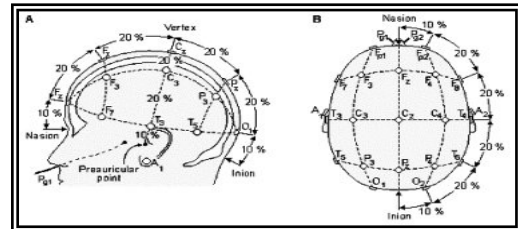


Fig 3.6. Percentage diagram of electrode placement [10],[11].

3.6 Electroencephalogram – Signal processing in the encephalogram

The electroencephalogram is the device that takes EEG signals from cortical electrodes and processes them through calibration, pre-amplification, analog-to-digital conversion and power amplification. The results are graphically displayed on the printer or on the machine screen.

3.6.1 Block diagram of an electroencephalograph

The block diagram (fig. 3.8) highlights the stages of collection, selection and pre-amplification of EEG signals. At the end of the amplification stage, an amplification factor of the order of 10^6 is obtained, with the preservation of the integrity of the signals. v fig 3.7 and fig 3.8



Fig 3.7. Electroencephalograph Bioscript Zwonitz 2000. Nicolae Stelian Stanciu 2018 [20]

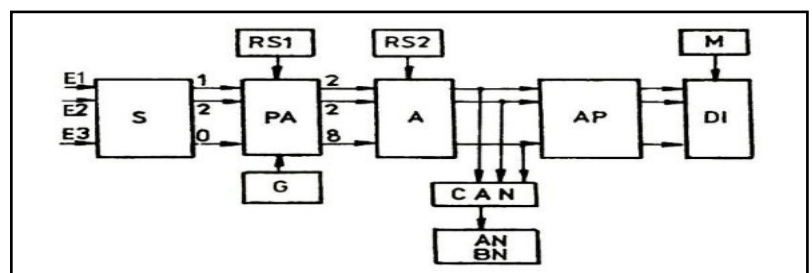


Fig. 3.8 Block diagram of an electroencephalograph. E1,E2,. E8 - electrodes; S – Selector – Frank Network; PA – preamplifier; G – Generator – reference voltage (potential); RS1, RS2 - sensitivity control systems; A - amplifier; AP – power amplifier; DI - printer; M - paper tape motor; CAN – analog-to-digital converter; ANBN – Digital System for Data Analysis. [15],[20].

3.8 Usefulness of the encephalograph – Obtaining EEG power spectra

The EEG power spectrum is obtained by the Fourier transform, after filtering, digitizing and storing the signal. To reduce noise, a Gaussian window (Hamming) is applied. The frequency resolution increases with the duration of the recording. The weight of the average EEG power (square amplitude) in relation to the total power emitted is calculated for the frequencies Δ , θ , α , β and BHF. The areas analyzed are: Clinical (0–26 Hz), BHF (26–62 Hz) and Integral (0–62 Hz). In the thesis thesis, the relative (weighted) power was used, calculated on each cortical area, with a step of 0.5 Hz.

3.7 Working mode and stages of processing EEG and PEV signals

3.7.1 Working method and obtaining the database – General

The subject is placed in a semi-dark room, in a horizontal position, with the EEG collection system applied to the scalp. It is requested to keep the eyes open or to alternately cover one eye with an opaque bandage, for exposure to flashing white light. The distance between the stimulated eye and the light source is adjusted by the operator. The electrodes are properly placed, and visual stimulation is done with standardized energies (0.3 J; 0.6 J; 1.2 J) at the frequency of 1 Hz. Recording lasts about 60 s and after removing the artifacts, it can be repeated after 60–90 s. The results are found on the display or rotating paper belt.



Fig 3.12 Subject to Flah testing – Nicolae Stelian Stanciu
Neurophysiology Laboratory 2018

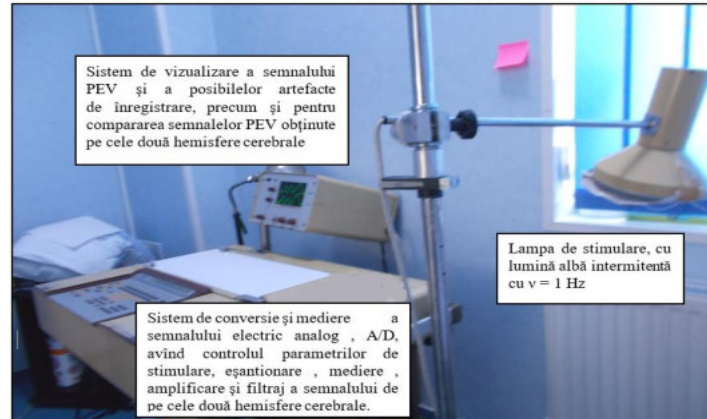


Fig 3.13. Equipment necessary for EEG and PEV recording. Obs.control system -digital recording, White Flash 0.6 J.Clinica de Neurologie Laboratory of Neurophysiology 2018.Stanciu Nicolae

3.7.2 Working mode for the analysis of experimental data of EEG activity

EEG analysis is performed with the Bioscript Zwonitz 2000 electroencephalograph, which collects and processes the subject's electrical signals. The data obtained are EEG pathway recordings, either at rest or in flash stimulation (0.6 J). Subsequently, they are entered into a computer, where they are processed: the artifacts are eliminated, the relative electrical power on cortical areas and frequency ranges is calculated, and the results are represented in EEG mapping.

The main stages are:

1. Obtaining the electrical power weights at intervals of 60–180 s.
2. Disposal of artifacts.
3. Making EEG maps for the integral range 0–62 Hz.
4. Segmentation of the integral range into 8 domains of 7.5 Hz + 1 domain of 2 Hz (0–7.5 Hz; 7.5–15 Hz; 15–22.5 Hz; 22.5–30 Hz; 30–37.5 Hz; 37.5–45 Hz; 45–52.5 Hz; 52.5–60 Hz; 60–62 Hz).
5. Segmentation of each range into 0.5 Hz steps and calculation of the relative power by the MapEEG 2011 program.
6. Getting tables of values in TXT format.
7. Converting tables to XLS format for later use.

Contributions to the analysis of bioelectrical signals in neurocortical activity to improve useful information in medical diagnosis

3.7.3 Experimental protocol

Steps 1–7 constitute an experimental worksheet. Several worksheets form an experimental protocol. Examples: Work tab 1 and Work tab 2.

PROTOCOLUL EXPERIMENTAL - FISA DE LUCRU NR.1 3.7.3.a

| NR CRT | EXPERIMENT | MULOACE DE LUCRU | ETAPE ALE MODULUI DE LUCRU | REALIZATORI | UTILITATEA |
|--------|--|--|--|---|--|
| 1 | INREGISTRAREA VALORILOR PUTERII RELATIVE EEG LA REPAUS | 11 SISTEM DE CULEGERE ELECTROZI 12 ELECTROENCEFALOGRAF 13 PROGRAM MAPERG 2011 PENTRU ELIMINAREA ARTEFACTELOR | ETAPA DE CULEGERE SEMNAL EEG ETAPA 1-2 ETAPA 3 ETAPA 4 ETAPA 5 ETAPA 6A ETAPA 6B ETAPA 6C ETAPA 7A ETAPA 7B ETAPA 7C ETAPA 7D ETAPA 7E | ASISTENT LABORANT WALTER TEUCH CERCET STINTIF PRINC D M PSATTA FIZICIAN COLABORATOR NICOLAE STELIAN STANGIU | 1-Experiment necesar pentru obtinerea spectrului de putere pe dimensi de frecventa diferite ale activitatii electrice cerebrale si pe zonele corticale la repaus 2-Experiment necesar pentru obtinerea hartiilor de corelatie zonale la repaus 3-Experiment necesar pentru obtinerea frecventelor valorilor de corelatie pe zonele corticale la repaus 4-Experiment necesar hartiilor de corelatie a frecventelor din diverse dimensi de activitate corticale la repaus |

PROTOCOLUL EXPERIMENTAL - FISA DE LUCRU NR.2 3.7.3.b

| NR CRT | EXPERIMENT | MULOACE DE LUCRU | ETAPE ALE MODULUI DE LUCRU | REALIZATORI | UTILITATEA |
|--------|---|--|--|---|--|
| 1 | INREGISTRAREA VALORILOR PUTERII RELATIVE EEG LA FLASH | 11 SISTEM DE CULEGERE ELECTROZI 12 ELECTROENCEFALOGRAF 13 PROGRAM MAPERG 2011 PENTRU ELIMINAREA ARTEFACTELOR | ETAPA DE CULEGERE SEMNAL EEO ETAPA 1-2 ETAPA 3 ETAPA 4 ETAPA 5 ETAPA 6A ETAPA 6B ETAPA 6C ETAPA 7A ETAPA 7B ETAPA 7C ETAPA 7D ETAPA 7E | ASISTENT LABORANT WALTER TEUCH CERCET STINTIF PRINC D M PSATTA FIZICIAN COLABORATOR NICOLAE STELIAN STANGIU | 1-Experiment necesar pentru obtinerea spectrului de putere pe dimensi de frecventa diferite ale activitatii electrice cerebrale si pe zonele corticale la flash 2-Experiment necesar pentru obtinerea hartiilor de corelatie zonale la flash 3-Experiment necesar pentru obtinerea frecventelor valorilor de corelatie pe zonele corticale la flash 4-Experiment necesar hartiilor de corelatie a frecventelor din diverse dimensi de activitate corticale la flash 5-Experiment necesar obtinerii variatiilor relative de putere ponderata pe zone si pe frecvente 6- Experiment necesar obtinerii cunost de sensibilitate maxime la stimularea flash a structurilor neuronale |

3.7.5 Stage 8A–8F. Obtaining EEG correlation values

For the analysis of the zonal and frequency correlations of EEG power, steps 8A–8F, performed in Excel, are applied, according to the methodology in the literature:

- Step 8A: Apply the Correlation function (Tools → Data Analysis → Correlation).
- Step 8B: Define the data retrieval domain.
- Step 8C: obtaining the diagonally segmented correlation table.
- Step 8D: Completing the table by adding diagonal values.
- Step 8E: Apply the IF function to obtain a complete table of zonal correlations.
- Step 8F: making the final table with the EEG power zonal correlation values ponderate.

These steps allow the correlations between cortical areas and between frequency ranges to be highlighted, constituting the basis for the identification of relevant EEG markers.

3.7.6 Steps A–E. Obtaining VEP data and methods for determining amplitude and latency

To obtain the amplitude and latency values of the VEP signal, the BIOSCRIPT – ZWÖNITZ 2000 electroencephalograph is used, connected to an analog-to-digital conversion interface and a PC computer.

The main stages are:

1. Signal filtering in the range of 0.3–70 Hz.
2. Repeat flash stimulation 25 times to mediate the VEP response and eliminate artifacts.
3. Recording on a time basis of 250 ms.
4. Digitization at 256 bit/channel (8 bit resolution).
5. Signal mediation is done according to the relationship

$$M(t) = [M(t-1) + V(t)]/2 \quad (3.1)$$

$M(t-1)$ = mean of the previous amplitudes, $V(t)$ = signal amplitude at time t

Steps A–E:

- A: Obtaining the signal from the electroencephalograph.
- B: Display viewing and graphic printing. v.fig 3.16
- C: Point-by-point analysis of the graph and extraction of pairs (t, V(t)).
- D: Calculation of amplitude by peak-to-peak method or relative method (difference between two signal peaks at different latencies). v.fig.3.17
- E: Simultaneous determination of amplitudes,latencies, and obtaining tables (t, V(t)).

Amplitudes are measured relative to an isoelectric line considered 0 V.

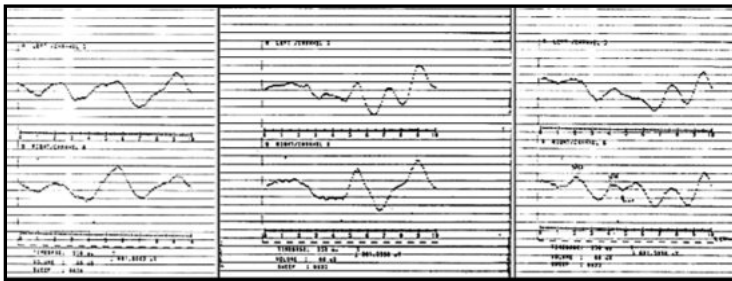


Fig 3.16 Stage B. Visually evoked potentials, printed on paper, in neurophysiological studies of conduction pathways, in pathology - neurophysiology laboratory.2015

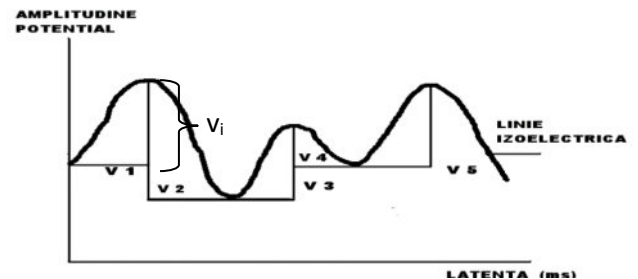


Fig 3.17. Stage D. Relative method of obtaining the amplitude of the VEP after Stanciu Nicolae Stelian [18],[20],[21]

3.7.7 Types of experiments for the study of EEG and VEP

EEG – Normal and Pathological

- Stand by: in the 0–26 Hz, 26–62 Hz (BHF), 0–62 Hz bands.
- 0.6 J flash: in the same frequency bands.
- Analysis of Flash–Rest Relative Variation: for weighted EEG power.

Pathologies analyzed:

- Obsessive-compulsive syndrome (OCS)
- Bipolar syndrome (BS)
- Autism
- Alzheimer's
- Schizophrenia

VEP – Normal and Pathological

- VEP amplitude as a function of flash stimulation energy (0.3 J; 0.6 J; 1.2 J).
- Analysis of VEP latencies in relation to stimulation energy.
- Study of the amplitudes and latencies of the VEP according to the stimulation of the conduction pathway (left/right hemisphere).

Pathologies analyzed:

- Neuropathy
- Epilepsy with left hemisphere focus

In these cases, changes in VEP amplitudes and latencies in the case of flash stimulation are investigated, in order to highlight the functional peculiarities of neuronal activity.

RESULTS ACHIEVED

Chapter 4 Study of EEG-weighted electrical power and its relative variation Rest – Flash in Normal and Pathological cases

4.1 Study of EEG-weighted electrical power

4.1.1 General

The EEG analysis was performed by numerical processing (v.3.13), obtaining the weight of the average EEG power (square amplitude) in relation to the total power recorded during the session (180 s). The evaluation was performed on all EEG frequencies (Δ , θ , α , β , BHF), in the 19 standard points on the scalp, for three frequency domains:

- Clinical range: 0–26 Hz
- BHF range: 26–62 Hz
- Full range: 0–62 Hz

The relative power was calculated as the zonal distribution of energy in the unit of time, with a step of 0.5 Hz, for each cortical area. The scalp has been divided into 5 functional areas:

- Zone I: prefrontal (PF1, PF2)
- Zone II: front (F7, F3, Fz, F4, F8)
- Zone III: central-temporal (T3, C3, Cz, C4, T4)
- Zone IV: parieto-temporal (T5, P3, Pz, P4, T6)
- Zone V: occipital (O1, O2)

4.2 Way of working in the processing of experimental data

The experimental work was structured according to a block scheme (figure 4.2), using data from the source [23].

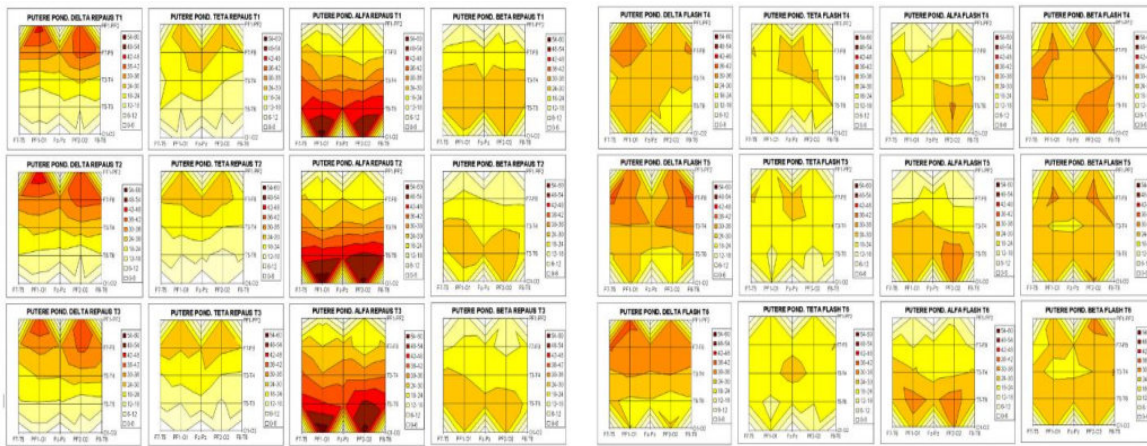
4.3 EEG weighted electrical power – characteristics

EEG-weighted power is influenced by multiple variables:

1. It is an averaged value of electricity in the unit of time.
2. The variety of cortical areas and frequency ranges influences the result.
3. It reflects the type of neural activity specific to the area analyzed.
4. A distinction is made between Normal and Pathological neurophysiological states.
5. It is sensitive to the type of pathology present.
6. It is influenced by the functional state: Sleep or Flash.
7. It changes depending on the state of Wakefulness or Sleep.
8. It is conditioned by the parameters of Flash stimulation (emotion, stress, intensity, duration, frequency, type, wavelength, stimulated eyes – AO, OD, OS).
9. It has a zonal wave character, dependent on time, frequency and cortical location

4.3. Differentiated characterisation of EEG-weighted power in the Clinic and BHF, in the Rest and Flash states, for Normal and Pathological cases.

4.3.1 / 4.3.2. Comparative analysis of EEG-weighted power in the Clinical domain (0–26 Hz), in the Rest (left) and Flash (right) states, for Normal cases, according to the moments T1, T2, T3 and Flash stimulation at T4. v.fig 4.3 a,b



S.4.3. Fig 4.3.a and 4.3.b Clinical Normal EEG relative power, on cortical areas: fig. Left Rest, at times T1, T2, T3 and fig. right-b Flash, at times T4,T5,T6.

Note: After Flash stimulation, there is a cortical redistribution of relative power, which over time returns to the cortical distribution at rest.

Fig 4.3 a Puterea electrică ponderată în domeniul clinic funcție de timp, Normal, Repaus - T1,T2,T3 - Nicolae Stelian Stanciu 2021 [12],[45]

Fig 4.3 b Puterea electrică ponderată în domeniul clinic funcție de timp, Normal, Flash - T4,T5,T6 - Nicolae Stelian Stanciu 2021 [12],[45]

4.3.3. Evaluation of BHF-mediated weighted power (26–62 Hz), in the Sleep and Flash states, for Normal subjects. v.fig 4.4

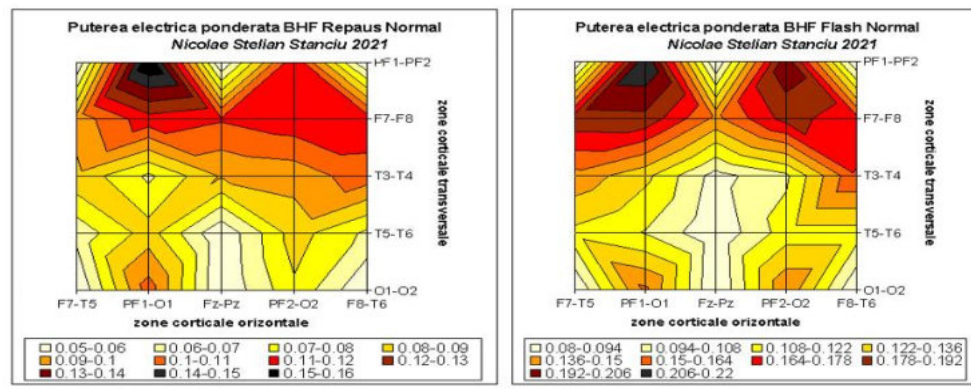


Fig 4.4 Weighted electrical power mediated in the B.H.F domain at rest and Flash, Normal – Nicolae Stelian Stanciu 2021 [12]. Obs. There is an increase in BHF-weighted power in Flash in all brain areas, but more pronounced in Prefrontal, Frontal and Temporal [12]

4.3.4.a / 4.3.4.b. Statistical frequency distribution of EEG weighted power in Rest (a) and Flash (b), in Clinical,Normal,cortical areas and frequency bands

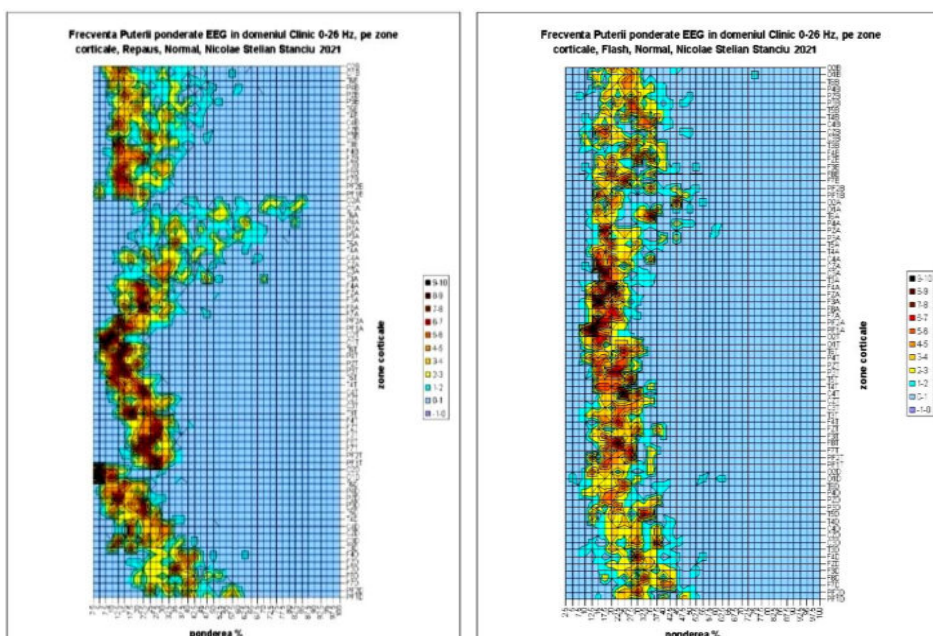
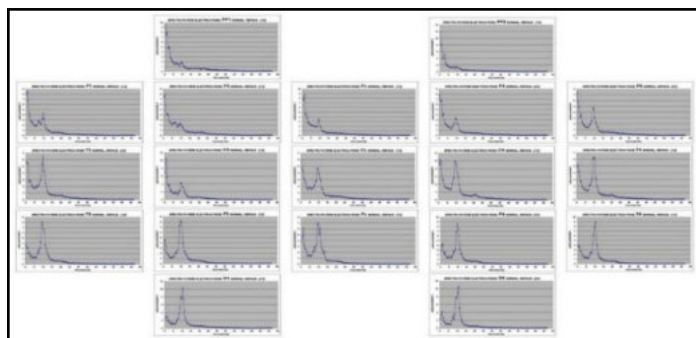


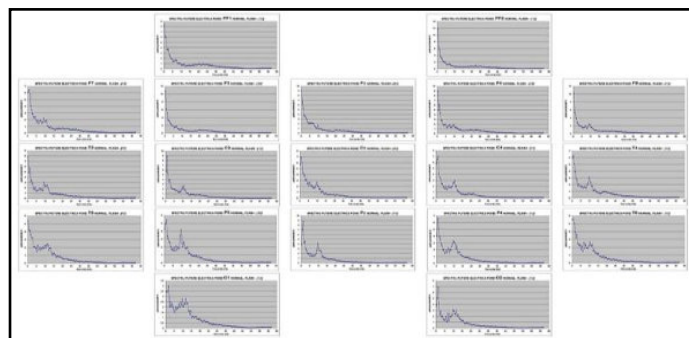
Fig 4.5 left for 4.3.4.a – Statistical frequency of the relative EEG power values on the cortical areas and the frequency of electrical activity, at Rest, Clinical.

Fig 4.5 right for 4.3.4.b - Statistical frequency of the relative EEG power values on the cortical areas and the frequency of electrical activity, in the Clinical field at Flash. At rest there is a separation of the values of the frequency domains of the activity so :d the Delta is separated from the Theta grouped with Alpha, which are both separated from the Beta. In Flash, the splitting of the value domains no longer exists, and there is a continuity in the value domains of the energy quantity. Power was considered to be the energy mediated by the unit of time. Nicolae Stelian Stanciu 2021,[12],[42]

4.3.6.a / 4.3.6.b. EEG weighted power spectrum according to cortical area and frequency (0–62 Hz), in the Sleep and Flash states, for Normal subjects

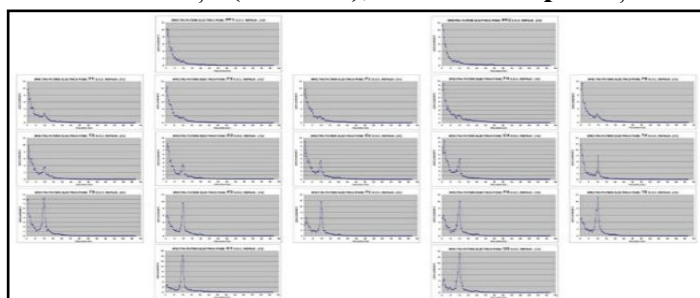


Mapping – **Fig 4.7 a** Weighted Power Spectrum. EEG depending on the cortical area and frequency. 0-62 Hz . Rest, Normal – Stanciu Nicolae Stelian [12] 2020

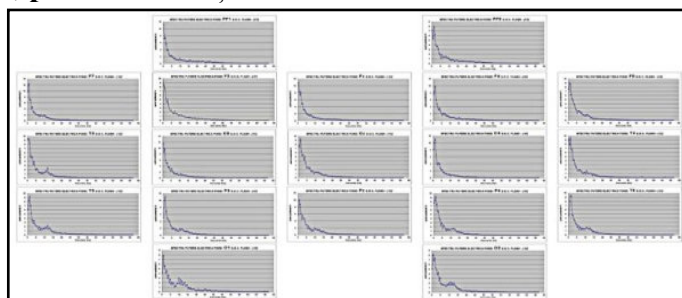


Mapping – **Fig 4.7 b** Weighted power spectrum. EEG depending on the cortical area and frequency. 0-62 Hz. Flash, Normal – Stanciu Nicolae Stelian [12] 2020

4.3.8.a / 4.3.8.b. Spectrul de putere ponderată EEG în funcție de zona corticală și frecvență (0–62 Hz), în stările Repaus și Flash, pentru subiecți cu S.O.C.



Mapping – **Fig 4.9 a** Pond power spectrum. EEG depending on the cortical area and frequency. 0-62 Hz, SOC rest, Stanciu Nicolae Stelian [12] 2020



Mapping - **Fig 4.9 b** Pond power spectrum. EEG depending on the cortical area and frequency. 0-62 Hz, Flash SOC, Stanciu Nicolae Stelian [12] 2020

4.3.9 EEG-weighted electrical power spectrum and frequency of neuronal activity (0–62 Hz) as a zonal mean, in the Rest and Flash states, for Normal and Pathological cases.

Significant differences were found between the two categories, both in spectrum amplitude and frequency distribution.

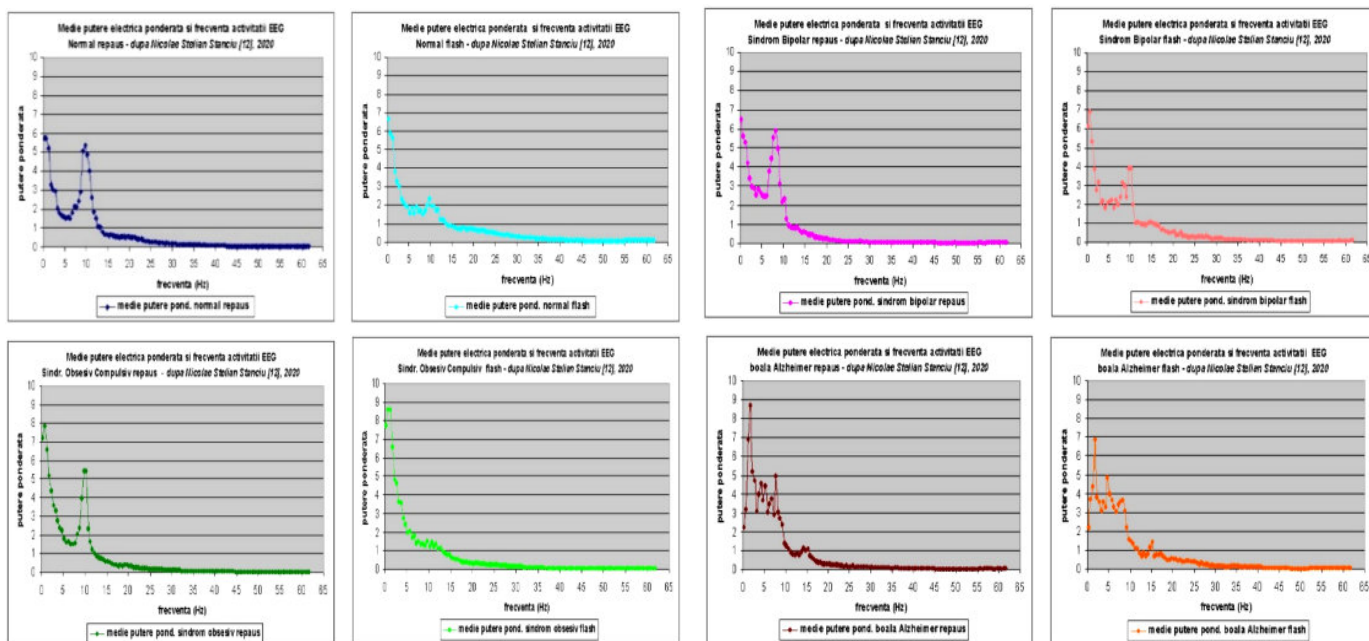


Fig 4.10.a Average weighted power at rest and flash in the range of 0-62 Hz Normal and S.O.C. Stanciu N.S

Fig 4.10.b Mean weighted power at rest and flash in the range 0-62 Hz S.B and Alzheimer's, Stanciu N.S [12]

4.3.10 EEG weighted power wave depending on the frequency of activity and cortical area, for normal cases.

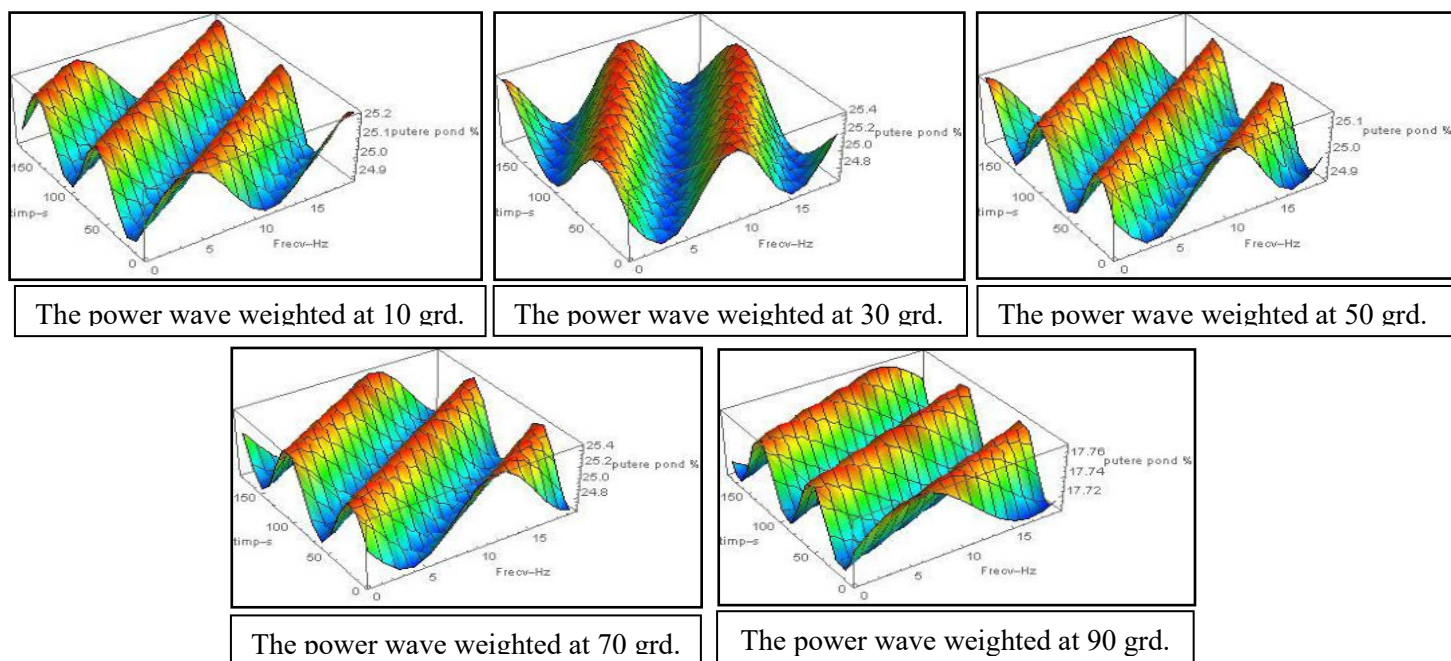


Fig 4.11 Electric power wave weighted as a function of cortical area (angle at center), frequency and time – Stanciu N.S 2021 [12], [23]

4.4 Reactivity of neuronal structures to Flash stimulation, Normal&Pathological cases

4.4.1 Neural reactivity – General

Percent neuronal reactivity is defined as the relative variation in EEG-weighted power between the state of Rest and the state of Flash stimulation, according to the relationship :

$$R = \frac{(P - P_0)}{P_0} \cdot 100 = R\% \quad (4.1)$$

where P_0 is the weighted power at rest, and P is the weighted power at flash stimulation.

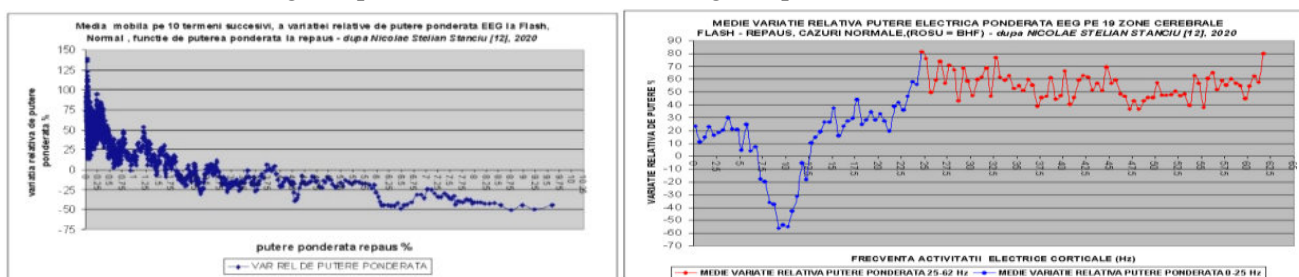


Fig 4.12 a,b Variation of weighted power and weighted power at rest – [23]

Obs Fig a on the left is the relative variation of weighted power EEG as a function of weighted power at rest
Fig b on the right is the relative variation of weighted power as a function of the frequency of cortical activity

4.4.6. The relative EEG-weighted power variation between Flash and Rest was analyzed on cortical areas, in the range of 0–62 Hz, for Normal cases.

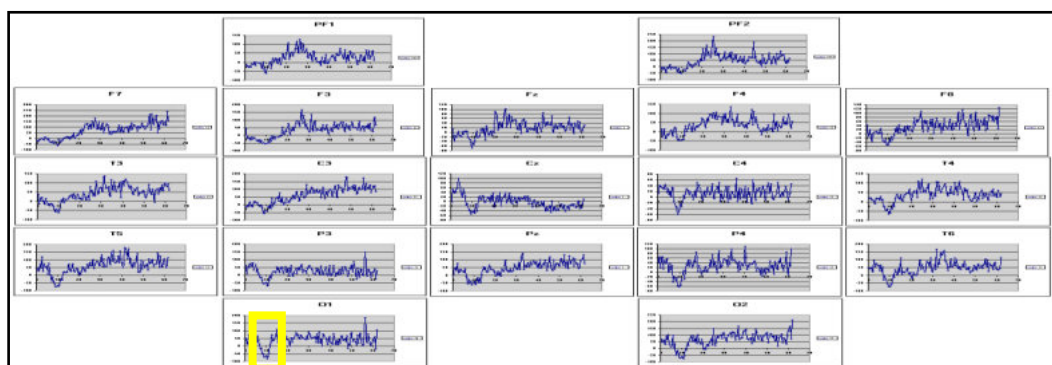


Fig 4.21 Mapping Relative Power Variation Flash-Rest by Zones and Frequency.. 0-62 Hz.Normal.Stanciu N.S. 2021.[12]

4.4.7. The weighted EEG power at rest and Flash, as well as the relative variation in the BHF range (26–62 Hz), were studied for normal cases.

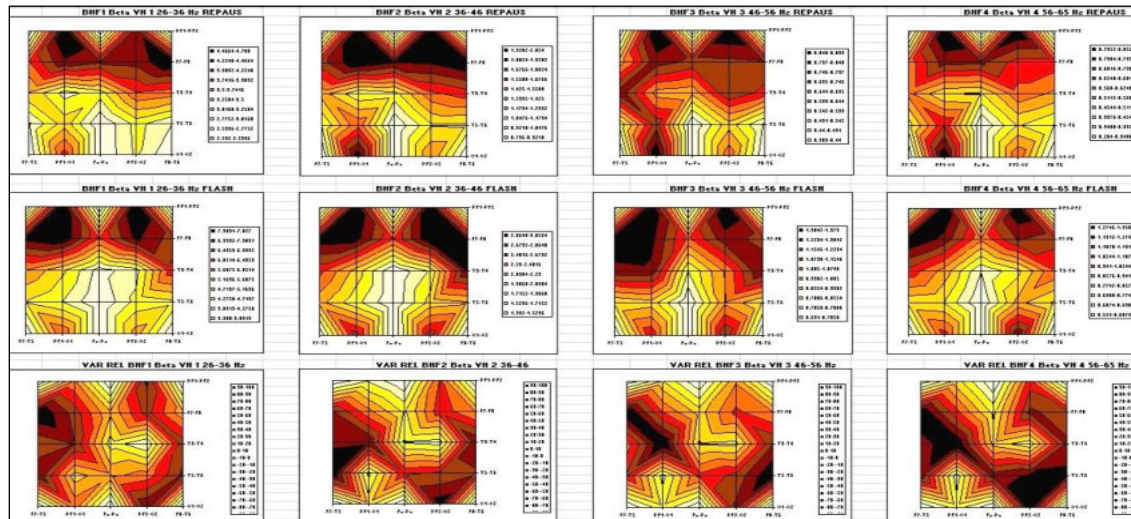


Fig 4.22. Weighted electrical power and relative variation in the BHF domain, zonal mapping, frequency function, Normal. **Note** The increase in weighted electrical power achieved by STG-DR interhemispheric transfer, with the increase in the frequency BHF - Nicolae Stelian Stanciu

4.5 The phenomenon of weighted power decrease in the Alpha Clinical domain

Following the Flash stimulation, a significant decrease in the weighted power was observed in the Alpha Clinic band (9.25–10.25 Hz), on all 19 cortical areas, and simultaneously with this there is also the phenomenon of Alpha activation in the bilateral frontal area F3 and F4. In addition, the phenomenon of the decrease in the weighted power at Flash stimulation is due to the energy consumption weighted in the unit of time for the processing of the electrical signal on the conduction pathways, but also in the neural structures, participating in this act of stimulation. The largest decreases were in the bilateral, parieto-temporal and central frontal (Fz) occipital regions. The relationship between the power of the Flash and that of the Rest depends on the Nation–Inion distance. The phenomenon is exponential in nature and involves a weighted energy consumption for the processing of the visual signal, both on the conduction pathways and in the neural structures involved, v (4.4) and fig 4.25

$$P^* = P = P_0 [47.432 - 1.386 \cdot \text{Exp}[0.0343 \cdot x]] \quad (\%). \quad (4.4)$$

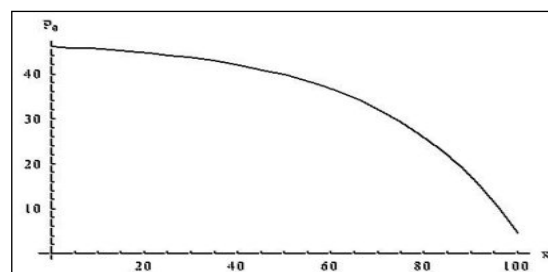


Fig. 4.25 Dependence on distance % Nasion – Inion of the power pond. Flash EEG in the Apha Clinical domain, 9.25–10.25 Hz. Stanciu Nicolae

4.6 R neuronal reactivity to Flash – pathological cases

Neuronal reactivity was investigated in the Normal and Pathological context, highlighting significant differences from normal cases, both in amplitude and in distribution on frequencies and cortical areas.

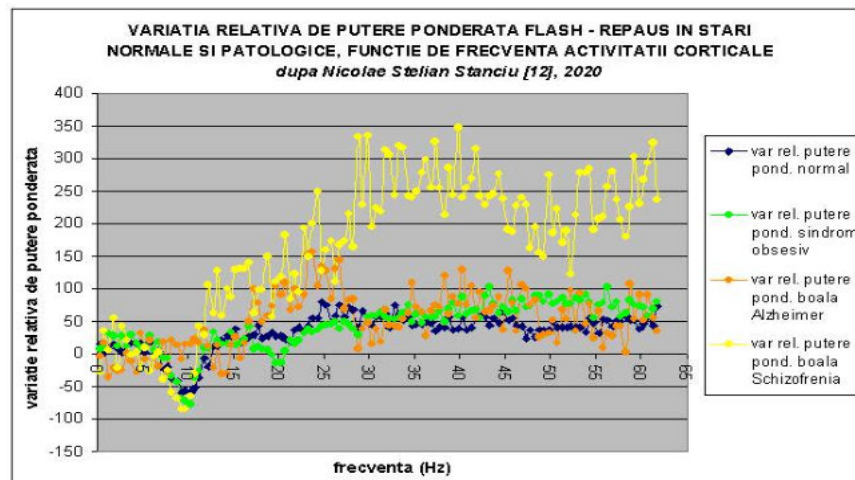


Fig 4.26 a. Comparative graphical description of the relative change in Flash-weighted power to normal and pathological cases. It is observed that the very high values of relative variation in both normal and pathological are found in the BHF frequency range, but in very complex pathology, these values are 6-7 times higher than in the normal case. The weighted relative power variation remains an indicator of neuronal sensitivity to flash stimulation, is rated R and is dependent on the specifics of the pathology. R = neuronal reactivity or sensitivity. [1][2]

4.7 R coefficient – normal vs. pathological comparison

4.7.1. Comparison of the R coefficient between Normal cases and Autism, in the Resting state.

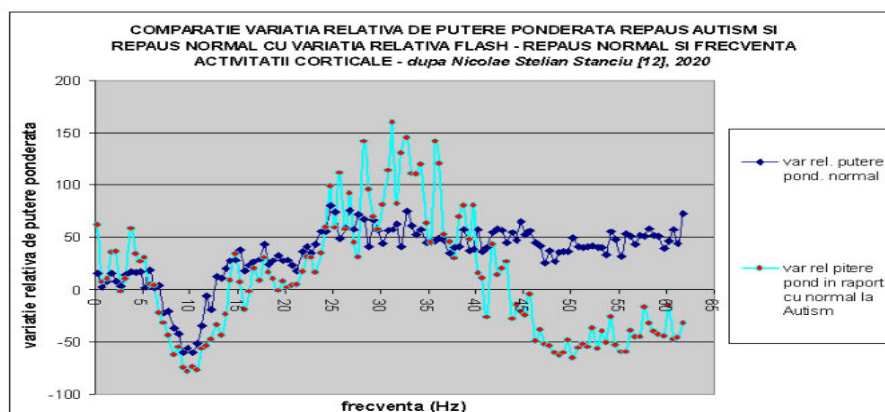


Fig 4.26 b. Comparative graphic description of the relative change in Flash-weighted power to normal and to the pathology Autism rest versus normal rest. **Obs** In autism, very large positive relative variations are observed between 26 and 45 Hz and negative variations between 45 and 62 Hz, compared to normal, which shows that starting with 35 Hz, the BHF domain practically does not work in the pathology of autism. [12]

4.7.2. Reactivitatea neuronală R la stimularea Flash în cazul bolii Schizofrenia.

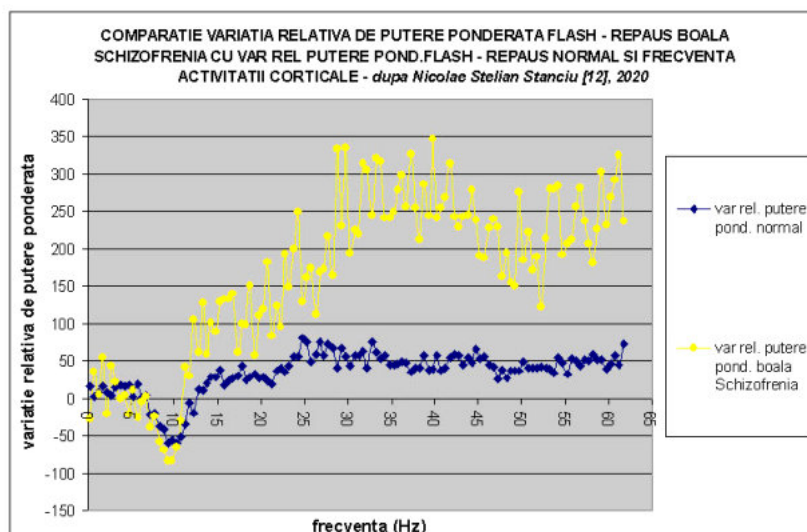


Fig 4.27 . Comparative graphical description of the relative variation of normal Flash-weighted power and pathology, schizophrenia, frequency function. It is observed that the very high values of relative variation to the pathological are found in the BHF frequency range, being much higher than in the normal case. A minimum of neuronal sensitivity is observed at about 50 Hz, for schizophrenia. The weighted relative power variation remains an indicator of neuronal sensitivity to flash stimulation and is dependent on the specificity of the pathology. In schizophrenia, the values are 6-7 times higher than normal. [12]

4.7.5. Reactivitatea neuronală R la stimularea Flash în cazul bolii Alzheimer.

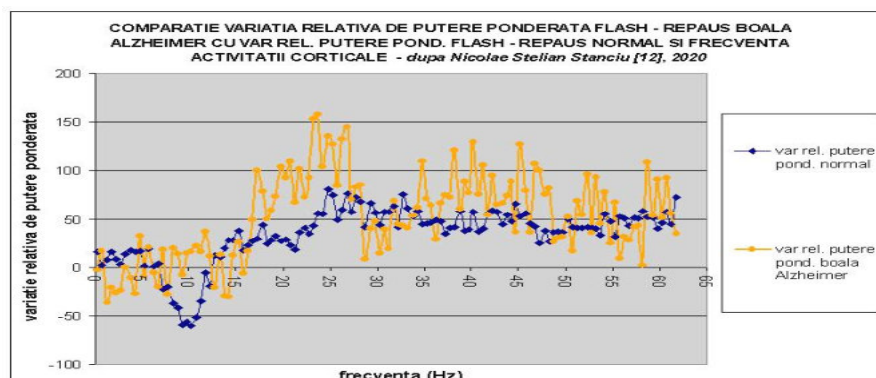


Fig 4.30 . Comparison Alzheimer's Disease – Normal at Relative Power Variation Weighted in the Frequency Range 0 – 62 Hz [12]

4.7.6. Relative EEG weighted power variation between Flash and Rest, on cortical areas, in the range of 0–62 Hz, for Alzheimer's cases.

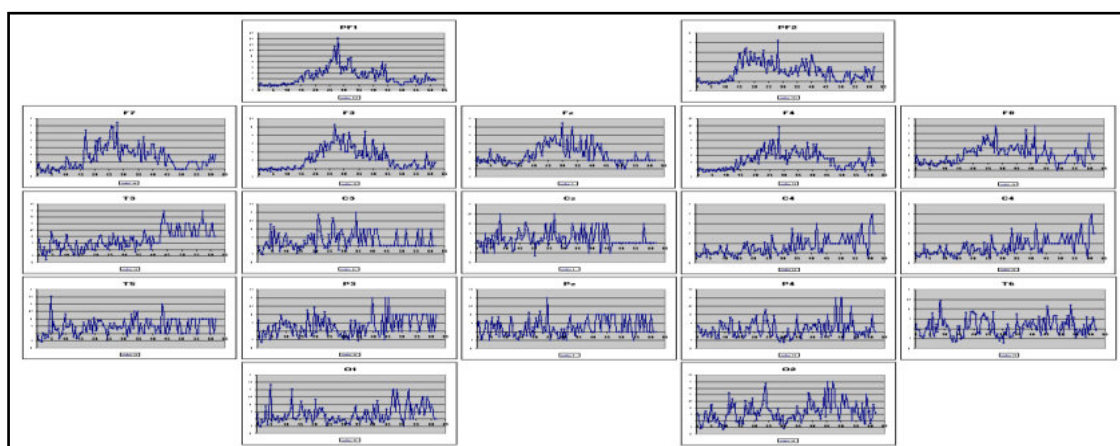


Fig 4.31 Mapping Relative power variation Flash-Rest R, by zones and frequency. 0-62 Hz. Alzheimer. Stanciu N.S. 2021,[12]

4.8 Function R – Indicator of Neuronal Functional Status

4.8.1. Experimental analysis of the relative EEG-weighted power variation

Experimental analysis of the relative variation of EEG-weighted power revealed a Boltzmann sigmoid limiting function, which describes the behavior of neuronal reactivity in the Clinical and BHF domains. It was found that there is a new electrical phenomenon – damped oscillation – which overlaps over the sigmoid limit, generating a complex functional relationship for neuronal activation over the entire frequency range.

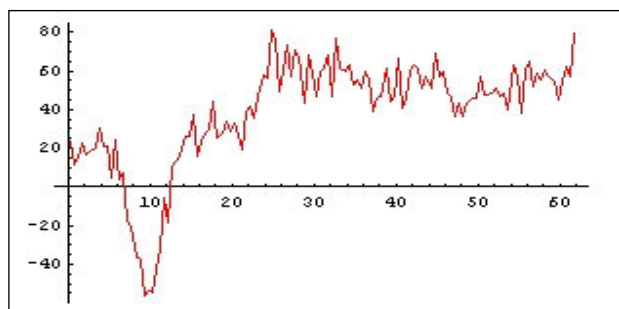


Fig 4.33 Graph experimental data for R-weighted relative power variation for EEG, Normal - Stanciu Nicolae Stelian 2024 [12]

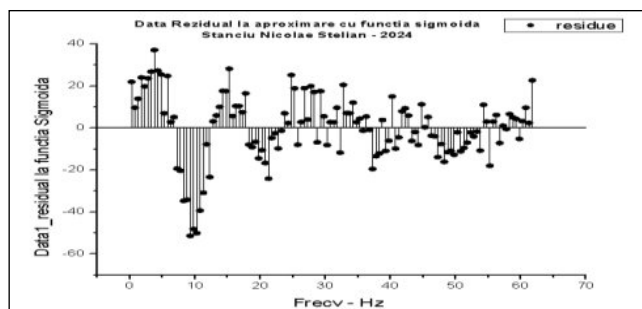


Fig 4.34 Graph of residual-error experimental data at the sigmoid fit function for the EEG-weighted relative power variation, Normal -Stanciu Nicolae Stelian 2024

4.8.2. Comparison between experimental data and the approximation function of R.

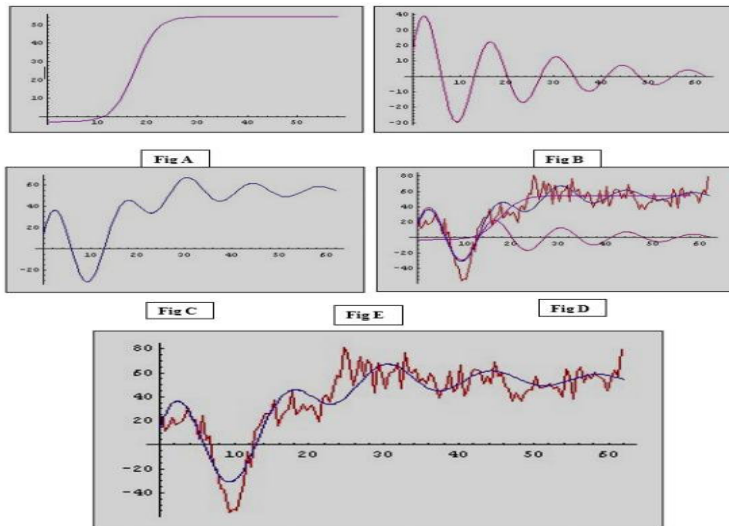


Fig 4.35 a - fig E-below. Comparison between the experimental data and the approximation function R obtained as a superposition of the sigmoid function and the damped oscillation function.

Fig 4.35 b - fig A,B,C,D-above. The types of functions involved in obtaining the approximation curves
fig A – Boltzman sigmoid,.
fig B – Decremental oscillation
fig C – resultant function,
fig D – Superposition of the reactivity approximation functions R Stanciu Nicolae Stelian 2024 [12]

4.8.3. Types of Functions Involved in Obtaining R Neuronal Reactivity Magnetization Curves and Description of EEG Weighted Power Variation

For the modeling of neuronal reactivity, three types of approximation functions were used:

- Boltzmann-type sigmoid function – for the limiting phenomenon (Fig. A, see fig. 4.35b)
- Damped oscillation function – for periodic decrease in amplitude (Fig. B, see fig. 4.35b)
- Approximation function of experimental data – the result of the overlapping of the two functions (Fig. C, v.fig 4.35b)

Figures D, A, B (v.fig 4.35b) illustrate the superposition of the functions to obtain the final approximation curve (Fig. E, v.fig 4.35a), leading to the function (v.fig 4.35b. C).

The functions involved in obtaining the approximation curves are:

The approximation function of the limiting phenomenon of the sigmoid function type–Fig A, v.fig 4.35b (4.5)

$$+ 54.55314 - \frac{57.35293}{1 + e^{0.45882(-17.58030 + x)}}$$

Approximation function of the damped oscillation phenomenon - Fig B, v.fig 4.35 b (4.6)

$$43.17271e^{-0.04009x} \sin[0.39463 + 0.44857x]$$

Experimental data approximation function – Fig C, v fig 4.35 b (4.7)

$$54.5531 - \frac{182674.72618}{3185.09824 + e^{0.45882x}} + 43.17271e^{-0.04009x} \sin[0.39463 + 0.44857x]$$

Figures D,A,B,v fig 4.35 b, indicate the superposition of the sigmoid functions and the damped oscillation function in order to obtain the final approximation function of the experimental data v. fig 4.35 a – E and show the possibility of comparing these data with the final obtaining of the function R v. fig 4.35 b -C[12]

Note. The "Mathematica" application displays many decimal places because it works with arbitrary precision and preserves all significant digits. Premature rounding would introduce errors and more inaccurate results, so I keep complete accuracy until the end. Here approximations of order 5 were made.

4.8.4 Conclusions on the R function

1. The relative variation of EEG weighted power (R or VRPP) is an indicator of neuronal functional status, with the role of a marker in the identification of pathologies.
2. R is generated by superimposing two processes: damped oscillation and sigmoid limiting (Boltzmann), on the 0–62 Hz range.
3. Damped oscillation is generated by resistive structures that reduce the amplitude of neuronal oscillations.
4. Between 0–12 Hz, damped oscillations dominate; between 12–62 Hz, sigmoid limiting dominates, with the appearance of relative highs and lows of R.
5. The period of damped oscillations is ~15 Hz, and of the free oscillations ~14.8 Hz. The decrease in the Alpha Clinic band (10 Hz) is the result of the overlapping of the two phenomena.
6. The derivative of the sigmoid function at semi-height indicates the degree of neuronal structural inhomogeneity.
7. The functions involved comply with the principle of superposition, being controlled by distinct mechanisms integrated into the variation R.
8. In severe pathologies (Schizophrenia, Alzheimer's), the R in the field of BHF can increase up to 600–700% or decrease to 50–80% (Autism), indicating deficiencies in the control of neuronal excitation and distortion of damped oscillation.

IMPORTANT NOTE

It is observed that the relative variation of weighted power highlights the pathology when its percentage values exceed values of 80% or when the values of pathological R decrease compared to those of Normal R with values between 10% - 100%. The frequency ranges that highlight functional deficiencies are:

- 1 – BHF + Clinical domain for highlighting the pathological frequency domains in the integral frequency range 0-62 Hz.
- 2 – the BHF domain with an average of 44 Hz and the area of β high frequencies or β 2 with an average of 22.5 Hz, located at the lower edge of the BHF domain, for highlighting the brain areas with pathological activity.

It is observed that a coefficient called **stimulation reactivity** can be defined $R = \text{the relative percentage variation of EEG Flash-Rest weighted power in BHF or neuronal reactivity}$, which would highlight various types of neocortical pathologies that have neuropsychiatric effects. In the case of this work, it is found that the higher R has values, the more complex and difficult the pathology is to be remedied.

4.9 Relative Variation of EEG-Weighted Power in Relation to the Time Domain, Normal

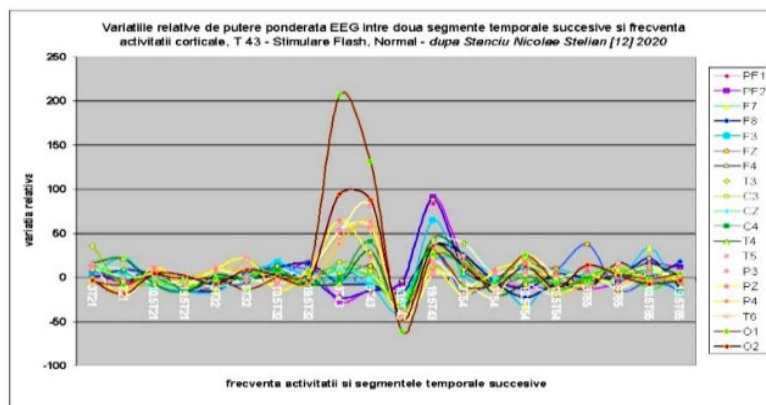


Fig 4.35.. The relative variation in EEG-weighted power between two successive time segments and frequency. cortical activity, Normal - [12]

4.9.1. How it works. EEG activity was segmented into 30-second intervals, resulting in:

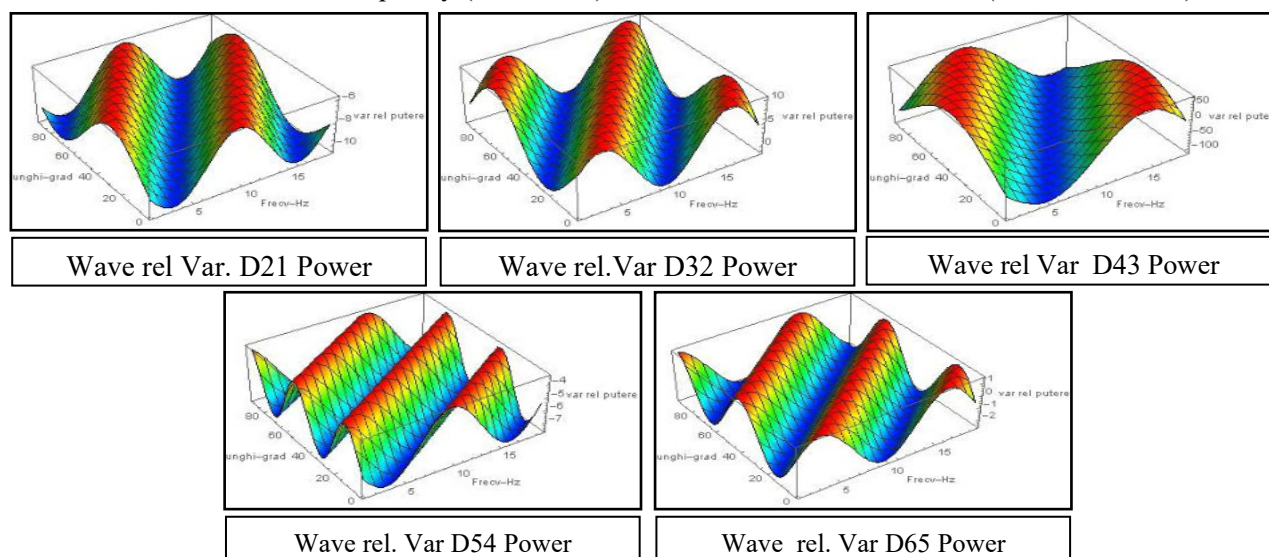
- Rest: 3 segments (90 seconds) T1, T2, T3
- Flash: 3 segments (90 seconds) T4, T5, T6

Total time : 180 seconds.

The relative weighted power variations were calculated between successive segments: D21, D32, D43, D54, D65, for each of the 19 cortical areas (see fig 4.35). It is observed that Flash excitation in T4 produces damped oscillations in the temporal range of the relative EEG-weighted power variation specific to each frequency range.

4.9.3 EEG Weighted Relative Power Variation Wave

The wave of the relative variation of EEG weighted power was analyzed according to the standard angle of the cortical area and the frequency (3–19.5 Hz), in the Clinical domain, Normal (Stanciu N, 2021)



4.10 Standard deviation of weighted relative power change EEG

4.10.2. Standard deviation relative variation as a function of frequency and standard angle, Normal.

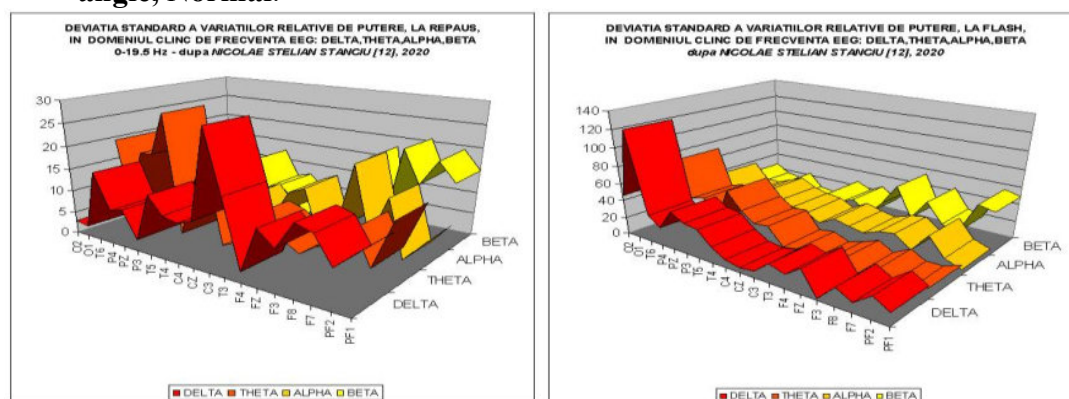
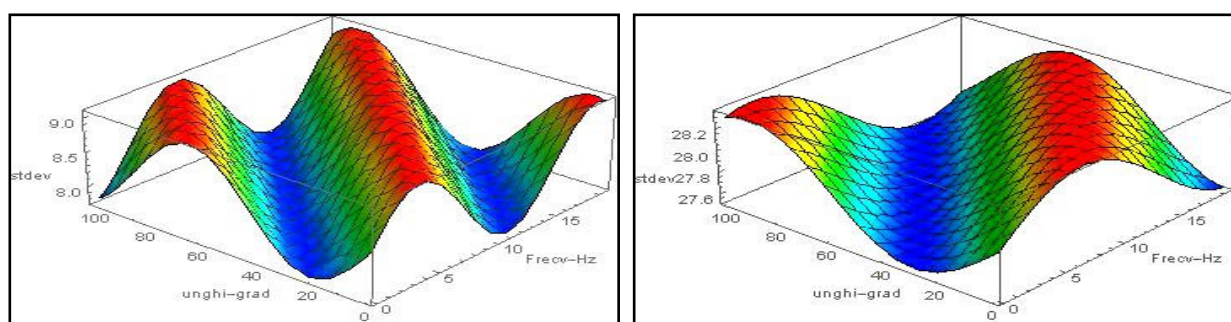


Fig 4.40 a,b. Standard deviation of the relative variations of EEG-weighted power, Clinically between two successive temporal segments, the frequency of cortical activity and the cortical area, Normal, **Rest-a** (left) and **Flash-b** (right) - Stanciu Nicolae Stelian [12],[31]

The time domains D1–D6 were analysed and the variations D21–D65 were graphically represented (see fig 4.40).

- At Flash, the amplitude of the standard deviation wave is greater than at rest.
- The standard deviation values are periodic, with a wave appearance, depending on the frequency and cortical area (see fig 4.41, Table 4.6, Table 4.7).

4.10.3. Standard deviation wave of the relative EEG-weighted power variation, as a function of cortical area and frequency, in the Rest and Flash states, for Normal cases



a) STDEV Weighted Relative Power Variation, Time Domains, Rest, Normal

b) STDEV Weighted Relative Power Variation, Flash Time Domains, Normal

Fig 4.41 Stdev wave var.weighted power a) Left-Rest and b) Right-Flash, Normal. Stdev wave of the relative variation of EEG weighted power in the time range Rest T1,T2,T3 and in the time range after Flash stimulation T4,T5,T6 with Flash in T4, depending on the standard angle of the 10-20% Jasper scheme and the frequency of electrical activity dome Clinic. **Note:** It is observed that there is an oscillatory process propagable from one cortical area to another, depending on the type of wave, the wave placed in the frequency range of the EEG electrical activity and the standard angle. Fig b) (top right). Analytical graphic description of the relative variation of the weighted power in Flash as a function of frequency and the standard angle, at Normal It is observed that in Flash the oscillator phenomenon changes its number of maximums reached along the Nazion-Inion line, decreasing its frequency with respect to Rest in space 3 D (stand angle - activity frequency - stdv). – Nicolae Stelian Stanciu 2021.[12]

Chapter 5 The concept of EEG and VEP correlation and correlation analysis

5.1 Correlation Analysis – General

Correlation is the concordance between events characterized by common parameters, making sense only in structures with similar components and operating laws. EEG and ERP (evoked related potential) offer non-invasive methods with millisecond temporal resolution for the study of brain functions.

- They are useful in highlighting cognitive abilities and differentiating functional brain states.
- Contributes to neurological and psychiatric diagnosis.
- EEG/ERP correlations reflect processes such as learning, sleep, memory consolidation.
- Correlating brain parameters with external stimulation or various pathologies is essential in the study of CNS functionality.

5.1.4 Linear correlation coefficient

The linear correlation coefficient removes the limitations of covariance and provides information about:

- Bond strength: indicates independence; large values indicate strong bonds.
- The meaning of the connection: positive, negative.

The general formula is expressed in the relation (5.5). [26] – Constantin Antonescu,Tudorel Andrei,Liviu Stelian Begu

$$r = \frac{\sum (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\left[\sum (x_i - \bar{x})^2\right] \left[\sum (y_i - \bar{y})^2\right]}} \quad (5.5)$$

5.3 EEG Zonal Correlation – Characteristics and Phenomena

5.3.2. Features EEG zonal correlation. It is maximum when two signals have a linear relationship, with identical amplitude, frequency and phase.

- Synchronization of signals is essential for sensory processing.
- Signal coherence implies maximum correlation ($r = 1$).
- In the case of EEG activity between brain regions, high correlation indicates processing synchronized.
- Pathology occurs when the neural system cannot control or delimit areas with linear processing, affecting coherence.

5.3.3. The zonal EEG correlation has an oscillatory character depending on the frequency of cortical activity, in normal cases.

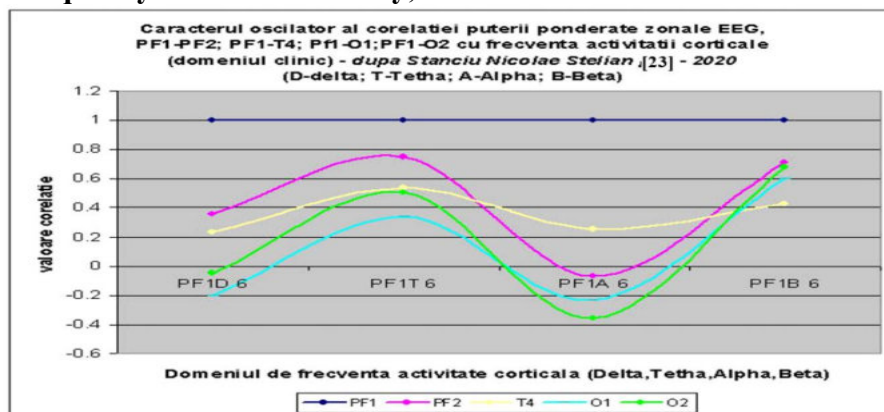


Fig 5.2 The oscillatory character of the correlation values in the activating zone PF1, with the zones T4,O1,O2, in various frequency ranges and at moment no. 6 of recording time. indicating a wave character - after Stanciu Nicolae Stelian [12],[23], 2020,2018

5.3.4 The phenomenon of decreasing the correlation of the area-weighted power as a function of the distance from the cortical activating area to the correlated one. Clinical.

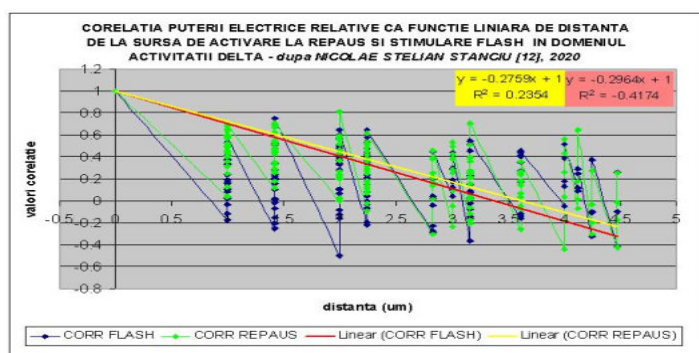


Fig 5.3 a.Decreasing the value of the correlation as a function of the distance from the active area to the correlated one.domain.Clinical.Delta.Normal. Stanciu N.S 2020.[12],[23].2018

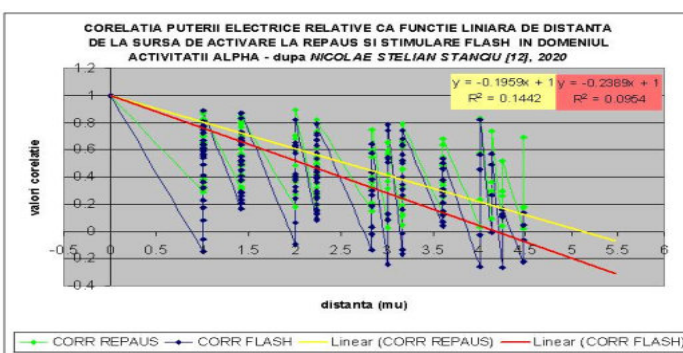


Fig 5.3 b. Decreasing the value of the correlation as a function of the distance from the active areaall to the correlated one.domain.Clinical Alpha.Normal. Stanciu N.S.2020.[12],[23].2018

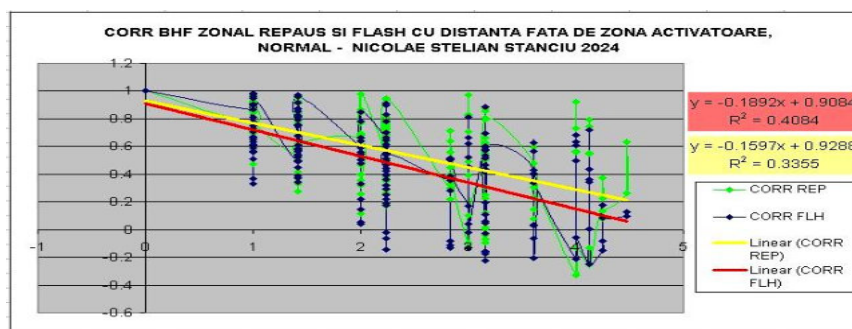


Fig 5.5. Decrease val.corel. depending on the distance of the active area.domains BHF, Stanciu N.S 2020,[12],[23],2018

5.3.6 Correlation spectra as a function of the distance between the activating area F7 and the activated, correlated area and domain. of frequency. Clinical, Rest and Flash Normal.

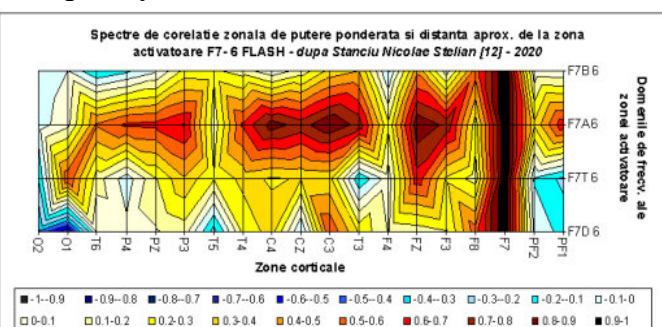
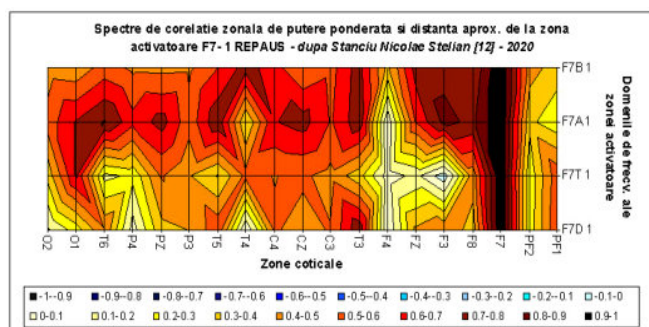


Fig 5.6. Spectrum of Corel. Depending on the distance of the activating area – correlated area. Zone F7 Rest-up and Flash-down, Normal.Stanciu N S 2020 [12],[23]

5.3.7. Decreasing the value of the zonal EEG correlation with the distance from the activating area, as well as decreasing the standard deviation at Flash compared to Rest, Normal.

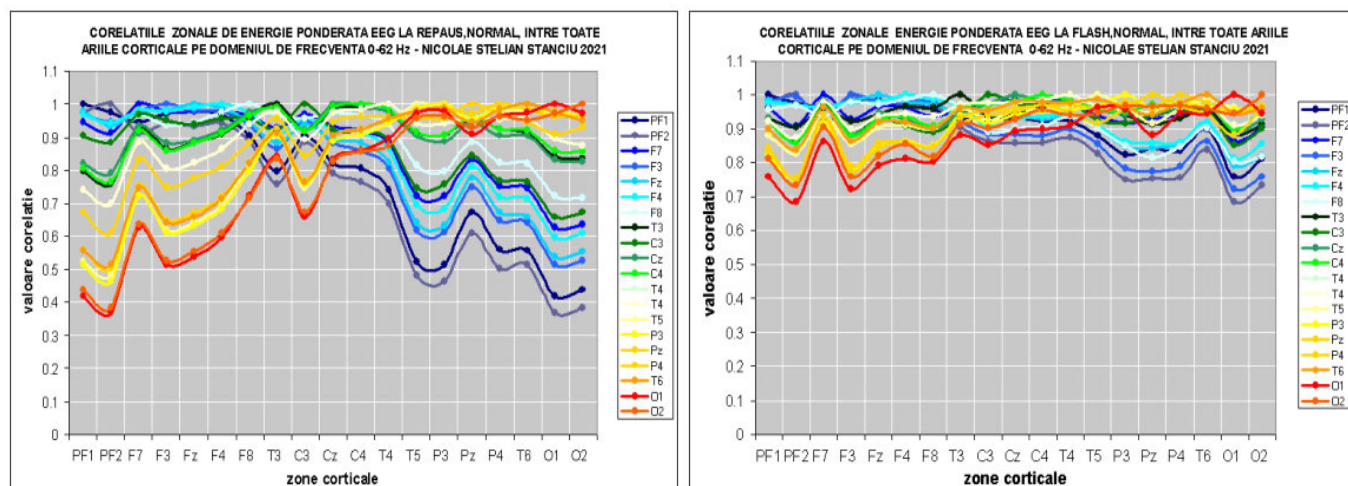


Fig. 5.7. The phenomenon of decreasing valorii.de correlation between power and evil. with dist.de to the triggering area to the correlated one. Flash-Rest, Normal.. Stanciu N S 2021, [12]

Note. It is observed both at rest and at Flash that the zonal correlation values decrease as the distance from the activating area where the correlation value is maximum, i.e. 1.v fig 5.6, fig 5.7..In addition, it is found that at rest the despair is greater than at Flash. This fact certifies the previous statements that support the fact that with the Flash stimulation the high correlation values are spatially restricted around the activating areas, where the level of value homogeneity is high. The increase of the correlation values means the increase of the linearity of the electrical phenomenon by increasing the value homogeneity around the activating areas. In this way, the electrical signal can propagate undistorted, both in terms of form and amplitude value, there being the phenomenon of the zonal distribution in phase of the zonal correlation values, i.e. of the coherence of the zonal correlation values. This phenomenon of the coherence of the zonal correlation values is evidently observed both at Rest and at Flash. This phenomenon constitutes the foundation and lays the foundations of the premises of the existence of the Visually Evoked Potential (VEP) at Flash stimulation. – Nicolae Stelian Stanciu 2021 [12]

5.3.8.a. The phenomenon of rotation of the values of the zonal EEG correlation in relation to the central area T3–T4, present at Flash and at rest, with the decrease of the standard deviation at Flash.

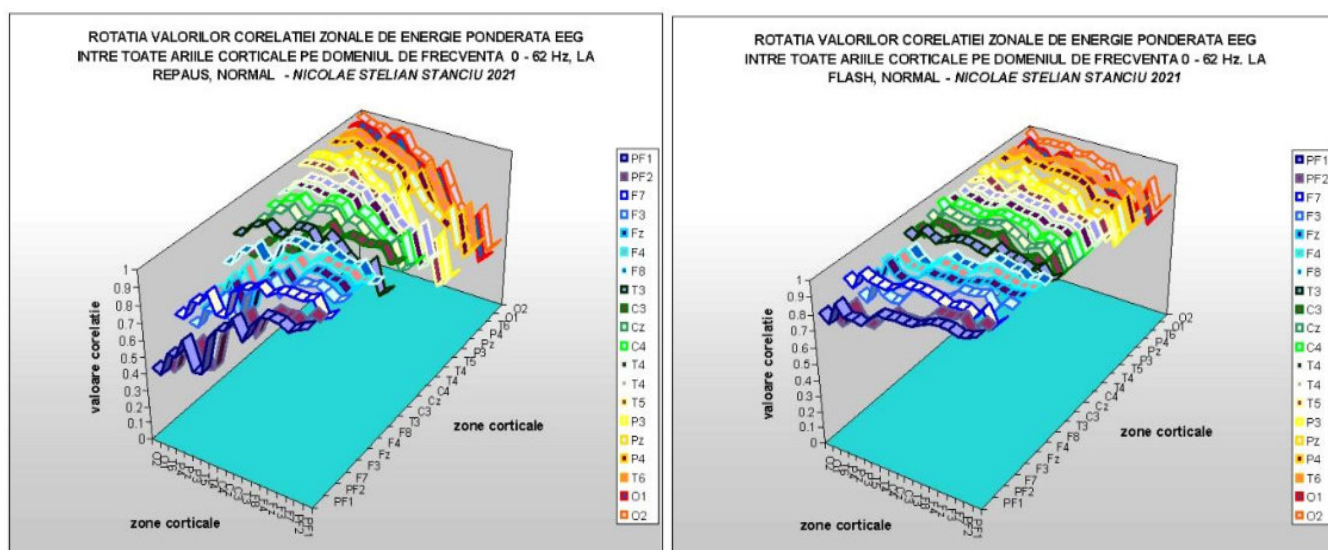


Fig .5.8. The phenomenon of rotation valorilor.de correlation power rel.. with dist.de to the triggering area to the correlated one. Rep-Fllh., compared to the T3-T4 area. Normal.. Stanciu N S 2021 [12]

5.4.1 The phenomenon of decreasing the mean value of the EEG-weighted power correlation of the frontal areas, as a function of the distance from the cortical activating area to the correlated one. Normal case. Analysis. (Nicolae Stelian Stanciu 2024), [12]

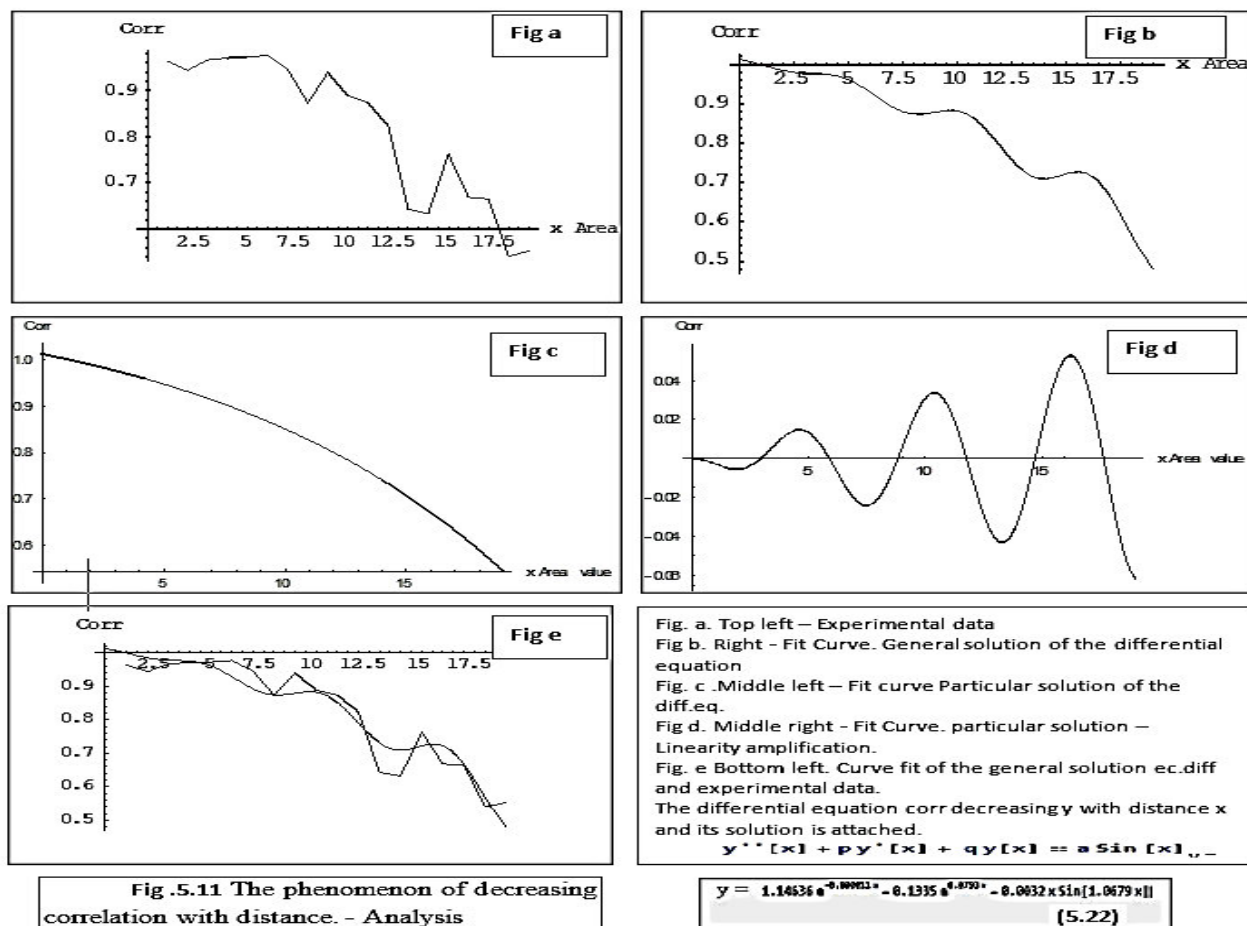


Fig .5.11 The phenomenon of decreasing correlation with distance. - Analysis

5.4.2 Decreasing the zonal EEG correlation and decreasing the standard deviation at Flash from Rest, Pathological, Bipolar Syndrome.

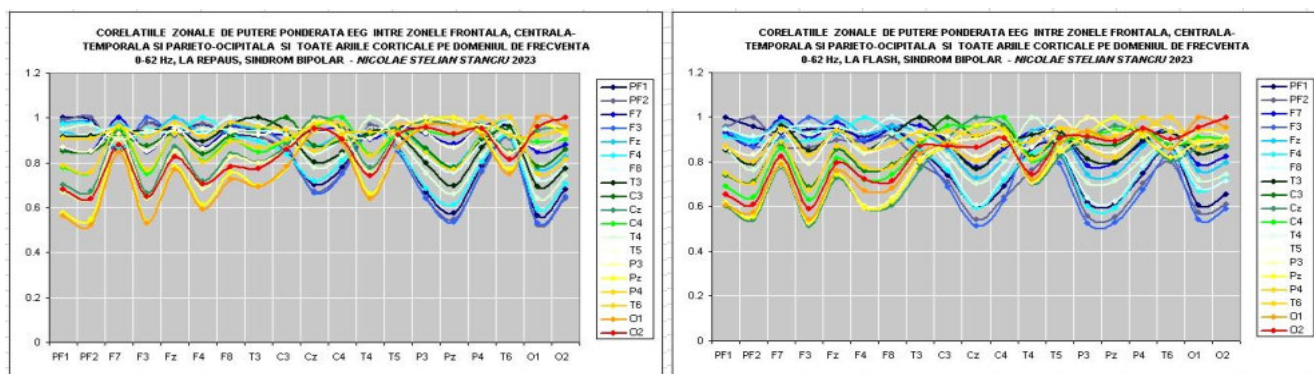


Fig .5.12. The phenomenon of decreasing valorii.de weighted power correlation. with dist.de to the activating area to the correlated one. Rep-Flh.,Sindr.Bipol.. Stanciu N S 2023, [12]

Note. It is observed both at rest and at Flash that the zonal correlation values decrease as the activation zone moves away from the activating zone where the correlation value is maximum, i.e. 1 v.fig.5.12 .In the pathological case of Bipolar Syndrome, it is found that in Flash the value of the dispersion of the correlation values is higher than in the Normal case. This situation indicates that the electrical signal is transmitted distorted both in shape and amplitude, forming a correlation image specific to the SB pathology.In the case of SB, the inhibitory mechanisms of the spread of correlation values in brain structures are affected. – Nicolae Stelian Stanciu 2023, [12]

5.4.3. The phenomenon of decreasing the zonal EEG correlation and reducing the standard deviation at Flash compared to Rest, Pathological Case Obsessive-Compulsive Syndrome (OCS).

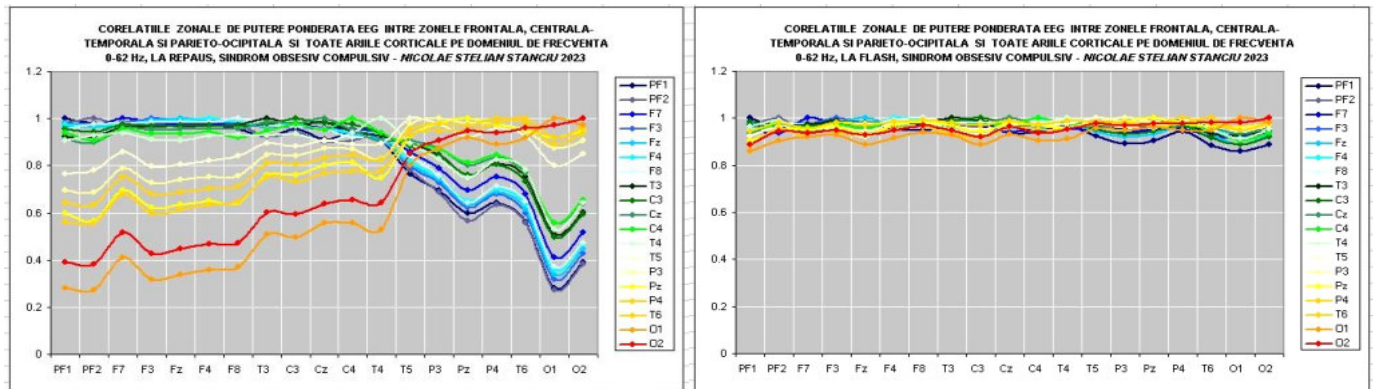


Fig 5.13tag. The phenomenon of decreasing valorii.de weighted power correlation. with dist.de to the triggering area to the correlated one. Rep-Flh.,Sindr. Obsesive. Compulsive. Stanciu N S 2023,[12]

Note. It is observed at rest and at Flash that the zonal correlation values differ greatly from those of the Normal case. In addition, it is found that compared to Normal, at SOC Rest, the correlation dispersion is low for the frontal and central temporal areas and high for the other areas, when the activation is frontal and central-temporal, and at Flash, the dispersion of the correlation is very small, for all activating areas. front. These facts indicate that at Flash stimulation the correlation values are high and are distributed in all brain areas, having a high level of value homogeneity. The structure behaves as a compact, functionally undifferentiated whole. The increase of the correlation values means the increase of the linearity of the electrical phenomenon by increasing the value homogeneity around the activating areas. In this pathological case, the electrical signal propagates undistorted, both in form and in amplitude value, between the central and frontal brain areas, there being no specific differentiation in the area of processing or generation of these signals. In SOC pathology, the control system of cortical inhibition, of the zonal spread of correlation values is affected, with pathology in the frontal and central-temporal area, with the existence of an uncontrolled positive amplification reaction of signals on all cortical areas to flash stimulation. This phenomenon is the basis of SOC.'s pathology. – Nicolae Stelian Stanciu 2023 , [12]

5.6. Correlation analysis of EEG signals The phenomenon of zonal correlation and frequency correlation of EEG weighted power. Description by Mappinglect

5.6.1. Weighted power correlation associated with EEG, Normal and Pathological zonal electrical activity. Features. Description by Cortical Spatial Mapping

Characteristics of the phenomenon of zonal correlation of cortical activity [7],[9],[12]:

1. The value of the zonal correlation of the electrical power relative to the flash increases as spatial spread with the increase of the frequency of the activity domain, from delta to beta, increases as a structuring order with the frequency of the flash activity domain, the structuring being made by the formation of persistent patterns in space, spatial patterns with high inertia.
2. The zonal correlation of the relative electrical power EEG behaves over time as an oscillator-wave phenomenon, which can take the form of a plane wave, being coherent on some spatial areas [12].
3. The best oscillations in time are observed in the prefrontal areas and they fade in amplitude and shape as the areas are farther away from the PF.
4. The zonal correlation of flash-weighted electrical power shows that as the frequency of activity increases, the spatial spread (dispersion) decreases relative to the resting state for Delta and Alpha and increases for Theta and Beta [12],[23].
5. As a general trend, the zonal correlation tends to decrease in value as spatial spread at flash versus rest, in relation to the activating area.

6. For high frequencies, e.g. Beta and BHF tend to spatially concentrate large correlation values in spatially limited areas, forming spatial patterns, in which the signal tends not to be altered in shape [12]. The spatially limited distribution of the high correlation values around the activating areas is based on the principle of minimum energy consumption.

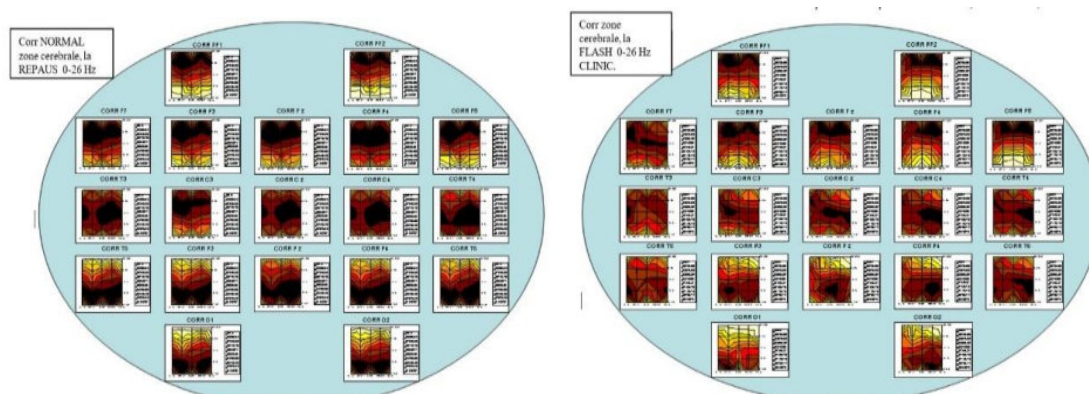
7. For rest and flash, the correlation values are oscillating in relation to the frequency of the field of activity. The values of the correlation of the relative zonal power tend to be symmetrical with respect to the median nasion-ion axis and show patterns at high frequencies both at rest and at flash [12].

8. In the correlation of EEG activity there are correlation patterns (models), which can have longitudinal symmetry in relation to the T3-T4 areas, or transverse symmetry in relation to the nasion-ion areas, both at Normal Rest and Flash, or Pathological Rest and Flash. In the case of frequency correlation, these patterns can appear as a distinct element of a specific pathology (Autism, Schizophrenia, etc.) [12],[23],[43].

5.6.2. EEG zonal weighted power correlation. Spatial cortical mapping.

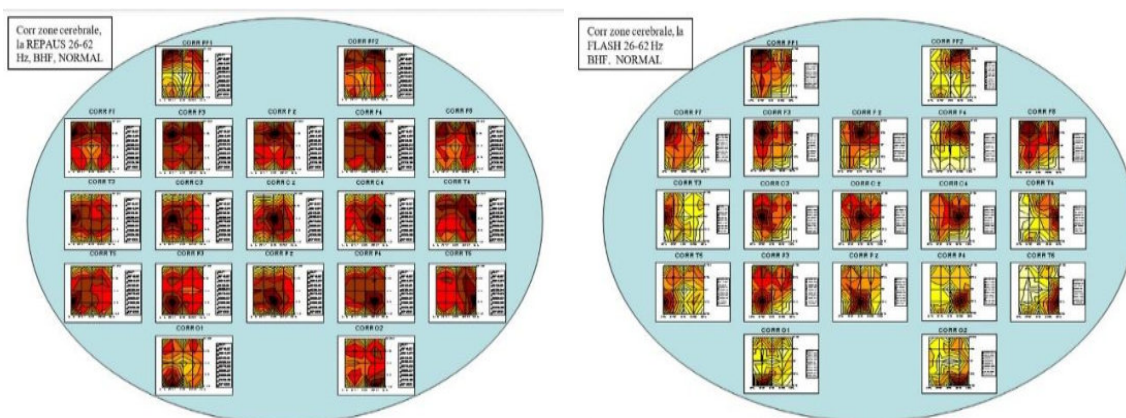
Normal case (0–62 Hz) [23]

a.b. Corel. in the CLINICAL field of frequency of electrical activity EEG, Normal, Rest & Flash



Map with the spatial dispositions on the cortical areas of the relative power zonal correlations in the Clinical range of EEG-26 Hz frequency, **Fig .5.18.a left rest, Fig .5.19 b right flash** normal. Stanciu N.S 2021,[12],[23],2020

c.d. Corel. in the BHF range of frequency of electrical activity EEG, Normal, Rest & Flash



Map with the spatial arrangements on the cortical areas of the relative power zonal correlations in the BHF range of EEG frequency 26-62 Hz, **Fig .5.20,c left rest, Fig .5.21.d right normal flash** . Stanciu N.S 2020,[12],[23],2020

5.6.5. EEG zonal weighted power correlation. Spatial cortical mapping.

Pathological case – Schizophrenia disease (0–62 Hz) [23]

A.B. In the CLINICAL field of frequency of electrical activity EEG, Schizophrenia, Rest & Flash

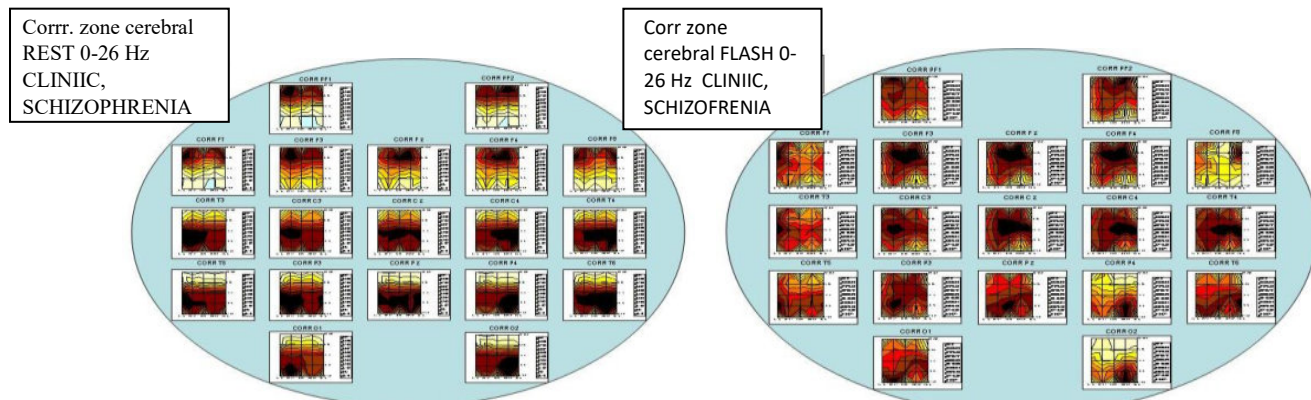


Fig. 5.30 Map with the spatial dispositions on the cortical areas of the zonal correlations of relative power in the Clinical range of EEG frequency 0-26 Hz, rest, Schizophrenia Disease – after N.S.Stanciu [12],[23],2020

Fig. 5.31 Map with the spatial dispositions on the cortical areas of the relative power zonal correlations in the Clinical EEG frequency range 0-26 Hz, Flash, Schizophrenia disease - after Ni S Stanciu [12],[23],2020

c.d. Corel. in the BHF field of frequency of electrical activity EEG, Schizophrenia, Rest & Flash

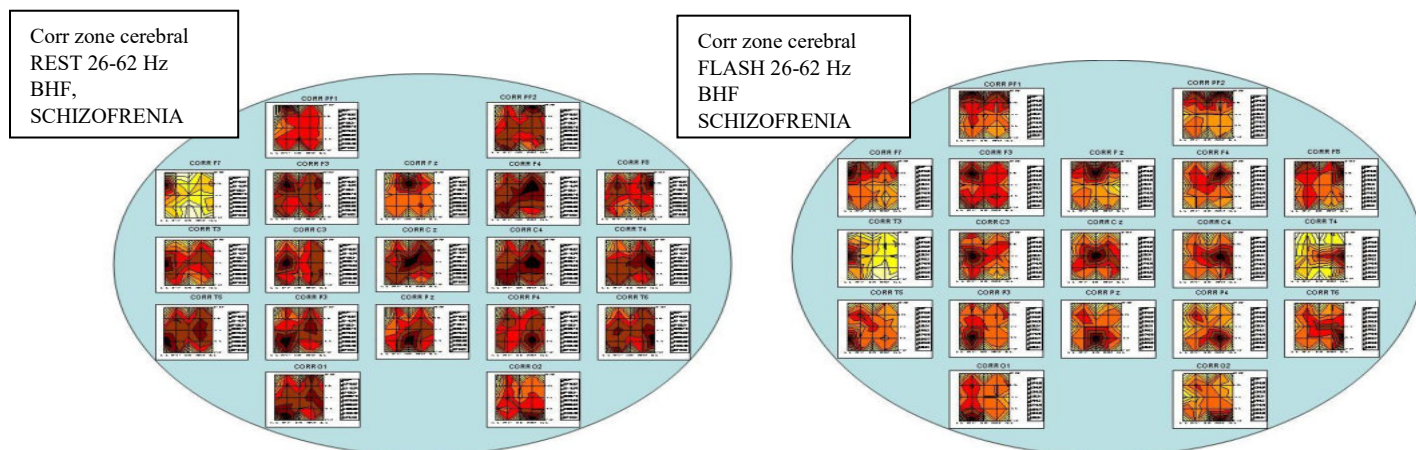


Fig. 5.32 Map with the spatial dispositions on the cortical areas of the zonal correlations of relative power in the BHF domain of frequency EEG 26-62 Hz, rest, Schizophrenia Disease - after N.S.Stanciu [12],[23],2020

Fig. 5.33 Map with the spatial dispositions on the cortical areas of the relative power zonal correlations in the BHF domain of frequency EEG 26-62 Hz, Flash, Schizophrenia Disease - after N S Stanciu [12],[23],2020

5.6.6.b.3. EEG weighted power zonal correlation on the Alpha Clinic 8–13 Hz, Flash, Normal domain – 30 cases

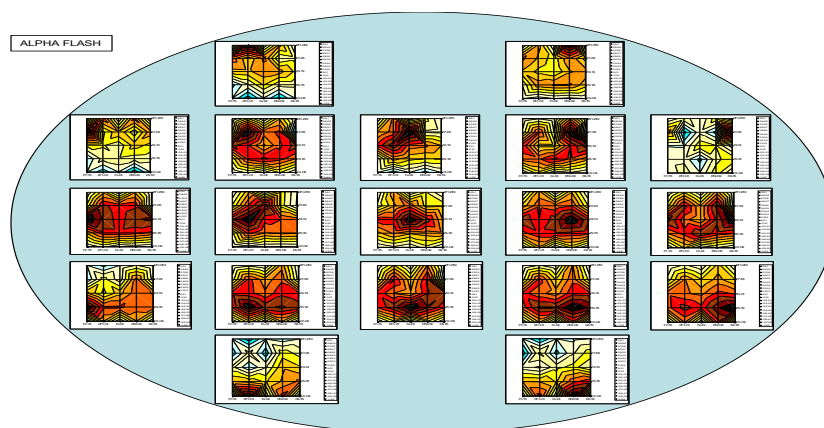


Fig. 5.39 Corr. Alpha Flash. Corel pattern in PF1-F7, F3-F4-T4-PF2, parietal-occipit P3-Pz-P4-T6, O1-O2. I distribute continuous symmetric values in pattern-after Stanciu N.S.[12],[23],2020

5.6.6.b.4. EEG Weighted Power Zonal Correlation on the Clinical Beta Range 13–26 Hz, Flash, Normal – 30 cases

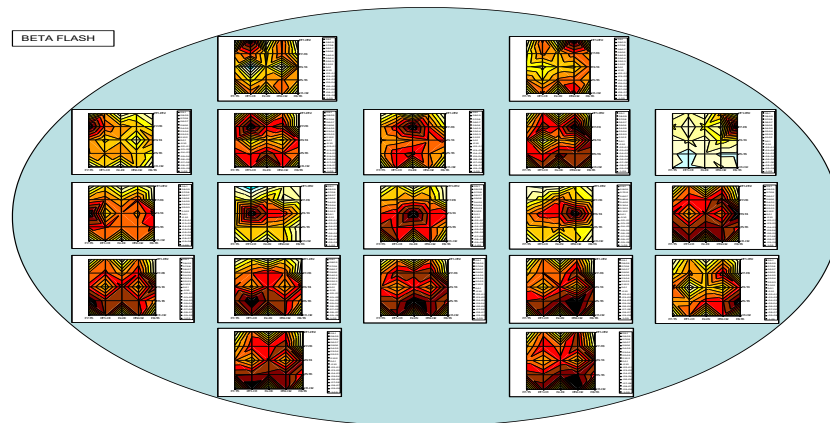


Fig. 5.41
Corr.Beta Flash.
Symmetric corel
pattern in PF1-
PF2-F7-F3-Fz-F8-
T3-T4,C3-
C4,temp-pariet-
occ. Distribution
of large
continuous waves,
in pattern-after
Stanciu N.S.
5121 5221 2020

5.7. Correlation analysis of EEG electrical signals. Frequency correlation of weighted power

5.7.1. Correlation of weighted electrical power associated with EEG, Normal and Pathological frequencies of electrical activity. Features

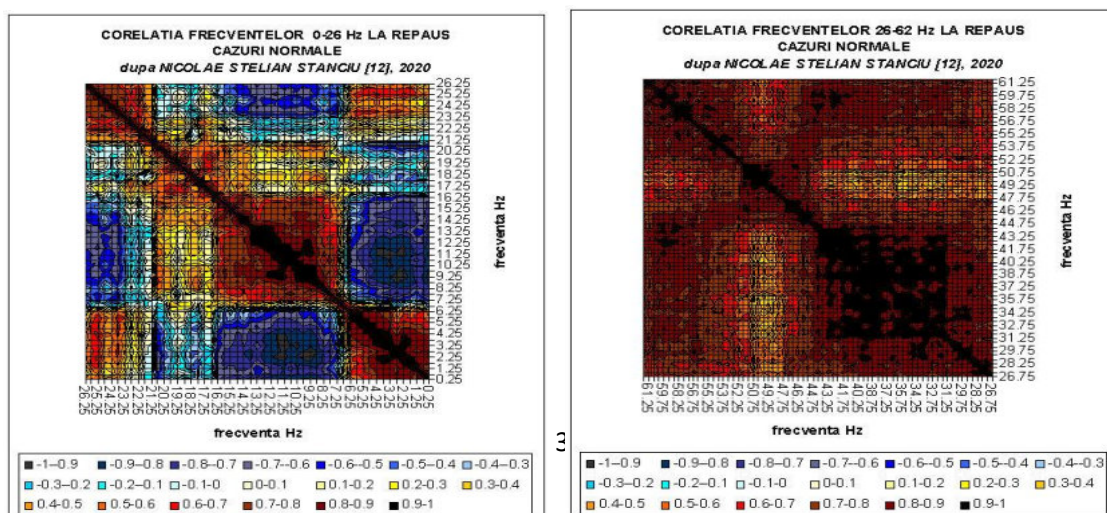
- There are well-defined frequency zones, which include values inside corelație fie pozitivă, fie negativă.
- In the normal case, there is an increase in the size of these areas that have internal correlation values of the same sign, with flash stimulation [43],[44].
- In the case of pathologies, two types of flash reactions can be distinguished:
 1. Scattering and segmentation of areas with medium and low correlation values but of the same sign on large areas of the frequency range (e.g. Obsessive Compulsive Syndrome)[44].
 2. The appearance within the frequency areas with a high degree of dysfunction of square areas containing very high correlation values, separated from each other by narrow frequency bands, both at rest and at flash (e.g. Schizophrenia, Autism, Alzheimer's) [12],[23],[44].

In order to measure the extension of the square areas and the dispersion of the functional pathology, the coefficient N was introduced. (see chapter 5, subchapter 5.76 and fig. 5.51–5.72, rel. 5.25)

5.7.1.a. Frequency-Weighted Power Correlation – Normal, Rest [12],[23]

Fig. 5.44 – Corel. in the Clinical, Normal, Rest domain – left

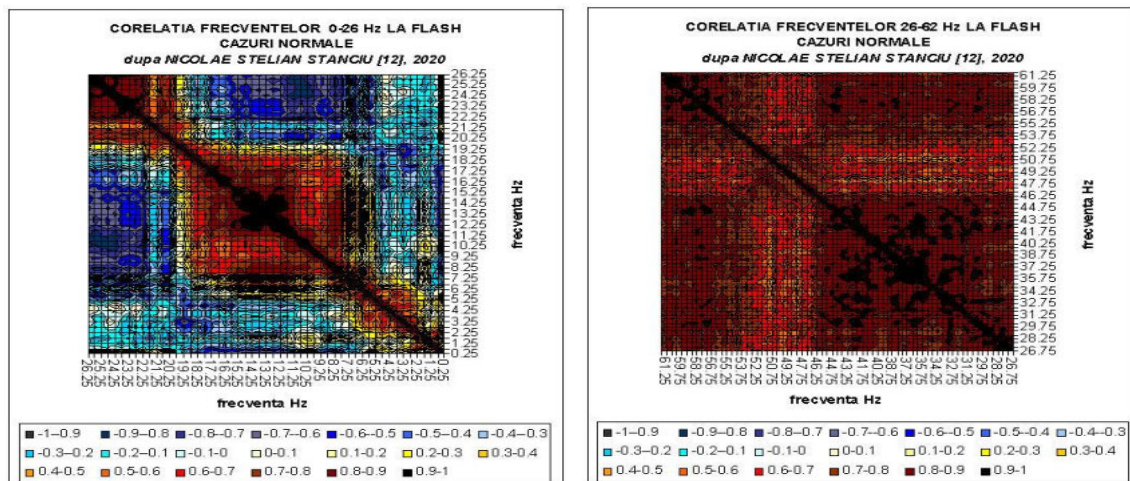
Fig. 5.45 – Corel. in the BHF domain, Normal, Rest – right



5.7.1.b. Frequency-weighted power correlation – Normal, Flash [12],[23]

Fig. 5.47 – Corel. in Clinical, Normal, Flash – left

Fig. 5.48 – Corel. in the BHF domain, Normal, Flash – right



5.7.3.a. Frequency-Weighted Power Correlation – Autism Disease, Rest [12],[23]

Fig. 5.56 – Corel. in Clinical, Autism, Rest – left

Fig. 5.57 – Corel. in the field of BHF, Autism, Rest – right

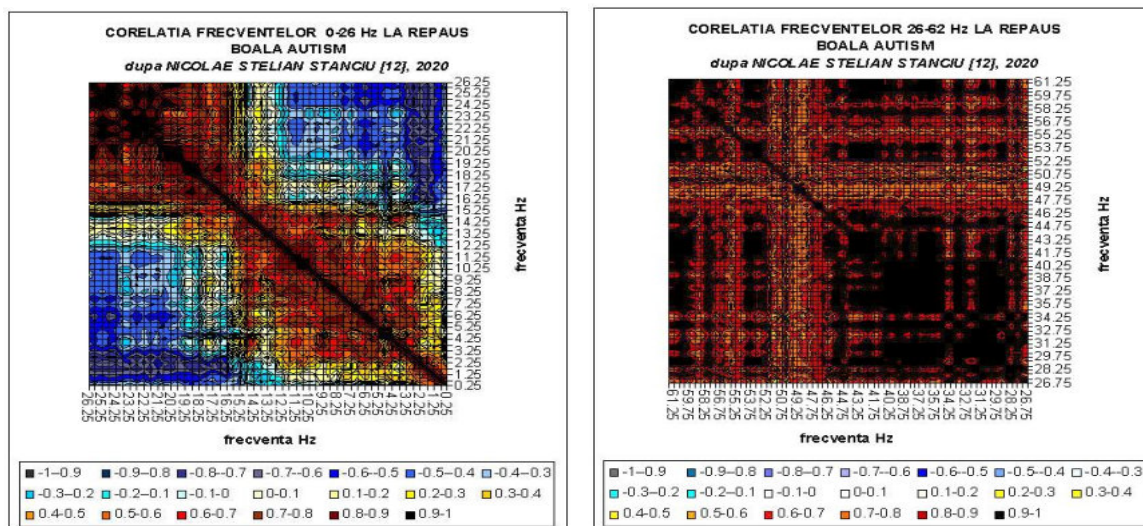


Fig. 5.56, Fig. 5.57 - pathological cases, autism disease,. Rest, Corel Map. Relative power associated with Frequencies in the Clinical and BHF domains – after Nicolae Stelian Stanciu [12],[23],[44],2020.

Note.

- In the Clinical domain, 2 distinct areas are observed, 0-15 Hz and 16-26 Hz, which have moderate correlation values within them and are practically negatively correlated. It is observed that the more the frequency of cortical activity increases, the more the correlation values within the separable domain increase. There are border areas, e.g. 15-16 Hz, which correlate both positively and negatively with various nearby frequency areas.

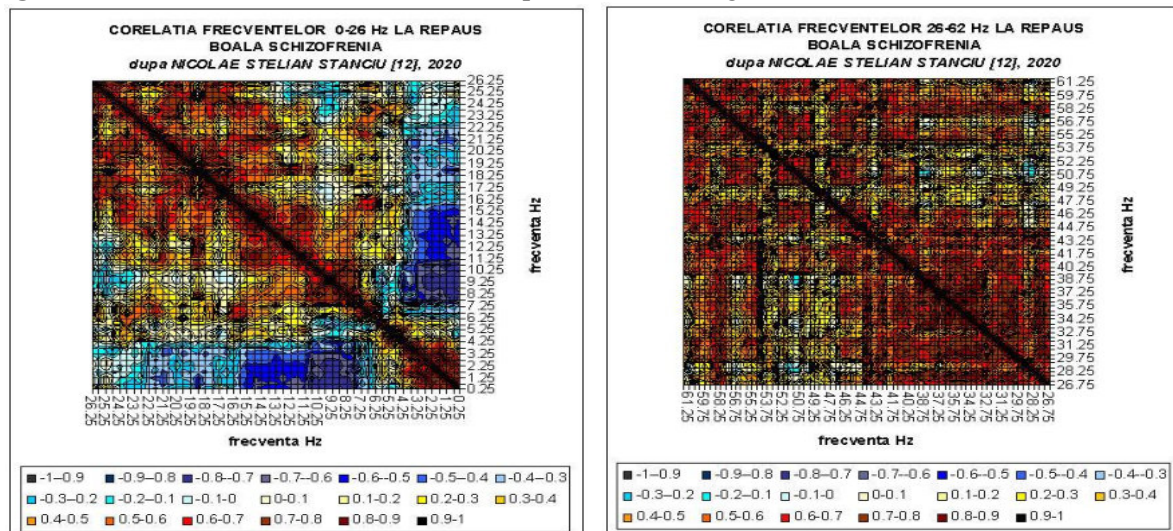
- In the BHF domain, it is observed the separation into 4 large, distinct correlation domains, which in turn form correlation clusters inside, like square correlation patterns, and each area in the cluster has 1.5-2 Hz x 1.5-2 Hz, having very high correlation values of weighted powers delimited inside (see page 110 below). The value of the frequency of 50 Hz corresponding to the minimum weighted power (v spectrum of BHF weighted power values, page 61, below), is the one that segments in the 4 quadrants the field of correlations BHF. De observed in the upper part of fig page 153, in quadrant II in the frequency range 50-62 Hz, the formation of square correlation clades, is a phenomenon that limits the high-performance cortical connections. [12],[23],[44].

5.7.5.a. Frequency-Weighted Power Correlation – Schizophrenia Disease, Rest

[12],[23],[44]

Fig. 5.65 – Corel. in Clinical, Schizophrenia, Rest – left

Fig. 5.66 – Corel. in the field of BHF, Schizophrenia, Rest – right



5.7.5.b. Frequency-Weighted Power Correlation – Schizophrenia Disease, Flash

[12],[23],[44]

Fig. 5.68 – Corel. in the Clinical field, Schizophrenia, Flash – left

Fig. 5.69 – Corel. in the field of BHF, Schizophrenia, Flash – right

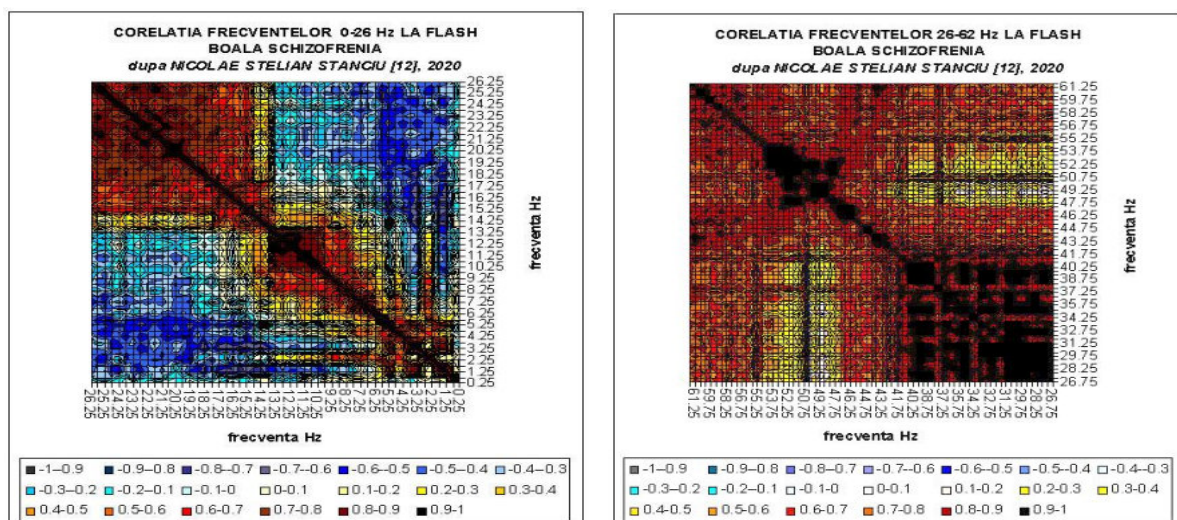


Fig. 5.68, Fig. 5.69 - Pathological Cases, Disease Schizophrenia, Flash, Coral Map Relative Power Associated Frequency on the Clinical Field and BHF. [12],[23],[44]. **Note.**

- In the Clinical field there are 3 distinct zones 0-6 Hz, 6-14 Hz, 14-26 Hz that have moderate or high correlation values within them. The Dom.Clinic has areas with positive or negative correlation values and the transition is gradual, or overlapping as continuous areas of values. The frequencies that divide the Clinical domain are 6 Hz, 10 Hz, 14 Hz and 17 Hz. There are cluster areas with a small area, with high correlation values, or inside another cluster with lower correlation values (e.g.: 10-14 Hz in 8-15 Hz), or in clusters of square pattern type (e.g. 0-4 Hz). These clusters can overlap.

- In the BHF domain, it is divided into 3 subdomains of dimensionally large correlation values, at the frequency of 49.54 Hz v. Fig. 5.69. Only at the lower limit of BHF is formed a sub-frequency range (26-40.5 Hz), which contains very high correlation values arranged in the form of a square pattern (2 Hz x 2 Hz).

If at rest see Fig. 5.66 the area with square patterns was arranged at higher frequencies of BHF, at flash v Fig. 5.69 it is arranged at lower frequencies of BHF. The quadratic patterns limit the qualitative cortical connections and lead to the idea of the existence of a positive zonal reaction, without interconnection, without the existence of cortical control between the quadratic areas that form the pattern, with a massive energy consumption of the respective structures in activity. This energy demand at the flash also explains the change in the position of the square correlation patterns towards lower frequencies of activity, which ensure a higher weighted power of the cortical activity performed at flash stimulation.— according to Nicolae Stelian Stanciu [12],[23],[44],2020.

Functional scattering coefficient of pathology

This coefficient denoted N is used in the EEG weighted power correlation analysis in relation to frequency, with the aim of highlighting the degree to which a neurocortical functional structure is affected by a specific pathology. It expresses the extent to which pathological elements are developed in neurostructure, reflecting the ability to transmit electrical signals between two cortical cortical areas, characterized by their frequencies and associated frequency domains.

The functional pathology level N can be defined in the space of correlation values and frequencies as being. the number of square-type areas existing in a frequency range of width $\Delta\nu$, which include in their inner area of width $\Delta\nu_0$, very high correlation values and which is bounded on its outside by the narrow frequency band, $\Delta\nu_m$ – the marginal frequency range. (see fig 5.71). The higher this N number of pathological areas in a given frequency range, the more structurally complex the pathology, or the more serious it is from a medical point of view and the more the value of the correlation increases. Ex.Flash (see fig 5.71, fig 5.72). The pathological level is equivalent to the degree of functional destructuring introduced by the existence of neuropsychiatric pathology. The pathological level was denoted with N - dimensionless number and was defined by the relationship:

$$N = \left(\frac{\Delta\nu}{\Delta\nu_0} \right)^2 = \left(\frac{\Delta\nu_0 + \Delta\nu_m}{\Delta\nu_0} \right)^2 = \left(1 + \frac{\Delta\nu_m}{\Delta\nu_0} \right)^2 \quad (5.23)$$

- The higher the N number, the more structurally complex or medically serious the pathology.
- The pathological level N is equivalent to the degree of functional destructuring introduced by the neuropsychiatric pathology.
- $\Delta\nu_m$ delimits the area with very high correlations from the neighboring areas with low values, behaving as an isolated system in terms of transmission and energy consumption.
- Within the square areas, the high correlation values are supported by a linearity that requires a high energy consumption, which can lead to the biological degradation of the neuronal structure.
- This coefficient does not directly indicate the affected brain area, but the frequency range associated with the pathology.
- It has been used in the description of pathologies: Autism, Schizophrenia, S.O.C., Bipolar Syndrome, Alzheimer's.

The more extensive the marginal domain, having a large share of the domain with high correlation values, the aspect becoming that of a point area with very high correlation values, the higher the pathological level of functional destructuring. $\Delta\nu_m$ – the marginal frequency domain, is the one that delimits the area with very high correlation values from the other areas of close frequency having low correlation values. Everything behaves like a practically isolated system in terms of signal transmission and energy consumed. where they are dominated by the existence of linearity, a phenomenon that in order to be sustained, requires a very high input of energy, which ultimately leads to the organic, biological degradation of the signal-emitting neuronal structure. This indicator of functional destructuring does not directly and precisely highlight the organic brain area structurally affected by the pathology, but only the frequency range associated with the existence of the specific pathology. The phenomenon of functional destructuring referred to in this doctoral thesis was used by the author in describing the pathology associated with Autism and Schizophrenia, being also noticed in the case of S.O.C., Bipolar Syndrome and Alzheimer's disease.

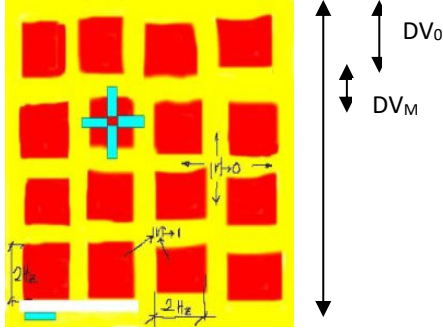


Fig 5.71 Numbers of pathological square areas

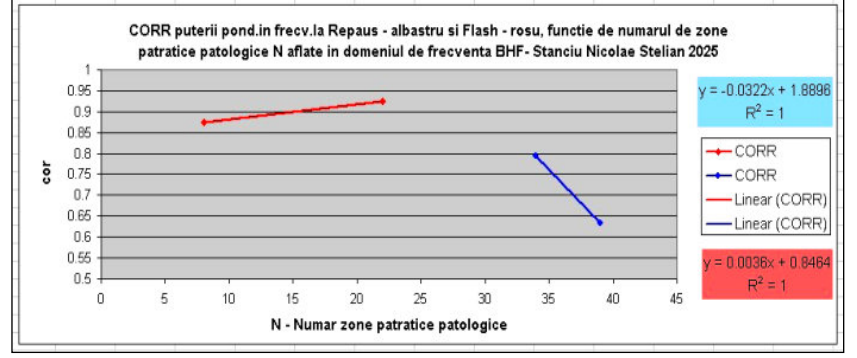


Fig 5.72 Weighted power correlation - mean in frequency as a function of N, number of pathological square areas in BHF. Stanciu Nicolae Stelian 2025

5.7.7. The pathology indicator using the EEG weighted power correlation in frequency domain - I.N.C.

I.N.C. – Indicator of Non-Correlation Compensation

It is an indicator of the existence and development of neuronal pathology, expressing the degree to which the sum of the correlation values associated with the EEG-weighted power in the average frequency domains Alpha and Beta 1 is compensated by the sum of the correlation values in the Delta, Beta 2 and BHF domains.

- Correlation compensation is associated with the state of normality.
- Non-compensation is associated with neuropsychiatric pathology.
- The analyzed range is 0–62 Hz, covering the entire neurophysiological activity.

The definition of I.N.C.

The total average of the correlation compensations is considered:

$$1. A_i = [\text{CORR}(\text{ALFA}) + \text{CORR}(\text{BETA } 1)]_i$$

$$2. B_i = [\text{CORR}(\text{DELTA}) + \text{CORR}(\text{BETA } 2) + \text{CORR}(\text{BHF})]_i,$$

where i is the index associated with the Hz-mediated frequency range.

| | | | | | | |
|------|------|-----|----|-------|-------|----|
| 2.25 | 6.25 | 9.5 | 12 | 15.75 | 22.25 | 44 |
|------|------|-----|----|-------|-------|----|

I.N.C is defined as the average of the correlation values on the fields indicated above

$$\text{I.N.C} = \frac{\sum_{i=1}^n (A_i + B_i)}{n} \quad (5.24)$$

where i is the index associated with the average frequency range. It has been observed that the phenomenon of correlation compensation occurs both at Rest and at Flash, for all Normal or Pathological categories. The I.N.C. was determined to be both Normal and Pathological - S.O.C. Bipolar SB Syndrome, Schizophrenia, Alzheimer's disease and Autism rest. It was found that in the Normal case there are the lowest values for I.N.C., compared to all types of pathologies investigated, both in Rest and Flash. It has been estimated from the experimentally observed that in the ideal case Normal, so that I.N.C = 0 so that:

$$\overline{A} + \overline{B} = 0 \Rightarrow \frac{\overline{A}}{\overline{B}} = -1, \text{adică } \frac{\sum_{i=1}^n A_i}{\sum_{i=1}^n B_i} = -1, \text{unde } \frac{\sum_{i=1}^n A_i}{n} = \overline{A}, \text{iar } \frac{\sum_{i=1}^n B_i}{n} = \overline{B}, \quad (5.25)$$

I.N.C Features

1. In the normal case, the I.N.C. values have the smallest, close to zero.
2. Ideally: I.N.C. values indicate a functional control system of EEG correlative activity.
3. The phenomenon of compensation occurs both at rest and in flash, in all categories (Normal and Pathological).

This relationship of the I.N.C (5.25) shows us that this phenomenon of compensation of the correlations of weighted power, exists in the entire range 0-62 Hz. of the frequencies of neuronal activity, which implies the existence of a control system of the functional correlation activity, associated with the correlation of the frequencies of the activity of the type of a reaction system.

5.7.8 . Correlation compensation effect. Exemplification NORMAL

Table 5.11

Correlation values by mean frequency domains. Table up-Sleep, down-Flash, Normal

| CORR MEDIE FRECV.REPAUS NORMAL | | | | | | | | |
|--------------------------------|------------|----------|----------|----------|----------|----------|----------|--|
| | 2.25 | 6.25 | 9.5 | 12 | 15.75 | 22.25 | 44 | |
| 2.25 | 1 | 0.138638 | -0.96425 | -0.92009 | -0.85503 | 0.071908 | 0.614867 | |
| 6.25 | 0.1386383 | 1 | -0.07325 | -0.10496 | 0.061399 | 0.128121 | -0.08547 | |
| 9.5 | -0.9642482 | -0.07325 | 1 | 0.958263 | 0.894397 | -0.15033 | -0.65191 | |
| 12 | -0.9200904 | -0.10496 | 0.958263 | 1 | 0.855341 | -0.25367 | -0.70418 | |
| 15.75 | -0.8550335 | 0.061399 | 0.894397 | 0.855341 | 1 | -0.01819 | -0.67054 | |
| 22.25 | 0.0719082 | 0.128121 | -0.15033 | -0.25367 | -0.01819 | 1 | 0.476301 | |
| 44 | 0.6148666 | -0.08547 | -0.65191 | -0.70418 | -0.67054 | 0.476301 | 1 | |

| CORR MEDIE FRECV.FLASH NORMAL | | | | | | | | |
|-------------------------------|----------|----------|----------|----------|----------|----------|----------|--|
| | 2.25 | 6.25 | 9.5 | 12 | 15.75 | 22.25 | 44 | |
| 2.25 | 1 | -0.256 | -0.58312 | -0.78155 | -0.9047 | 0.089981 | 0.165135 | |
| 6.25 | -0.256 | 1 | 0.477534 | 0.461229 | 0.353611 | -0.64784 | -0.65606 | |
| 9.5 | -0.58312 | 0.477534 | 1 | 0.90335 | 0.768266 | -0.66792 | -0.76302 | |
| 12 | -0.78155 | 0.461229 | 0.90335 | 1 | 0.932646 | -0.61355 | -0.6748 | |
| 15.75 | -0.9047 | 0.353611 | 0.768266 | 0.932646 | 1 | -0.39145 | -0.4407 | |
| 22.25 | 0.089981 | -0.64784 | -0.66792 | -0.61355 | -0.39145 | 1 | 0.899608 | |
| 44 | 0.165135 | -0.65606 | -0.76302 | -0.6748 | -0.4407 | 0.899608 | 1 | |

Table 5.12

Calculation of the coefficients A,B that enter into the relation of the compensation effect and the index of non-compensation of the I.N.C. Correlations Table Up-Rest, Bottom-Flash, Normal

| CALCUL I.N.C REPAUS NORMAL | | | | | | | | |
|----------------------------|--|----------|----------|----------|----------|----------|----------|----------|
| | A = CORR (ALFA) + CORR(BETA1) & B = CORR (DELTA) + CORR(BETA2) + CORR(BHF) | | | | | | | |
| Frecv.Hz | 2.25 | 6.25 | 9.5 | 12 | 15.75 | 22.25 | 44 | |
| A | -0.913124 | -0.03894 | 0.950887 | 0.937868 | 0.916579 | -0.14073 | -0.67555 | |
| B | 0.5622582 | 0.060429 | -0.58883 | -0.62598 | -0.51459 | 0.51607 | 0.697056 | I.N.C |
| MEDIE A,B | -0.1754329 | 0.010746 | 0.181027 | 0.155944 | 0.200994 | 0.187669 | 0.010755 | 0.571703 |

| CALCUL I.N.C FLASH NORMAL | | | | | | | | |
|---------------------------|--|----------|----------|----------|----------|----------|----------|----------|
| | A = CORR (ALFA) + CORR(BETA1) & B = CORR (DELTA) + CORR(BETA2) + CORR(BHF) | | | | | | | |
| Frecv.Hz | 2.25 | 6.25 | 9.5 | 12 | 15.75 | 22.25 | 44 | |
| A | -0.75646 | 0.430791 | 0.890539 | 0.945332 | 0.900304 | -0.55764 | -0.62618 | |
| B | 0.418372 | -0.51997 | -0.67135 | -0.68997 | -0.57895 | 0.663196 | 0.688248 | I.N.C |
| MEDIE A,B | -0.16904 | -0.04459 | 0.109593 | 0.127682 | 0.160678 | 0.052779 | 0.031036 | 0.268136 |

5.7.8.atag. The effect of compensating the value of the frequency-weighted power correlations Rest -Left and Flash- Right.Normal.

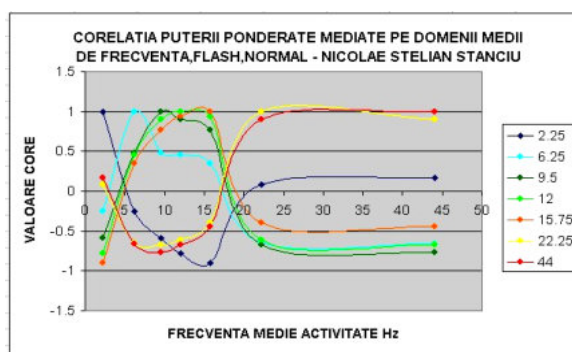
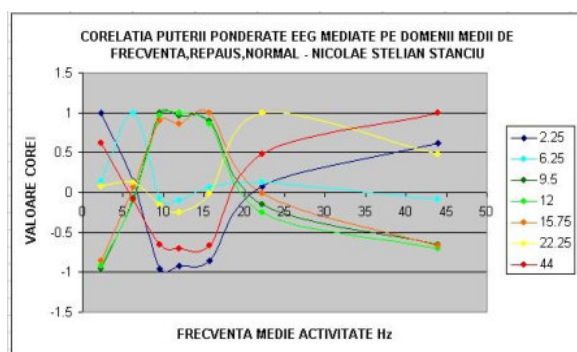


Fig 5.7.8

Correlation curves by frequency range 0-62 Hz Normal. Left Fig ; Flash-fig right.

Note.

The compensation effect is observed - Stanciu Nicolae Stelian 2025

5.7.9.a. The effect of compensating the value of the frequency-weighted power correlations Rest and Flash.Pathological – S.O.C.

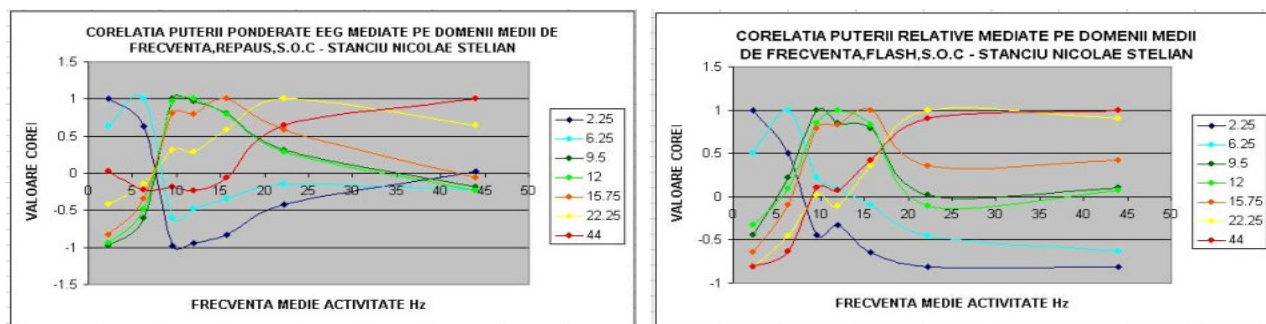


Fig.5.76,a,b: Compensation effect of the value of correlations on central and extreme domains of medium frequency,Pathological,S.O.C.. EEG-weighted power correlation, mediated by mean frequency domains, Rest, Pathological, S.O.C. **a--Left.-Rest & b-Right -Flash.**Stanciu Nicolae Stelian 2025

5.7.10. Conclusion on I.N.C.

Table 5.

I.N.C. to NORMAL and PATHOLOGICAL

| | I.N.C REPAUS | I.N.C. FLASH |
|----------------|--------------|--------------|
| NORMAL | 0.571703109 | 0.268136261 |
| S.O.C. | 1.054634222 | 1.335183808 |
| S.B. | 1.419761879 | 0.710680072 |
| SCHIZO | 1.679682809 | 0.711731748 |
| ALZHEIM | 0.961537595 | 0.586671941 |
| AUTISM | 1.369127498 | |

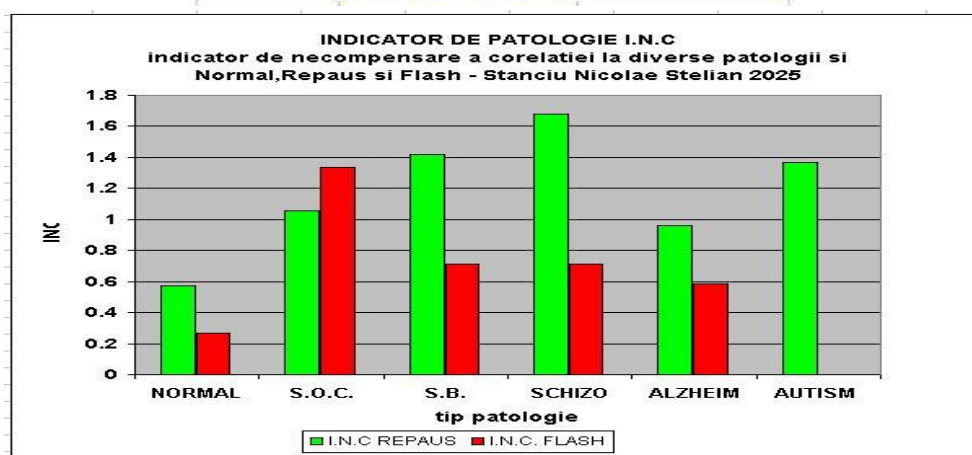


Fig.5.78tag. The pathology indicator I.N.C.la Normal and Pathological, Rest-green, Flash-red. Stanciu Nicolae Stelian [12]

Important! From what has been shown in this subchapter, it follows that the phenomenon of correlative activity in the frequency domain is very important because it highlights the transfer of electrical signal associated with all the frequency zones of the entire neurophysiological activity. The control of the value of the EEG-weighted electrical power correlation index is absolutely necessary because these values under certain conditions can lead to the appearance of pathology. This control is carried out in the case of the correlation of the EEG weighted power by frequency, by the phenomenon of compensation of these correlation values on certain frequency domains of cortical activity. The introduction of the index of non-compensation of the I.N.C. correlation is necessary because it shows the existence of a control system of the cerebral correlative activity, as well as the initiation and development of neuronal pathology when this control system is deficient or the pathology is very serious and affects this control system itself.

5.8. VEP Latency and Amplitude, Normal Case

Basis of VEP correlation analysis

5.8.1. Experimental VEP amplitudes at White Flash stimulation energies (0.3 J, 0.6 J, 1.2 J) for Right Hemisphere and Left Hemisphere, Normal case.

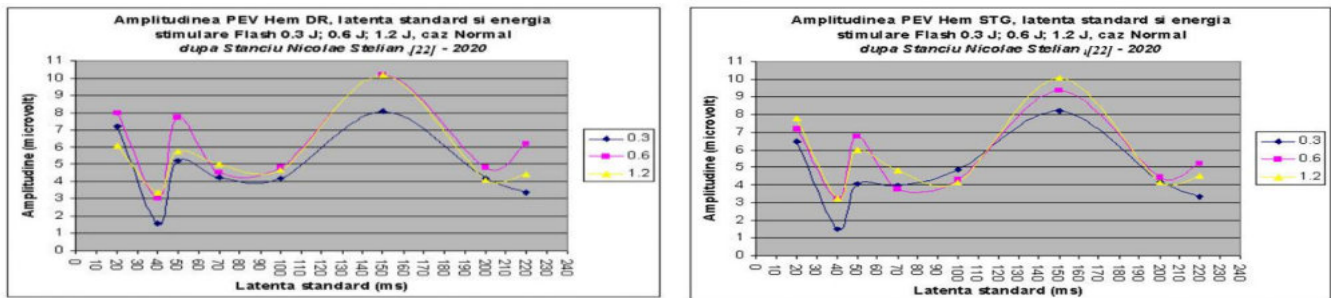


Fig 5.79 (a,b). The amplitudes of VE[at **Right Hem.(fig. left-a)** and **Hem.Left (fig. right-b)** in microvolts at White Flash stimulation with 0.3 J ; 0.6 J ; 1.2 J, The values of the amplitudes of the VEP signal are measured relatively, peak by peak, depending on the standard latency in milliseconds - ms. after Stanciu Nicolae Stelian [22],[43].

5.8.2. Experimental latencies of the VEP components at the same stimulation energies, for the Right Hemisphere and the Left Hemisphere, Normal case.

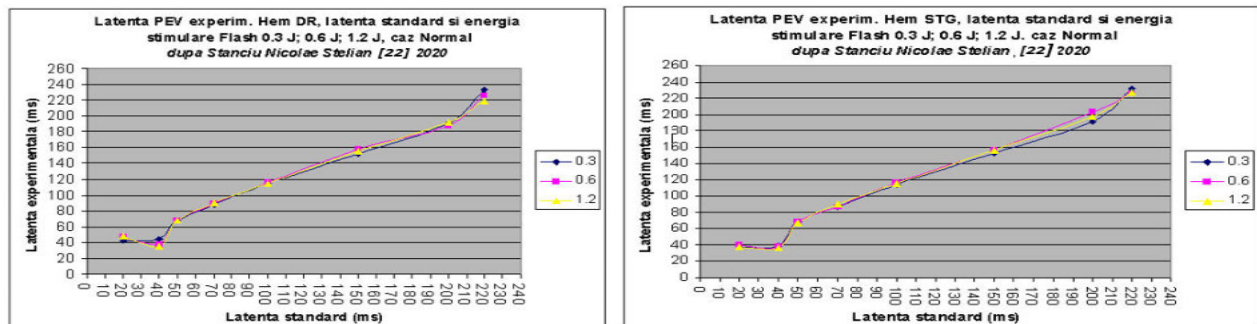


Fig 5.80 (a,b). Experim. comp. VEP latencies at **Hem. Right (left -a)** and **Hem. Left (right-b)** at White Flash stimulation with 0.3 J ; 0.6 J ; 1.2 J in milliseconds. The values of the latencies of the components of the VEP signal are measured relative to the peak level of signal amplitude.

5.9. Correlation analysis of VEP electrical signals, Normal

5.9.1. Characteristics of the VEP Amplitude and Latency Correlation Phenomenon

a) Correlation of amplitudes of VEP components – characteristics

1. Correlation values depend on the type of neural structures and processing pathways
2. In the Normal case, neural structures correlate differently with Flash stimulation
 - High positive correlation for excitative structures (N40, N70, N150, N220).
 - Negative correlation for inhibitory structures (P50, P100, P200) and retina (N20).
 - The situations are illustrated in graphs for both hemispheres.
3. High correlation between the Hippocampus and the CGL → the existence of the thalamic filter and visual awareness.
4. The reticulated formation correlates positively with flash energy and excitative structures, not with inhibitory ones.
5. The high amplitude correlation implies optimal energy consumption. Loss of this control → pathology.
6. In pathological cases, large deviations from normal values occur.

b) Correlation of the latencies of the VEP components – characteristics

- The latency correlation reflects the linearity of the propagation rate.
- High positive correlation if the velocity increases with the flash energy.
- Negative correlations for CGL and Hippocampus.
- Most of the components correlate positively, exceptions: CGL-N40,P50,Hippoc-N220.
- High correlation → pronounced linearity between speeds.
- The coherence of graphic forms → high correlation.
- High level of coherence in the CGL and Hippocampus, required for thalamic gates.
- The high amplitude correlation implies optimal energy consumption.
- In pathology, there are large deviations from normal.

5.9.2. VEP Amplitude Correlations. Overall treatment

At the White Flash energies (0.3 J, 0.6 J, 1.2 J), for the Right Hemisphere and the Left Hemisphere, Normal.

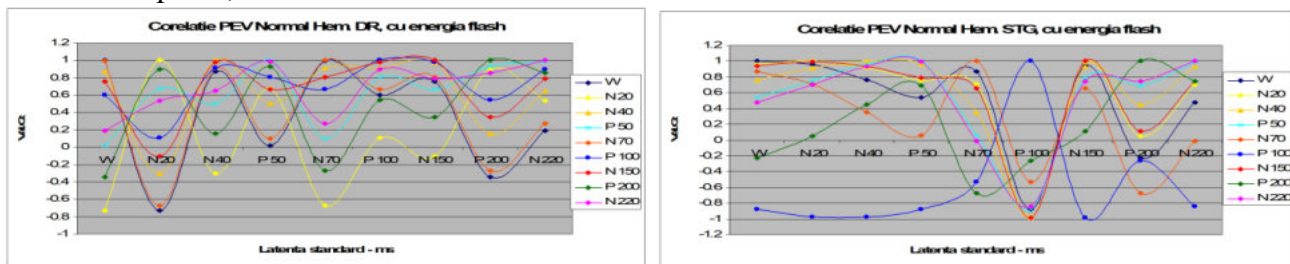
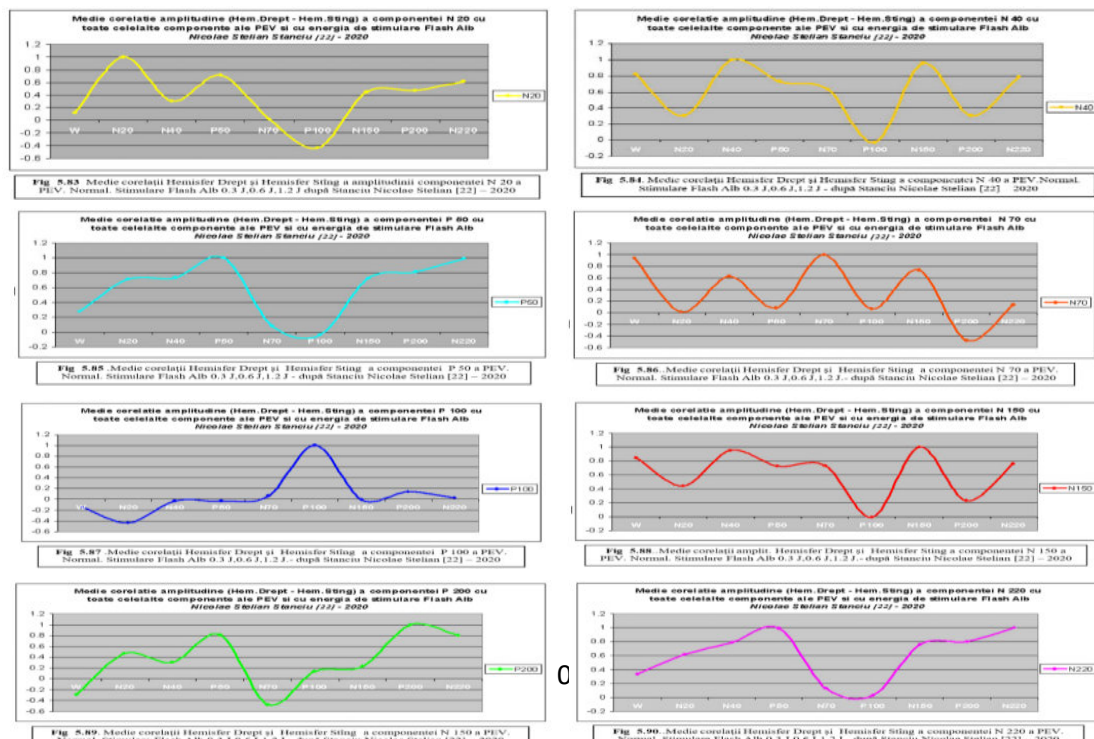


Fig.. 5.81.a,b. Corr VEP Hem DR, a-left, Hem STG, b-right. **Note** The correlation coefficient of the flash stimulation energy, which has a high positive value, is observed if the structures that process the electrical signal sent on the optic nerve are of the excitative type, which have the components PEV, N40, N70, N150, N220. Due to the existence of a retinal adaptation system to flash stimulation, there may be situations in which negative correlations occur with the N 20 component associated with the retina - v Hem DR. Notice the value of the correlation coefficient of the flash stimulation energy also has a negative value, if the structures are of the inhibitory type P50, P100, P200. These structural types oppose neuronal stimulation. Situations are at Hem DR

5.9.4. Average correlation of amplitudes of VEP components

5.9.4.a. Mean amplitude correlation PEV Hem.Left – Right Hem. Normal, Separate treatment[22].



5.10.1. VEP amplitudes and latencies

5.10.1.a. Amplitudes and latencies of VEP Pathological case, Right Heme and Left Hem. White Flash of 0.3 J,0.6 J, 1.2J, Neuropathy

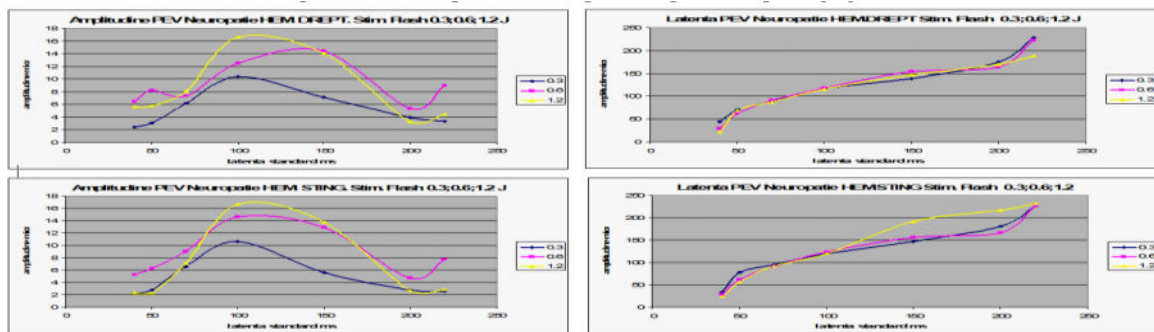


Fig 5.93 .a,b. a-stg Amplitude,b-dr PEV Signal Latency, Pathology, Neuropathy, White Flash Stimulation 0.3,0.6,1.2 J.- after Stanciu Nicolae Stelian [22] – 2020

5.10.2.a Amplitudes and latencies of VEP at White Flash state energies 0.3 J, 0.6 J, 1.2J, for Right and Left Hemisphere, Pathological, Epilepsy Left Hemisphere Focus.

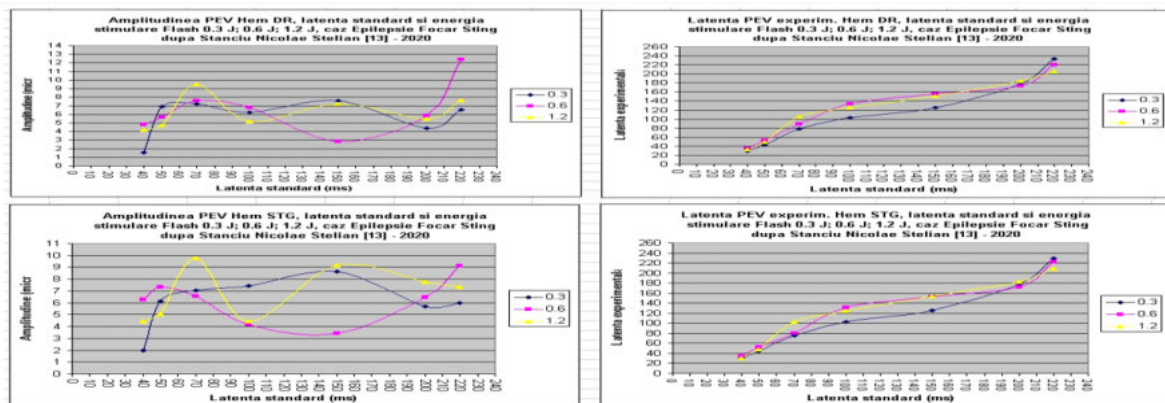


Fig 5.94 .a,b. a-stg Amplitude,b-dr VEP signal latency, Pathology,Epilepsy Heme Focus.Sting, White Flash Stimulation 0.3,0.6,1.2 J.- after Stanciu Nicolae Stelian [22] – 2020

5.10.3 VEP amplitude reaction control system

BETA Response Coefficient for Normal, Neuropathy and Epilepsy Heme Outbreak. Left

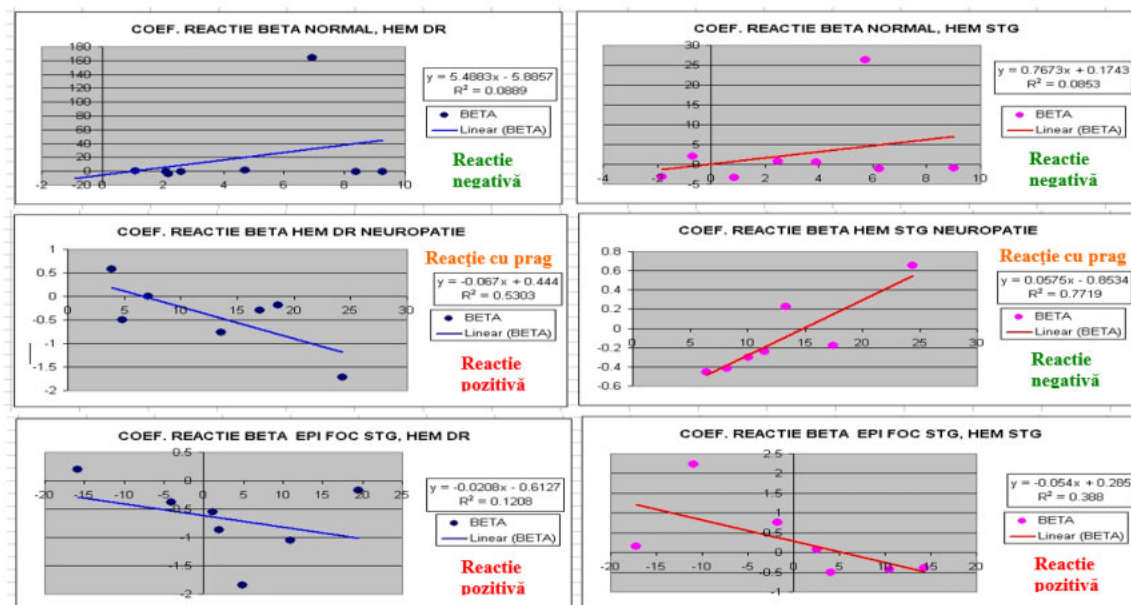


Fig 5.95 .a,b.Reaction coefficient Beta,a-fig left Hem DR and b-fig right Hem STG of the reaction system for VEP amplitude control. Types of reactions:Normal,Neuropathy,Epilepsy Heme Focus.STG, White Flash Stimulation 0.3.0.6.1.2 J.- Stanciu Nicolae Stelian [22] – 2020

5.10.4 VEP amplitude reaction control system.

Characteristics and Conclusions – Beta Pathology Indicator

Beta is the pathology indicator and is defined as;

$$\beta = \frac{A_0 - A}{A_0 A} \quad (5.26)$$

where $A_0 = TG\ 0.3 = \frac{A_{0.6} - A_{0.3}}{A_{0.6}}$, and $A = TG\ 0.6 = \frac{A_{1.2} - A_{0.6}}{A_{1.2}}$ and $\beta > 0$ – negative reaction, $\beta < 0$ – reacție pozitivă. (5.27). β is the reaction coefficient of the amplifier.

Talk v.fig 5.95 :

1. In Normal case: negative reaction to increased amplitude → adaptive control. Threshold = 0.
2. In Neuropathy: Hem.DR requires $\beta < 0$ (positive compensation). Hem.STG has a non-zero threshold.
3. In Epilepsy STG outbreak: $\beta < 0$ constant → generalized positive reaction, with no threshold → Amplified arousal.

Conclusion:

It is necessary to use the size of the Beta β reaction coefficient as a direct indicator in the analysis of neuronal pathologies that lead to direct effects on the control system of the amplitude of the VEP signal.

5.11. Correlation analysis of VEP signals Pathological

5.11.1. VEP Amplitude Correlations to White Flash energies (0.3 J, 0.6 J, 1.2 J), for Right Hemisphere and Left Hemisphere, Pathological case – Neuropathy. Treatment as a whole.

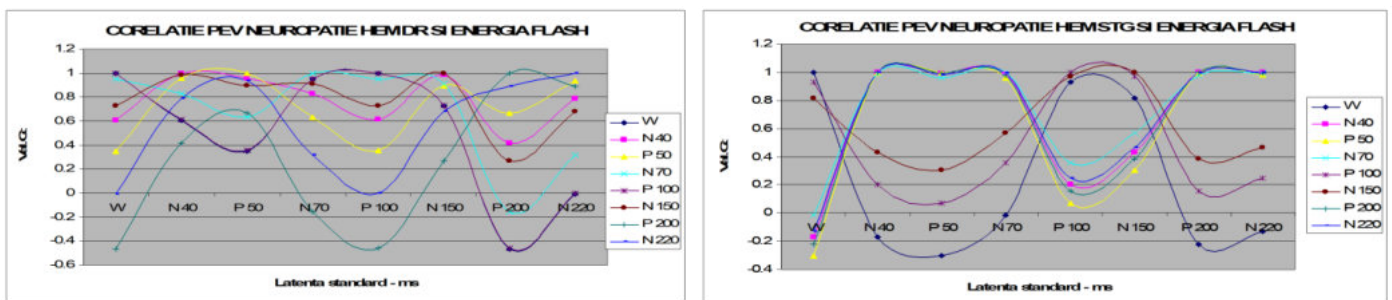


Fig 5.96 a,b. Amplitude correlation values of the VEP. Right Hem- up, Left Hem- down, Pathology, Neuropathy, White Flash Stimulation 0.3.0.6.1.2 J.- after Stanciu Nicolae Stelian [221.441] – 2020

5.11.2. Latency correlations of VEP components to White Flash energies (0.3 J, 0.6 J, 1.2 J), for Right Hemisphere and Left Hemisphere, Pathological case – Neuropathy. Treatment as a whole.

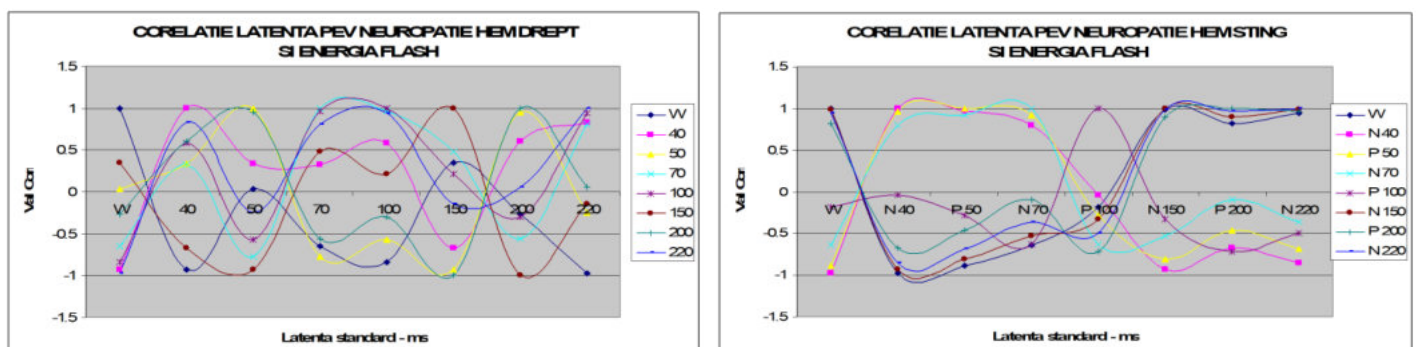


Fig 5.97 a,b. Latency correlation values of VEP components. Right Hem - up, Left Hem b- down, Pathology, Neuropathy, White Flash Stimulation 0.3,0.6,1.2 J.- after Stanciu Nicolae Stelian [22] –

Chapter 6. New fundamental phenomena of neuronal and neurocortical activity

6.1. The model of the dependence of the VEP signal on the energy of Flash stimulation and Adrian E.D.'s Law for White Flash stimulation (0.3 J, 0.6 J, 1.2 J), Left and Right Hemisphere.

- Information and representations from [19], 2021 were used.
- A biophysical relationship was introduced for the approximation of the experimental VEP, respecting the phenomena described by Adrian Edgar Douglas (1926) regarding the excitation of the nerve fiber and the processes related to neuronal processing.

Thus we used for modeling the VEP relationship :

$$(a * \text{Log}[y] + c) * \frac{\text{Exp}\left[x^{\frac{1}{3}}\right]}{x} * \left(\text{Cos}\left[\frac{2\pi}{200}x + d\right]\right)^2 + b \quad (6.1)$$

The terms of the experimental data estimation relationship have the following meanings :

$a * \text{Log}[y] + c$ - (6.2) - It is a term that is related to the phenomenon of increasing the frequency of nerve fiber discharges, which have the effect of a logarithmic increase in the abundance of the signals recorded with the increase in the energy of stimulation y . It is a term that introduces the Adrian E.D law to Flash stimulation. fiber related to the optical tract.

$\frac{\text{Exp}\left[x^{\frac{1}{3}}\right]}{x}$ - (6.3) - It is a compound term that actually contains two terms. The term from

the numerator shows that there are X neural structures that due to the multitude of types. and the large number of neurons that process information are structures that generate amplitudes of higher electrical potential, compared to those whose number of neurons is small. Thus, the neural structures of the RF will result in higher VEP processing than in the case of the CGL structure. The exponential factor expresses the phenomenon of neuronal acquisition for rapid signal processing, i.e. the phenomenon of neuronal recruitment.

Termul de la numitor $\frac{1}{x}$ - (6.4) indicates the phenomenon described by Adrian E.D related to adaptation to stinging. This term is taken into account, since there was no simultaneous excitation at the various Flash energy values, and between the moments of stimulation with different Flash energies there was a temporal gap in which the adaptation of the nerve fiber took place.

$\left(\text{Cos}\left[\frac{2\pi}{200}x + d\right]\right)^2$ - (6.5) .It is a quadratic term that depends on standard latency. This term

indicates the oscillatory character of the winding curve of the distribution of the electrical potential distributions with respect to the standard latency. The simultaneous existence of the described phenomena is indicated by the operation of multiplication between the terms that enter into the definition of the fit model. Finally, the relationship that approximates the experimental VEP is represented in 2D and 3D for the various energies of the Flash and the two cerebral hemispheres.

Final observation: The model simultaneously integrates the phenomena of logarithmic growth, neuronal recruitment, adaptation and oscillation. The relationship (6.1) approximates the experimental VEP and is plotted (2D and 3D) for various Flash energies and both cerebral hemispheres.

6.1.1. 3D comparative representation of VEP in logarithmic approximation, for the Right Hemisphere (left fig.) and Left Hemisphere (right fig.), at White Flash 0.3–1.2 J

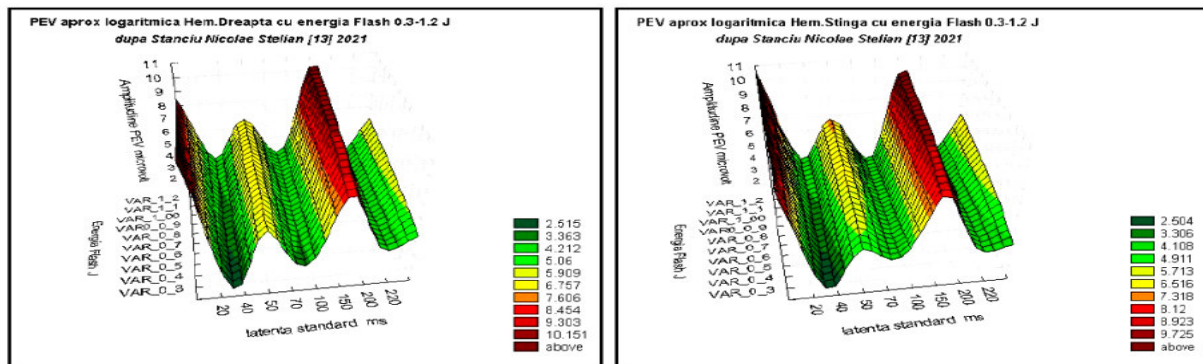


Fig 6.1.a,b. 3D comparative VEP representation in approx.logarithmic **Hem.DR** – **a, left**, and **Hem STG** – **b, right**. **Note** It can be seen from the 3D maps that the form of the VEP signal for both Hem DR and Hem STG is similar, being related to the logarithmic dependence of the amplitude as a function of the Flash stimulation energy. however, being of the inhibitory type, the logarrhythmic function has a negative sign, the minus sign showing that they oppose the excitation. There is also the retinal receptor system, which has included in its operation an adaptive system to flash excitation, which can provide inhibitory or excitatory reactions. [13],[19]

6.1.2. Logarithmic dependence of VEP amplitudes on stimulation energy Flash,Hem. DR & ST

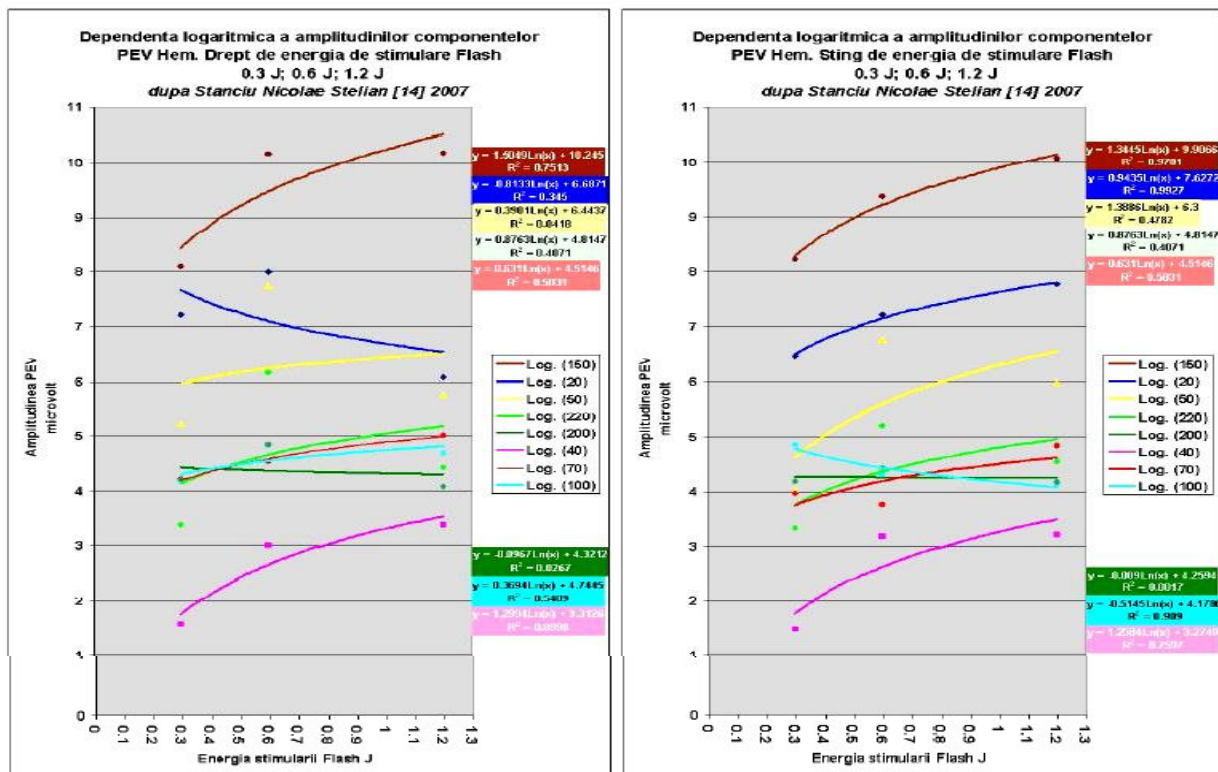


Fig. 6.2 Kogaritmnic Dependence of VEP Amplitudes **Hem DR** - **Left Fig** and **Hem STG** - **Right Fig** and White Flash Energy

Note. The neuro-functional links between the logarithmic dependencies of the amplitudes of the horns generating VEP on the energy of Flash stimulation highlight the direct connection between them and Adrian E.D.'s law. Right (see Fig left) as well as for Hem.Left (see Fig right). For both the excitatory and inhibitory components, Adrian E.D.'s law can be applied, bearing in mind that the latter produce a decrease in the excitation of the structure. In addition, there are retinal control systems with an adaptive role that regulate the excitatory level of the retina.

6.1.3. The model of the dependence of the amplitude of the VEP signal on the energy of Flash stimulation and Adrian E.D.'s law for the Left Hemisphere [19]

a.4 . VEP Left Hemisphere to Flash Stimulation Energies:

0.3 J (Blue), 0.6 J (Green), 1.2 J (Red)

```
Plot[ { 4.026475389889336` +  $\frac{1.3547482528545511` e^{x^{1/3}} \cos[1.5239637014257121` + \frac{\pi x}{100}]^2}{x}$ ,  
4.026475389889336` +  $\frac{2.275720363795404` e^{x^{1/3}} \cos[1.5239637014257121` + \frac{\pi x}{100}]^2}{x}$ ,  
4.026475389889336` +  $\frac{3.1966924747362575` e^{x^{1/3}} \cos[1.5239637014257121` + \frac{\pi x}{100}]^2}{x}$  }, {x, 20, 220},  
PlotStyle -> {{RGBColor[0, 0, 1]}, {RGBColor[0, 1, 0]}, {RGBColor[1, 0, 0]}}
```

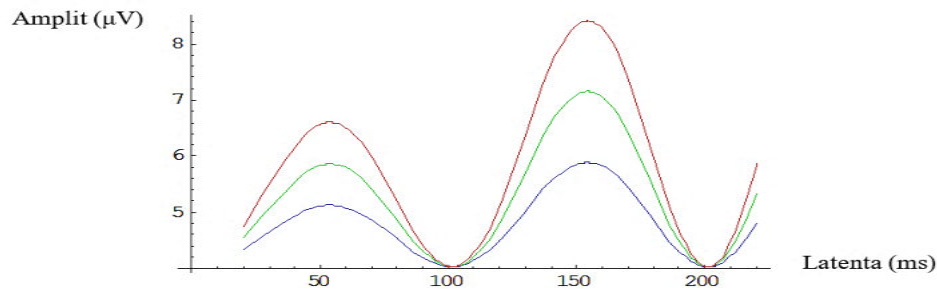


Fig 6.6 VEP Hem.Sting signal at flash stimulation White 0.3 J (blue),0.6 J (green),1.2 J (red),obtained with the function performed in the model of the dependence of the amplitude of the VEP components on the energy of flash stimulation Nicolae Stelian Stanciu [13],[19]

a.5. Dependence of the Left Hemisphere Visual Evoked Potential on stimulation energy and standard latency. 3D visualization variant.

Expresia modelului PEV in programul Mathematica este descrisa mai jos:

```
Plot3D[ (1.328681897251409` Log[y] + 2.9544451227454354` ) +  $\frac{2.275720363795404` e^{x^{1/3}} \cos[1.5239637014257121` + \frac{\pi x}{100}]^2}{x}$ ,  
{x, 20, 220}, {y, 0.3, 1.2}, PlotPoints -> 40, AxesLabel -> {"latency", "Energy of stimuli", "VEP Amplit"}, ViewPoint -> {25, -45, 15},  
ColorFunction -> Hue]
```

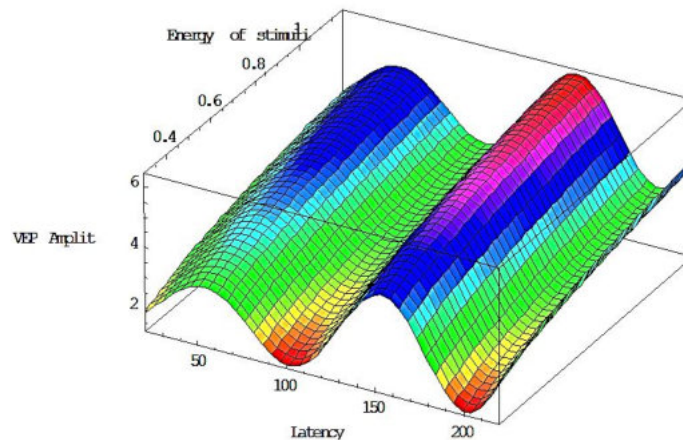


Fig 6.7 VEP 3D Hem.Sting,standard latency and Adrian's law, adapted to Flash stimulation Nicolae Stelian Stanciu [13], [19]

6.1.4. Model of the dependence of the VEP signal amplitude on the energy of Flash stimulation and Adrian E.D.'s law for the Right Hemisphere [19]

b.4. VEP Hemisphere Right to Flash Stimulation Energies:

0.3 J (Blue), 0.6 J (Green), 1.2 J (Red)

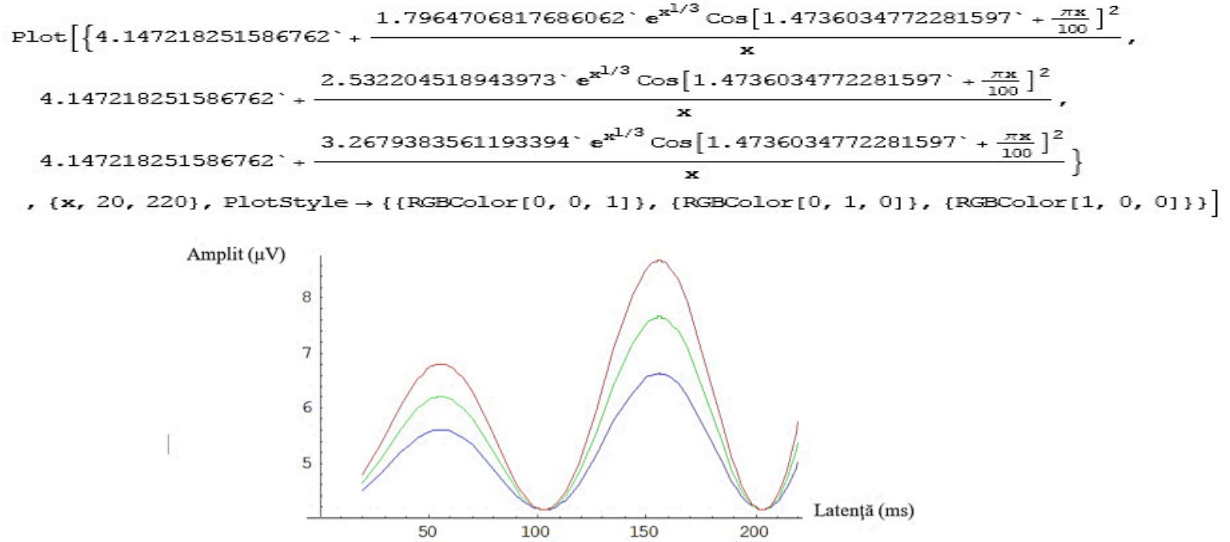


Fig 6.11 VEP Signal Hem.Right to Flash Stimulation White 0.3 J (blue),0.6 J (green),1.2 J (red),obtained with the function performed in the model of the dependence of the amplitude of the VEP components on the energy of flash stimulation Nicolae Stelian Stanciu 2021 [13], [19]

b.5. Dependence of the Right Hemisphere Visually Evoked Potential on stimulation energy and standard latency. 3D view variant

```
Plot3D[(1.0614395583071095`*Log[y]+3.0744150434060997`)+ $\frac{1.7964706817686062 \cdot e^{x^{1/3}} \cos[1.4736034772281597 + \frac{\pi x}{100}]^2}{x}$ ,
{x, 20, 220}, {y, 0.3, 1.2}, PlotPoints -> 40, AxesLabel -> {"Latency", "Energy of stimuli", "VEP Amplit"},
ColorFunction -> Hue]
```

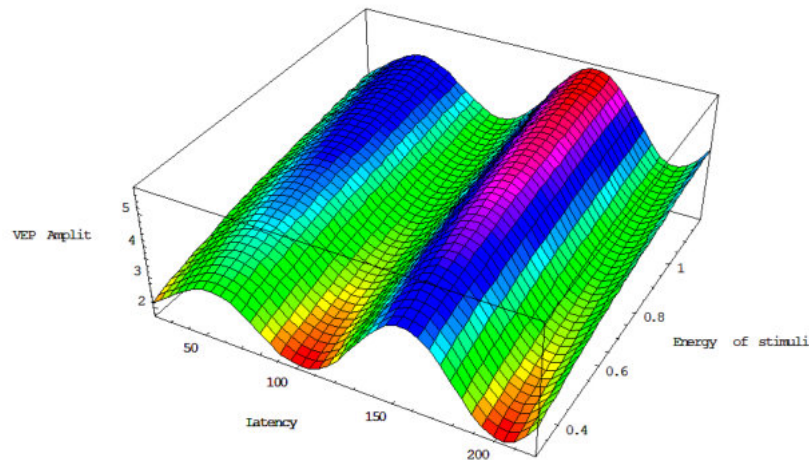


Fig 6.12 VEP 3D Hem.Straight, standard latency and Adrian's law, adapted to Flash stimulation Nicolae Stelian Stanciu [13], [19]

6.1.6. Conclusions on the VEP model in the Adrian E.D. approximation.

The VEP fit model validates the experimental data through terms that describe:

- Neural adaptation
- Neural recruitment
- The model supports Adrian E.D.'s law regarding the logarithmic increase of the amplitude of impulses with the intensity of stimulation.
- The standard latency variable is used as a parameter. The model represents the winding of the distribution of electrical potential on the CRGC–CRC and CRRC pathways, recorded at the neocortical level (zones 17, 18 bilateral occipital O1 and O2).
- The model uses average values of the amplitudes of the VEP components.
- The fit curve was achieved through multiple trials (2012), revised and improved (2020–2021).
- The structural and functional correspondence between the nerve fibers studied by Adrian E.D. (plantar, tibial) and the optic nerve is highlighted.
- The model shows the existence of a well-defined functional unit at the level of nerve fiber transmission, which links the VEP signal to the recruitment and attenuation phenomena, according to the physiological law described by Adrian Edgar Douglas

6.2. The relationship between the phenomenon of interhemispheric correlation and the amplitude of the VEP signal

6.2.1. Objectives

- Establishing an empirical relationship between the parameters of mean interhemispheric correlation (amplitude and latency) and the energy of flash stimulation.
- Correlation of these parameters with the amplitude of the VEP signal under optimal stimulation conditions (0.6 J).

6.2.2. Way of working

1. Use of experimental values of interhemispheric correlation for amplitudes.
2. Use of experimental values of interhemispheric correlation for latencies.
3. Reference to the VEP obtained at optimal stimulation (0.6 J).
4. Application of several empirical models to unite the correlation values with the VEP amplitude.
5. Analysis of optimization of the empirical relationship, calculation of extremes and establishment of relationships between parameters.
6. Conclusions regarding the connection between the linearity of amplitudes/latencies and the shape of the VEP signal.

6.2.3. Experimental Date

- Interhemispheric correlation values for amplitudes and latencies of VEP components.
- Obtaining the dependency function between these parameters and the average value Left–Right Hemisphere at White Flash 0.6 J.

Table 6.1 – Interhemispheric correlations for amplitude and latency, VEP obtained by the formula of correlations vs. experimental VEP.

| Legatura dintre formula corelatiilor de amplit si latenta si Amplit PEV | | | | | | |
|---|----------------------|----------------------|-------------|--|----------|--------------------------|
| Latent Standard ms | Corr InterHem Amplit | Corr InterHem Latent | PEV EXPERIM | PEV VAR TEOR FORM APROX | EROR % | corr PEV exp si PEV teor |
| 20 | 0.627810361 | 0.447221389 | 7.604766667 | $4.73 \cdot (B2^2 \cdot C2) / (B42^3 \cdot 35 \cdot C4)$ | -26.2166 | 0.800716523 |
| 40 | 0.501801424 | 0.701402901 | 3.097155 | 5.054555234 | 63.2035 | |
| 50 | 0.641596994 | 0.392522914 | 7.245268938 | 6.13370343 | -15.3419 | |
| 70 | 0.602637156 | 0.372138643 | 4.1521625 | 5.722756676 | 37.82594 | |
| 100 | 0.364502582 | 0.733774578 | 4.56747635 | 4.937579022 | 8.103001 | |
| 150 | 0.823690453 | 0.640416436 | 9.77477 | 9.913099589 | 1.414157 | |
| 200 | 0.826809434 | 0.286345753 | 4.63948 | 4.35041099 | -6.23063 | |
| 220 | 0.426934113 | 0.583102316 | 5.689126 | 5.004873138 | -12.0274 | |
| | | | | Medie | 6.34125 | |

Table 6.2 – Interhemispheric correlations for amplitude and latency and VEP obtained by the formula of interhemispheric correlations, case Normal.

| AVERAGE $\checkmark \times \checkmark \checkmark = 4.73 \cdot (B2^2 \cdot C2) / (B2^3 \cdot C2) + (B2^3 \cdot C2^1.5)$ | | | | | | | |
|--|-----|------------|------------|------------|----------|----------|----------|
| | A | B | C | D | E | F | G |
| 1 | | corr DR-S1 | corr DR-S1 | PEV medii | Form B | | FORM |
| 2 | 20 | 0.62781 | 0.447221 | 7.604767 | -0.1206 | 0.274576 | 5.957994 |
| 3 | 40 | 0.501801 | 0.701403 | 3.097155 | 0.401536 | -0.24756 | 4.993741 |
| 4 | 50 | 0.641597 | 0.392523 | 7.245269 | -0.32481 | 0.478785 | 6.543019 |
| 5 | 70 | 0.602637 | 0.372139 | 4.152163 | -0.19817 | 0.352147 | 6.105775 |
| 6 | 100 | 0.364503 | 0.733775 | 4.567476 | 0.493149 | -0.33917 | 4.803406 |
| 7 | 150 | 0.82369 | 0.640416 | 9.77477 | -1.61007 | 1.764042 | 10.49788 |
| 8 | 200 | 0.826809 | 0.286346 | 4.63948 | 2.278118 | -2.12414 | 3.503822 |
| 9 | 220 | 0.426934 | 0.583102 | 5.689126 | 0.312646 | -0.15867 | 5.074859 |
| 10 | | Corr Ampl | Corr Lat | PEV STG-DR | | | |

6.2.4. Comparison between experimental and VEP obtained by the formula of interhemispheric correlations

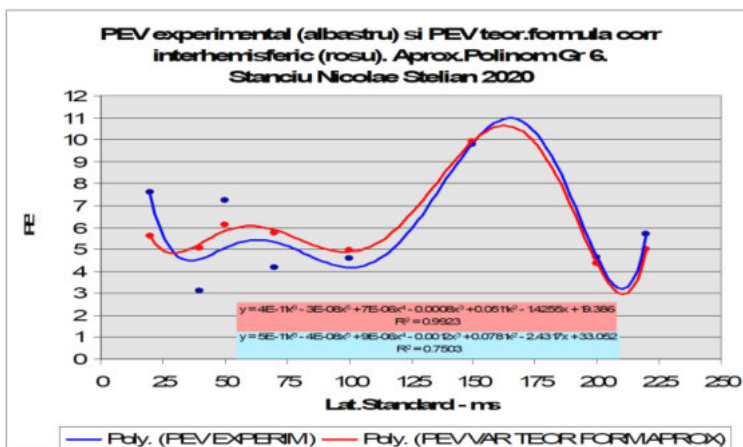


Fig 6.14. VEP obtained by the formula of interhemispheric correlations of Amplitudes and Latencies. **Note.** The similarity of the shapes associated with the graphs made by approximations of the raw experimental data of VEP and those obtained by the theoretical function of the interhemispheric correlation of VEP is observed, for the entire range of standard latency values, between 10 – 225 ms, in a polynomial representation of $grd.6$. Moreover, the numerical values associated with these representations are very close to each other for the domain associated with the CRRC secondary paths. This shows that there is a direct link between the generation of VEP in the brain structures specific to the visual analyzer and the level of interhemispheric correlation of the amplitudes and latencies of this signal.

6.2.6. VEP formula with interhemispheric correlation values

$$4.73496 - \frac{x1 \cdot x2}{2 \cdot x1^{3.34863} - x2 - x2^{1.52894}} \quad (6.2)$$

X1 = VAL. CORR INTERHEMISPHERICE AMPLITUDE

X2 = VAL. INTERHEMISPHERIC CORR LATENCY

Note It is observed that there are various domains of the correlation values of amplitudes and latencies on which the amplitude of the VEP depends.

- 1) the correlations of amplitude and interhemispheric latencies are simultaneously positive of high value, then VEP increases in amplitude.
- 2) the amplitude correlations are negative with high values and the latency correlations are positive, simultaneously, then the VEP decreases in amplitude.
- 3) the correlations of either amplitudes or latencies have a value of 0, the value of the VEP amplitude is unchanged in the value of 4.73 microvolts. This fact occurs when the dependence between the amplitudes and latencies of the signals on the two hemispheres is of the nonlinear type.

The VEP function shows that the value of the level of positive correlations on the two simultaneously conditioned hemispheres leads to an increase in the value of the VEP, and the existence of the negative interhemispheric correlation indicates that the VEP decreases in amplitude. The relationship of function also shows that the amplitude of VEP depends on the nonlinearity of the electrical activity and the phenomenon of the electrical signal conduction in the two cerebral hemispheres. [13] 2021

6.2.8 3D representation of the fit curve of the obtained VEP with the interhemispheric correlation values of amplitudes and latencies

```
Plot3D[4.73 -  $\frac{x_1 x_2}{2 x_1^{3.35} - x_2^{1.53} - x_2}$ , {x1, 0, 1}, {x2, 0, 1}, ViewPoint -> {-4.000, -1.480, 2.070},  
ColorFunction -> Hue, FaceGrids -> All, AxesLabel -> {"Corel Amp Interhem", "Corel Lat Interhem", "Ampl PEV -formula Corel"}]
```

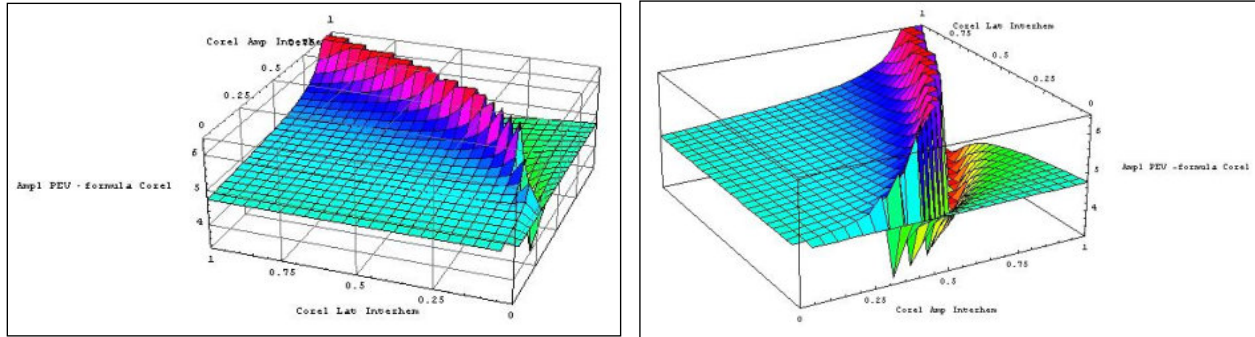


Fig 6.16 3D representation of the fit curve of VEP val.de interhemispheric correlation of amplitudes and latencies, **left-frontal view, right-side view**, –Stanciu Nicolae Stelian [13] 2021

6.2.10. Conclusions on the empirical link established between the formula of interhemispheric correlations and the formation of VEP

- If the interhemispheric correlation (amplitude and latency) is maximum → the VEP conduction process is linear, and the VEP amplitude is minimal.
- There is a threshold of the VEP amplitude associated with the interhemispheric non-correlation state, the maximum value being 4.734959490190364 μ V.
- Overlapping states with different degrees of non-correlation → increasing the VEP amplitude.
- The maximum value is obtained when the interhemispheric correlations of amplitude and latency = 1 → the processing and conduction systems behave as linear systems.

Overall conclusion

The VEP signal is closely dependent on the mode of interhemispheric correlation:

- Correlation of latencies → synchronization of the transmission of electrical signals. (the same speed of transmission of electrical signal on the conduction paths for the two hemispheres)
- Correlation of amplitudes → similarity of signal shape between the two hemispheres. (The same speed of neuronal recruitment to structures of the same type, located on the transmission pathways, in the two cerebral hemispheres)

Both conditions simultaneously met for the transmission of the electrical signal on the hemispherical conduction paths, lead to the generation of VEP without deficiencies and to the optimal processing of information in the occipital area.

6.3. VEP Reaction Amplification System

Reaction Amplifier Electrical System

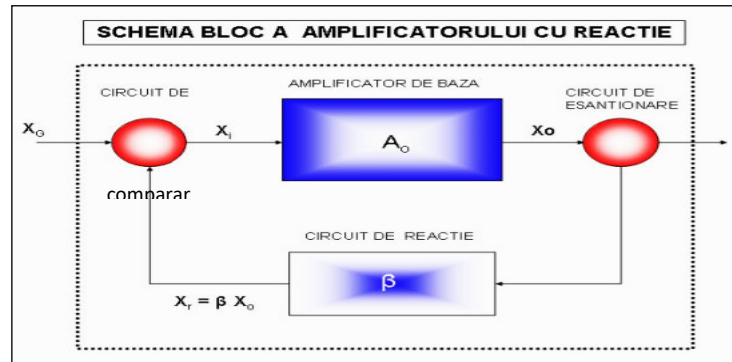


Fig. 6.21 Block diagram of the reaction amplifier – Stanciu Nicolae Stelian [36]

$$\text{REACTION COEFFICIENT } \beta \text{ From } A = \frac{A_0}{1 + \beta \cdot A_0} \Rightarrow 1 + \beta \cdot A_0 = \frac{A_0}{A} \Rightarrow \beta \cdot A_0 = \frac{A_0}{A} - 1 \Rightarrow \beta = \frac{A_0 - A}{A \cdot A_0} \quad (6.14)$$

6.3.2. Data extrapolation

In order to obtain the numerical values of the amplitude of the VEP components at the stimulation of very low energy value – mesoptic vision, the data of the VEP amplitudes obtained at the stimulation values of 0.3 J, 0.6 J, 1.2 J are extrapolated for the stimulation value of 0 J.

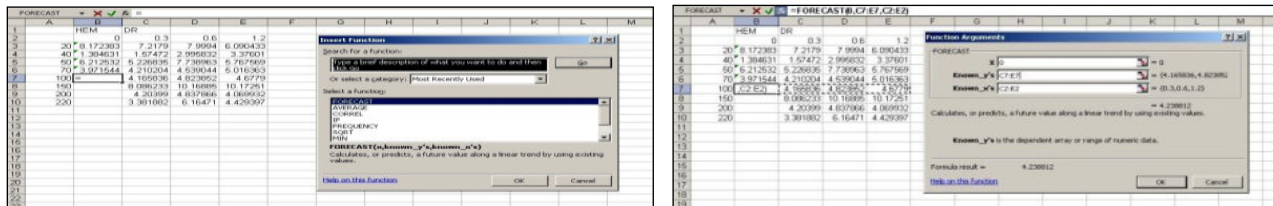


Fig 6.22 a,b The FORECAST function and steps in its use..a - the introduction of the FORCAST function, v.up and b – the result of the FORCAST function, v.down. Stanciu Nicolae Stelian 2024

6.3.4. Dependence of the Reaction Coefficient on the components of VEP, Hem, DR and Hem. STG, Standard Latency and Energy Stimulation White Flash

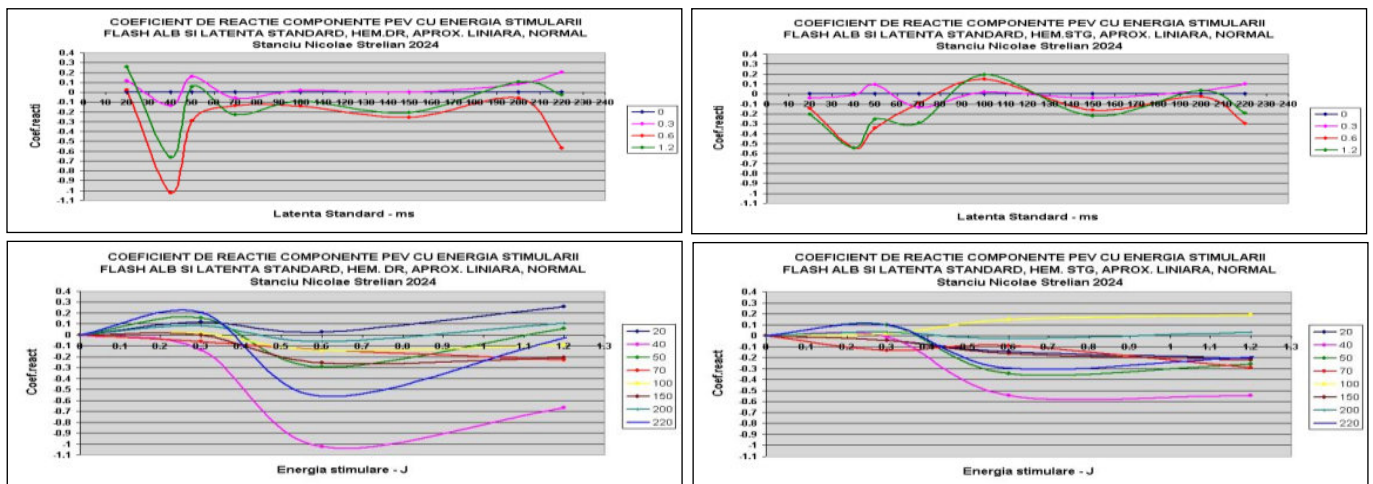


Fig 6.23 a,b . Dependence of the reaction coefficient of VEP Hem DR components on Standard Latency and Flash Energy – a - Col STG / Dependence of the reaction coefficient of VEV Hem STG components on Standard Latency and Flash Energy – b - Col.DR-SUS depending on Flash energy and LOW depending on the latency of VEP components. Aprox.linear

6.4 Visually evoked potential, effect of subcortical, cortical and neocortical activities, in Gauss approximation of the components

6.4.1. Purpose of the paper

The purpose of the paper is to highlight the fact that the VEP signal appears as a result of the superimposition of the electrical activities of the neuronal structures existing on the transmission pathways of the electrical signal coming from the retinal receptor, up to the level of the RF and the hippocampus, but also to highlight the fact that VEP is the result of a probabilistic electrical activity existing within each neuronal structure generating VEP signal components that are associated with systems with normal.. In addition, I would like to show that even if VEP is the result of a neural activity, this activity fulfills a very well-defined role in obtaining the visual signal and deleting the distortions and interferences that occur in the transport, ana; and signal processing and obtaining the accuracy of visual information.

6.4.2. Working method

The decomposition of the VEP signal is performed, in the form of sequences of Gauss-type functions, which have their maximum values at the points of the transport path that are associated with the specific standard latencies. It takes into account the law of Adrian E.D. which refers to the relationship between the amplitude of the signal and the level of stimulation, treated by the undersigned in Scientific Report No. 5

Work with the Mathematica application (approx. ord.5) and obtain the value of the VEP amplitude of type

$$1.6532 (2.7942 + 3 e^{-\frac{(-150+x)^2}{1250}} \sqrt{\frac{2}{\pi}} + 8/5 e^{-\frac{2}{625} (-70+x)^2} \sqrt{\frac{2}{\pi}} + 2 e^{-\frac{2}{225} (-20+x)^2} \sqrt{\frac{2}{\pi}}) (0.95781 + 0.12487 \text{ Log}[y])$$

where y is the flash stimulation energy, and x is the standard latency. (6.15)

6.4.3. VEP representations

(A - at 0.3 J, 0.6 J, 1.2 J, B - on generator neuronal components - function of lat. Stand., C - reprez. 3D function of the logarithm of flash stimulation).

Figure 6.25 Representation of VEP expressions at 0.3 J, 0.6 J, 1.2 J

B. Figure 6.26 The VEP components considered as Gaussian electrical voltage distributions overlapping on the latency axis forming the VEP signal, is represented below for VEP 0.6 J

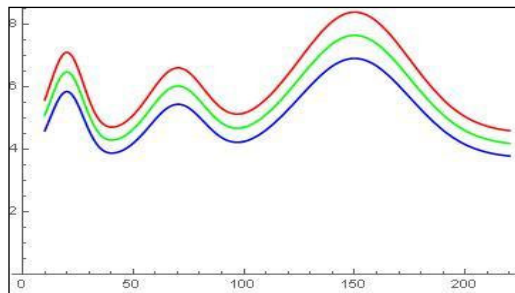


figura 6,25

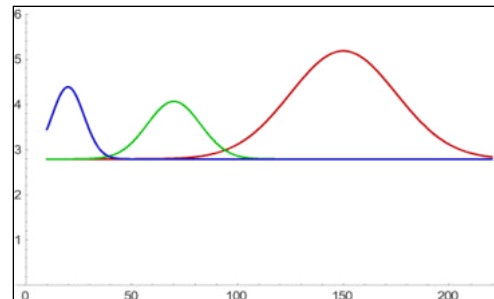


figura 6.26

C. Figure 6.27 Representation of VEP expression taking into account the logarithm of stimulation for flash stimulation values ranging from 0.3 J, - 1.2 J in Figure 3D, v.bottom, **a-side, b-top**

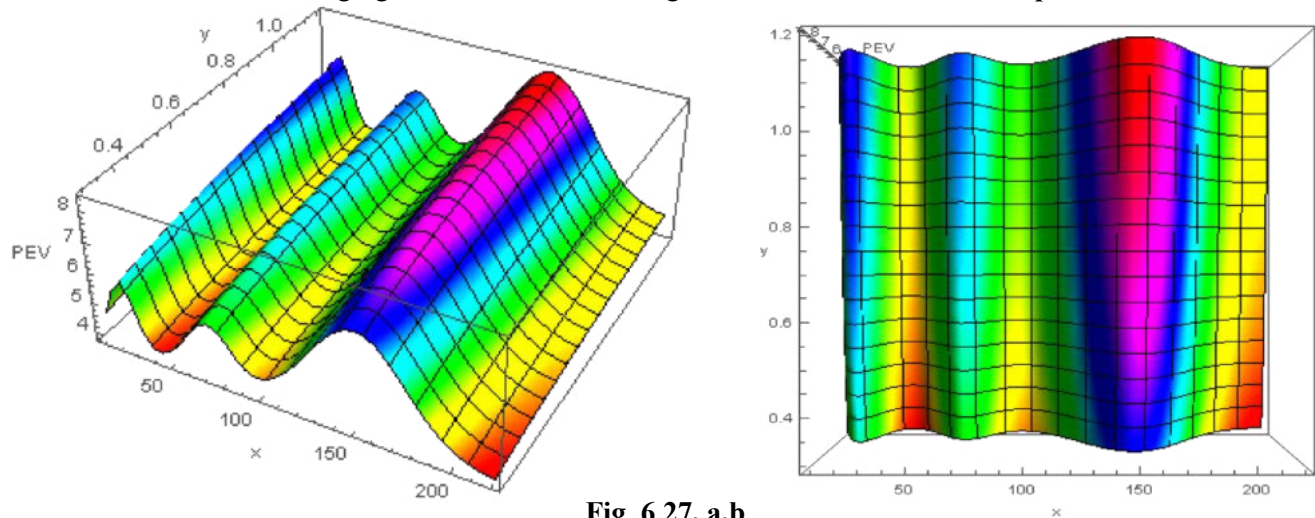


Fig 6.27, a,b

a- left, - lateral
b- right,-top

6.4.4. Obtaining the differential equation that characterizes the VEP signal and the electrical generating system of the VEP

Taking the relation VEP (6.15) as the solution of the differential equation

$$y''' + a y'' + b y' + c y = 0, \quad (6.17)$$

a system of equations is obtained that have as solution a, b, c that satisfy the given equation (approx ord 5)

$$\{ \{a \rightarrow 2.42794, b \rightarrow 0.47403, c \rightarrow 0.00263\} \}$$

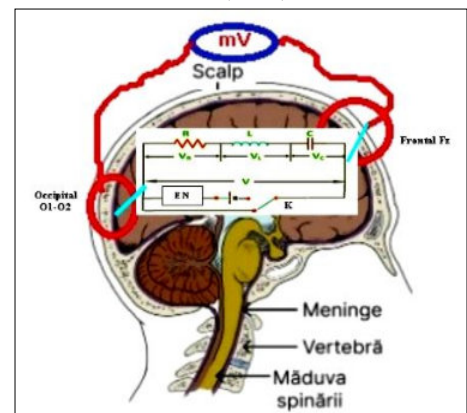
values that introduced in the relation of the expression of the VEP equation, lead to obtaining the differential equation of VEP

DIFFERENTIAL EQUATION WHICH DESCRIBES VEP – 02.03.2025 Stanciu Nicolae Stelian [12],[13]

$$= y''' + a y'' + b y' + c y = 0 \quad / . \{ a \rightarrow 2.4279437076528025^{\circ}, b \rightarrow 0.47403309857066633^{\circ}, c \rightarrow 0.00263355737594036^{\circ} \} \\ = 0.00263356 y + 0.474033 y' + 2.42794 y'' + y^{[3]} = 0 \quad (6.18)$$

Fig 6.2.7,c . Circuit Generator VEP [46],[47] modif,Stanciu N.S 2025

Analyzing the differential equation of VEP, described above, it is found that it describes complex electrical phenomena associated with a series R, L, C circuit to which a nonlinear system or component is linked - e.g. diode type, amplifier or amplifier system, oscillator system. If the nonlinear element is not taken into account, then it can be considered for a system of type R,L,C in transient regime – at discharge on capacity C, when the discharge is made periodically, the voltage measured on it, which respects the differential equation $a y'' + b y' + c = 0$ (6.19)



where $a = LC = 1/W_0^2$ where $W_0 = 1/[LC]^{0.5}$ is the pulsation (6.20)

$b = t_0$ is the capacitor discharge time. **Value results**, $L = 2.42 \text{ H}$, $R = 0.474 \Omega$,

$C = 1/c = 379.71 \text{ F}$, $\tau = RC = 179.99 \text{ s}$, $\omega_0 = 1/(LC)^{0.5} = 0.033 \text{ rad/s}$, $\nu_0 = 0.005255 \text{ Hz}$, $T_0 = 190 \text{ s} \approx \tau$, $R_c = (L/C)^{0.5} = 0.07983 \Omega$, $Q = R_c/R = 0.1684$, $\text{iar } d = 1/Q = 5.9382$.

Tipuri de elemente non linear conexe circuit R,L,C

Schmitt Trigger Oscillator

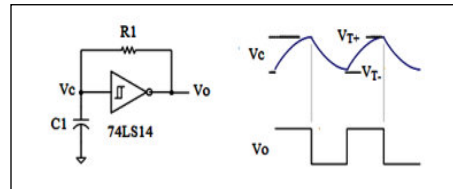


Fig. 6.28. Schmitt Trigger Oscillator Schematic and Signal

Integrator Oscillator

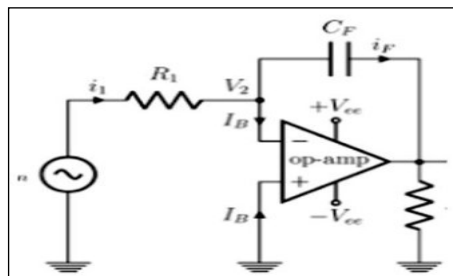


Fig. 6.29. Schema Integrator Oscillator

Chapter 7 – Program for the diagnosis of neuropsychiatric pathology

Neuropsychical Diagnose Application

Designed and manufactured by Stud. Drd. Stanciu Nicolae Stelian, 2025

7.1. Purpose of use of the program and stage of implementation

- Highlighting the cortical areas with normal or pathological functioning, in states of:
- visual rest (stand-by),
- flash stimulation,
- successive rest-flash regime.
- Analysis by frequency ranges: Δ , Θ , α , β , BHF (separately or globally, 0–62 Hz).
- The program is at an early stage, but can be developed by introducing new ones. pathology indicators.

7.2. Method of implementation

- The program is a succession of VBA algorithms, organized into macro-sequences.
- Uses predefined Excel functions (Summa, Correlation, Selection, Elimination) and complex logical functions (IF, OR, AND, EQUAL).
- Conditional mathematical operations define specific coefficients.
- The results are tabulated and represented in the form of 2D maps, estimating the intensity normal or pathological activity.

Contributions to the analysis of bioelectrical signals in neurocortical activity to improve useful information in medical diagnosis

7.3. How it works

- Probabilistic comparison of the similarity of the power correlation coefficient sign EEG ponderate.
- Analysis on 19 cortical areas and on each frequency range.
- Classification in the Normal category (10–20 cases) is made in compliance with:
 1. cantitative limit (Media, Stdev, CV, R),
 2. social criteria (responsibility, vigilance, attention, professionalism, adaptability, emotional maturity).

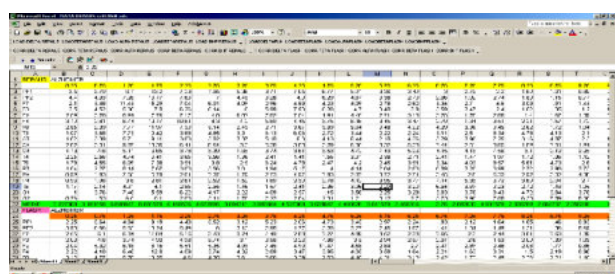
7.4. Stages of implementation of the programme

The program has 12 successive stages. Example:

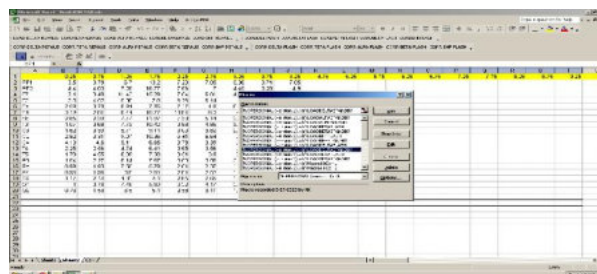
- Stage 1 – initialization,
- Stage 2 – primary processing,
- Stage 4 – selection,
- Steps 5–6 – calculation of coefficients,
- Stages 11–12 – generating the map variant A.

There are 12 stages in the implementation of the program, numbered from 1 to 12. Example below.

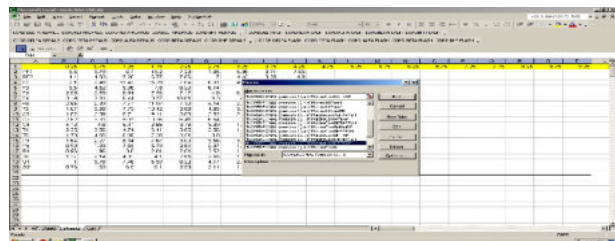
Stage nr 1



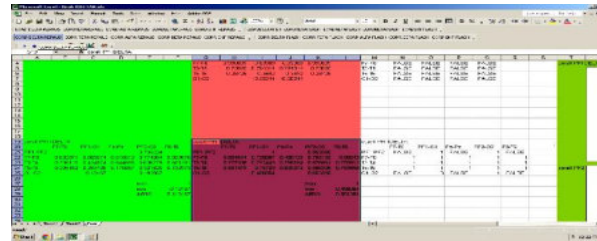
Stage nr 2



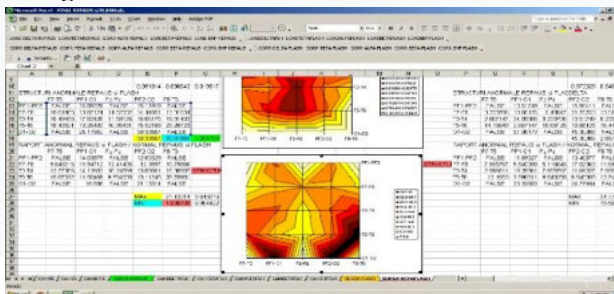
Stage nr 4



Stage nr 5-6



Stage nr 11-12



7.6. Neuropsychical Diagnose Application – Software Schema
Rest and Flash – Nicolae Stelian Stanciu 2025

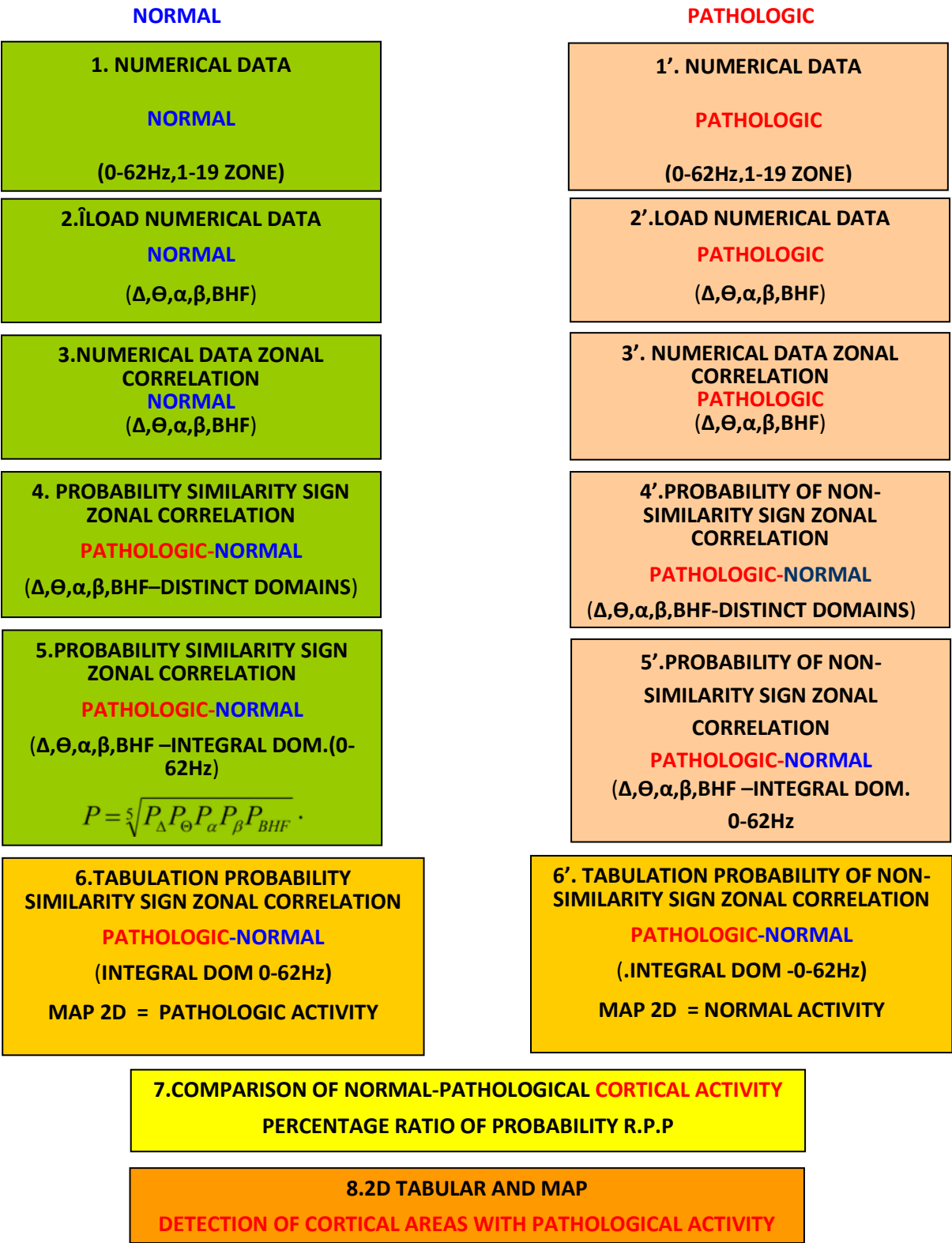


Fig. 7.3 Software Schema Neuropsychical Diagnose – Stanciu Nicolae Stelian 2024

7.7. Usefulness of the program

1. Narrow frequency ranges → greater accuracy in detecting pathological areas.
2. Analysis on Normal-Pathological brain components, either singular or on average values.
3. The possibility of expanding the database for normal and pathological cases.
4. Comparison of data between various pathologies, at rest and Flash stimulation.
5. Analysis by distinct fields or by the integral field of frequency.
6. Determination of the evolution of the pathology over time → establishing the stage of the disease.
7. The possibility of highlighting psychiatric pathologies by the R coefficient (EEG reactivity).
8. Development of the program through new modules for:
 - Clinical activities,
 - Analysis of wakefulness, sleep or dreaming, or creative activities
 - Evaluation of neocortical activity in various emotional states.

Chapter 8 – Conclusions

8.1. Results obtained. General conclusions

- The work is based on current clinical realities, especially in neuropsychiatric pathologies at the center of world medical research. They are increasingly influencing mental and social health, having a major impact on future human development.
- The doctoral thesis brings elements of scientific novelty, intended for both specialized medical professionals and electrical engineers, providing support for the development of modern clinical technologies.
- The paper describes for the first time the neuropsychiatric pathology through the prism of the electrical phenomenon associated with neuronal processes and cortical activity, going beyond the limits of the usual electroencephalographic clinical study.
- The analysis includes the area of very high frequencies (BHF), highlighting specific markers of both neurological and psychiatric pathology.
- The proposed goal is achieved through the use of new methods and techniques of clinical investigation, applied in the context of the initiated research.
- The paper develops an innovative way of detecting the foundation of pathology, using the electrical and biophysical description, complementing and supporting the current biological and biochemical descriptions of deficient neuronal activities.
- The main contribution consists in the integration of the biophysical and electrical approach in the analysis of neuropsychiatric pathologies, strengthening the approach of preserving individual and social health.

8.2. Personal and original contributions in the doctoral thesis

The work is a strictly personal one, resulting from the research activity carried out as a collaborator of the neurophysiology laboratory, aiming to support patients with neurological and neuropsychiatric pathologies. The original contributions consist in the realization and development of methods, maps and models of EEG analysis, summarized below:

Major contributions

1. Creation of EEG zonal relative power maps for Clinical, BHF and Integral frequency domains.
2. EEG weighted electrical power spectrum (0–62 Hz), depending on the cortical area, in Rest and Flash states, Normal case.
3. EEG Weighted Electrical Power Density (0–62 Hz), Sleep and Flash, Normal.
4. EEG autocorrelation functions on cortical areas, Rest and Flash, Normal.
5. Weighted EEG spectrum (0–62 Hz), Rest and Flash, S.O.C. case
6. Weighted EEG spectrum and frequency of activity (0–62 Hz), zonal mean, for cases Normal and Pathological (OCD, Bipolar, Alzheimer's, Schizophrenia, Autism).
7. Time-weighted, frequency, and cortical area weighted EEG power wave, Normal.
8. R neuronal reactivity to Flash stimulation, Normal cases.
9. Cortical reactivity to Flash stimulation, Normal case (R coefficient).
10. Average weighted power spectrum at Rest and Flash, depending on cortical areas and average frequency.
11. Weighted EEG relative variation, frequency function and cortical area, Normal.
12. Probability of increase/decrease of the relative EEG variation at Flash–Rest, Normal case, on the Delta, Theta, Alpha, Beta, BHF domains.
- 13–17. Mapping and statistical distributions of EEG relative variation at Flash–Rest, by cortical areas and frequency domains (Normal, BHF).
- 18–19. The phenomenon of decreased EEG power weighted by Flash stimulation in the Alpha Clinical domain (9.25–10.25 Hz).
- 20–23. Neuronal Reactivity to Flash Stimulation in Pathological Cases (Schizophrenia). EEG mapping and relative variations on cortical areas.
- 24–26. Neuronal reactivity to Flash stimulation in Alzheimer's cases. EEG mapping and relative variations on cortical areas.
27. Neuronal reactivity R to Flash stimulation as an indicator of normal or pathological functional states.
- 28–32. Functions and approximation curves of neuronal reactivity, EEG relative variation and standard deviation (Rest–Flash).
33. Demonstration of the relationship between the correlation coefficient and the parameters of linear regression (own contribution at the beginning of doctoral studies).

34–41 EEG zonal correlation analysis:

- characteristics,
- oscillator behavior,
- decreasing the values of the correlation with the distance from the activating area,
- Zonal correlation spectra,
- Spatial restriction to Flash,
- the phenomenon of rotation of correlation values (T3–T4 area),
- CV coefficient of EEG zonal correlation value (0–65 Hz).

The values of contributions

- The paper introduces new methods of EEG analysis (mapping, relative variations, neuronal reactivity).
- Highlights specific markers for major pathologies (OCD, Bipolar, Alzheimer's, Schizophrenia, Autism).
- Demonstrates functional relationships between correlation and regression, strengthening the mathematical foundation of EEG analysis.
- Integrates the clinical–biophysical–electrical approach, providing an innovative framework for diagnosis and research.

Phenomenology of EEG and VEP correlation

42. Decreasing the mean value of the correlation of the frontal zonal relative power as a function of the distance from the cortical activating area, case Normal.
43. Decreasing the value of the zonal EEG correlation and decreasing the standard deviation of Flash from Rest, Pathological case – Bipolar Syndrome.
44. Similar phenomenon, Pathological case – Obsessive Compulsive Syndrome (OCS).
45. Similar phenomenon, Pathological case – Schizophrenia.
46. Similar phenomenon, Pathological case – Alzheimer's disease.

EEG mapping and characteristics

47. Mapping – autocorrelation functions on cortical areas, Rest and Flash, Normal.
48. General characteristics of the EEG zonal correlation phenomenon.
49. Mapping – the correlation of weighted electrical power associated with the cortical areas, Normal, Rest and Flash, on the Clinical, BHF, Integral domains.
50. Mapping – zonal EEG correlation, pathological cases (S.O.C, Alzheimer's, Schizophrenia), Rest and Flash, on Clinical, BHF, Integral domains.
51. Mapping – weighted EEG correlation associated with cortical frequencies, Rest and Flash, Clinical, BHF, Integral, Normal domains.

52. Mapping – weighted EEG correlation associated with cortical frequencies, Pathological case – S.O.C.
53. Mapping – weighted EEG correlation associated with cortical frequencies, Pathological case – Autism.
54. Mapping – weighted EEG correlation associated with cortical frequencies, Pathological case – Alzheimer's.
55. Mapping – weighted EEG correlation associated with cortical frequencies, Pathological case – Schizophrenia.

VEP Analysis – Normal and Pathological

56. VEP correlation analysis – amplitude and latency at White Flash energies (0.3 J, 0.6 J, 1.2 J), Right and Left Hemisphere, Normal.
57. The model of the dependence of the VEP signal on the energy of Flash stimulation and Adrian E.D.'s law, for both hemispheres.
58. Correlation analysis between the amplitudes of the VEP components, mean Left-Right Hemisphere, Normal.
59. VEP amplitudes and latencies at White Flash energies (0.3 J, 0.6 J, 1.2 J), Pathological case – Neuropathy.
60. VEP amplitudes and latencies at the same energies, with the calculation of the Beta coefficient, Normal cases, Neuropathy, Epilepsy focus Left Hemisphere.
61. Reaction system for controlling VEP amplitude – Beta coefficient as pathology index.
62. Correlations of VEP amplitude to White Flash energies, Pathological case – Neuropathy.
63. Correlations of VEP latency to White Flash energies, Pathological case – Neuropathy.
64. The relationship between the interhemispheric correlation and the VEP amplitude – obtaining the relationship between Corr Amplitude and Corr Latency, at the maximum value of the VEP amplitude.

Electrical and biophysical modeling of VEP

65. VEP's Reaction Amplification System.
66. The dependence of the reaction coefficient of the VEP components (Hem. Right and Left) by standard latency and stimulation energy.
67. VEP as an effect of subcortical, cortical and neocortical activities – Gaussian approximation.
68. Obtaining the differential equation characteristic of the VEP signal and the generating electrical system.
69. Determination of the equivalent electrical assembly associated with the visual system generating VEP.

Software applications and new indicators

70. Program for the diagnosis of neuropsychiatric pathology (Neuropsychical Diagnose Application).
71. The author's introduction of new coefficients for highlighting and diagnosing neurophysiological pathologies: R, N, Beta, I.N.C.

Added value of contributions (42–71)

- Extension of EEG and VEP analysis to multiple major pathologies (Bipolar, O.S.C, Schizophrenia, Alzheimer's, Autism).
- Development of complex mappings for zonal, frequency and interhemispheric correlations.
- Introduction of the Beta coefficient as a direct indicator of pathology.
- Modeling of VEP by differential equations and equivalent electrical systems (RLC + nonlinear elements).
- Creation of a neuropsychiatric diagnostic software program, with VBA algorithms and 2D maps.
- Definition of new original coefficients (R, N, Beta, I.N.C.) for differentiated neurological-psychiatric diagnosis.

Elements of scientific novelty – structuring by chapters

Chapter 4 – Fundamental phenomena of cerebral electrical activity

1. Weighted power distribution by zonal cortical domains.
2. Weighted power distribution in relation to the Flash stimulation energy.
3. Weighted power distribution by frequency domains.
4. Weighted power distribution by time domains.
5. Weighted power distribution by functional categories (Normal vs. Pathological).
6. The oscillatory character of the wave type, depending on the frequency of activity and the cortical area.
7. The relative variation of weighted power (Reactivity) at Normal and Pathological.
8. Reactivity depending on frequency, cortical areas and temporal range.
9. Analysis of the R function – types of functions involved in approximation curves.
10. The wave of the relative variation of power weighted over time, depending on the standard angle and frequency (Clinical, Normal).
11. The wave of the standard deviation of the relative change in weighted power at rest and Flash.

Chapter 5 – EEG and VEP correlations

1. The oscillatory character of the zonal EEG correlation with the frequency of cortical activity (Normal).

2. The phenomenon of decreasing the zonal EEG correlation as a function of the distance from the activating area (Clinical, Normal).
3. Decreasing the correlation coefficient depending on the distance between the active area and the correlated area (Clinic and BHF, Normal).
4. The phenomenon of rotation of the values of the zonal EEG correlation in relation to the central area T3–T4, at Flash and Rest, with the decrease of the standard deviation (Normal).
5. Decreasing the mean value of the zonal EEG correlation with distance, both in Normal and Pathological.
6. Zonal EEG correlation – characteristics, description by cortical spatial mapping.
7. Cortical spatial mapping of the zonal EEG correlation (Normal, 0–62 Hz).
8. Weighted EEG correlation associated with cortical activity frequencies (Normal and Pathological).
9. Level of functional pathology – functional scattering coefficient.
10. Compensating effect of the correlation (Normal vs. Pathological).
11. VEP correlation analysis.
12. Correlation of amplitudes of VEP components.
13. Correlation of VEP component latencies.
14. VEP amplitude and latency, Pathological case.
15. Comparison of VEP amplitude and Beta coefficient (Normal, Neuropathy, Epilepsy, Hem. Left). Pathology indicator Beta.

Chapter 6 – Biophysical models and approximations of the VEP

- I Model of the dependence of the amplitude of the VEP signal on the energy of Flash stimulation and Adrian's Law E.D. (0.3 J, 0.6 J, 1.2 J, Left and Right Hemisphere).
- II Relationship between interhemispheric correlation and VEP signal amplitude.
- III The VEP Reaction Amplification System – the electrical system of the amplifier.
- IV VEP as an effect of subcortical, cortical and neocortical activities – Gaussian approximation of the components.

Chapter 7 – Program for Neuropsychiatric Diagnosis

- a. Development of the Neuropsychical Diagnosis Application for highlighting neuronal and neuropsychiatric pathologies.
- b. Introduction of new original coefficients for differentiated diagnosis:
- c. R – index of neuronal reactivity (depending on the frequency of activity).
- d. N – pathology scattering index.
- e. BETA – reaction coefficient in the amplification system with reaction of neuronal sensitivity to Flash stimulation.
- f. I.N.C. – indice de necompensare a valorilor de corelație.

The value of scientific novelty

1. The thesis introduces new fundamental phenomena in the analysis of cerebral electrical activity.
2. It extends the study of EEG and VEP through spatial mapping, interhemispheric correlations and biophysical models.
3. It proposes original indicators (R, N, Beta, I.N.C.) for differentiated neurological-psychiatric diagnosis.
4. It integrates the electrical and biophysical approach with clinical analysis, providing an innovative framework for research and practical engineering application.

8.3. Personal and original contributions through scientific publication activity

The author of this doctoral thesis has a continuous scientific activity, materialized in works published over the years, which reflect the evolution of research and original contributions in the field of neurophysiology and neuronal biophysics.

List of published works

1. EEG signal correlation analysis in Normal or Pathological cases, Vol. 1 – Estfalia Publishing House, 2020 (forthcoming, 2025).
2. An electrical engineering perspective on neuromodulation. Characteristic of the magnetic stimulation procedure – U.P.B. Sci Bull, Serie C, Vol. 86, Iss. 4, 2024.
3. Correlation of electric EEG and VEP Signals in Normal Neuro-Physiological Brain Activity – Springer Nature, IFMBE Proceedings, Meditech 2000, 2022.
4. Correlation Analysis of the EEG Signals in Normal and Pathological Cases – IEEE, 12th International Symposium on Advanced Topics in Electrical Engineering (ATEE), 2021.
5. Features of the Electrical Cortical Signal in Steady State and in White Flash Stimulation – IEEE, 11th International Symposium on Advanced Topics in Electrical Engineering (ATEE), 2019.
6. Biophysics of Neuronal Excitation – Estfalia Publishing House, 2017–2018.
7. Biophysical aspects of the propagation of nerve influx in the visual system – Estfalia Publishing House, 2013–2015.
8. Biophysical features of the VEP – European Biophysics Journal, EBSA & British Biophysical Society Congress, London, 2007, Springer.
9. Biophysical features of the VEP – UMF Carol Davila, National Conference of Biophysics, București, 2007.
10. Coherence between neocortical areas electrical activity recorded in Monopolar and Source derivation – Romanian Journal of Neurology, Romanian Academy, 2006.

8.4. Prospects for further development

The current work constitutes a foundation for future developments in the field of description and highlighting of neuropsychiatric pathologies. It is not exhaustive, but opens up research directions that can be deepened and extended by new methods and techniques.

Future development directions

1. Extension of the database for Normal and Pathological cases.
2. Analysis and refinement of pathology markers, in order to increase the accuracy in detecting and staging the disease.
3. Development of mathematical and biophysical methods for the analysis of electrical phenomena, in order to understand in detail the neuropsychiatric pathological processes.
4. Elaboration of electrical assemblies according to theoretical models, capable of evading pathology by invasive or non-invasive methods.
5. Development of the Neuropsychical Diagnosis program for the analysis of other cortical states:
 - Normal: emotional states, dream states;
 - Pathological: confusional states, derealization, mania, schizophrenic delirium.
6. Integration of the study of VEP amplitudes and latencies and their correlations in the diagnostic program.
7. Incorporation of the original coefficients (R, N, BETA, I.N.C.) in the new form of the Neuropsychical Diagnosis program, in order to increase the resolution and effectiveness of the diagnosis of the affected cortical areas.

The value of these insights

- They ensure the continuity of research and open up new possibilities for differentiated diagnosis.
- Integrates the clinical-biophysical-electrical approach with software applications and experimental models.
- Contributes to the development of innovative tools for the detection and treatment of neuropsychiatric pathologies.

BIBLIOGRAFIE

- [1] - *Aurel Popescu* - Fundamentele Biofizicii Medicale / Ed.All / 1994.
[2] – *Mihaela Morega* – Bioelectromagnetism curs UPB / 2018 Editura Matrix Rom 1999 (actualizat în note de curs, UPB 2000 - 2025)
[3] - *Jakko Malmivuo, Robert Plonsey* – Bioeletromagnetism / January 1995/Oxford University Press / New York

- [4] – *Adrian Edgar Douglas* / The impulses produced by sensory nerve endings. Impulses from pain Receptors – From physiological laboratory Cambridge – Journal of Physiology / Volume 62 / Issue 1 / Pages i– iv, 1 – 128 /October 30, 1926.
- [5] - *Vasilescu V, Mărgineanu D.G* - Introducere in neurobiofizica / Ed. Stiintifica si Enciclopedica / 1979.
- [6] - *Brown A.G* - Nerve Cells and Nervous Systems / Ed Springer Verlag London Limited / 1991.
- [7] - *Josef Dudel* - Neurobiophysics - Excitation, its Conduction and Sinaptic Transmission / Ed. Springer / 1987
- [8] - *Edmond Nicolau, Constantin Balaceanu Stolnici* - Elements of Neurocibernetics / Ed Stiintifica / 1967
- [9] – *Adrian E.D* - Experiments on the nervous system / British Medical / 1950 / Experiments in the nervous system. (The Stephen Paget memorial lecture delivered at University College London, 22 November 1950.) Conquesty Res. Def. Soc. 39, pp. 2-14 (preluat din Biographical Memoirs of Fellows of the Royal Society, Vol. 25, 1979, online:
- [10] - *Internet* - [www. google.com](http://www.google.com) & [altavista .com](http://www.altavista.com) / Electroencephalography./ <http://buttler.cc.tut.fi/~malmivuo/bem/bembook> - Sharbrough F., Chatrian G.E., Lesser R.P., Lüder H, Nuwer M., Picton T.W.- American Encephalography Society Guidelines for Standard Electrode Position Nomenclature / J. Clinic Neurophysiology nr.8 / 2002.
- [11] - *Dumitru Constantin, Ignat Dana Craiu, Carneb Adela Sirbu, Cristian Dinu Popescu, Tudor Lupescu, Niculina Butoianu* – Electroencefalografia clasica si moderna – Editura Medicala / 2008
- [12] - *Stanciu Nicolae Stelian* - Analiza de corelație de semnal EEG în cazuri normale și patologice val 1 – Ed Estfalia. / 2020 sub tipar în 2025. Până în prezent, 2025 carea nu este editată integral și nici publicată.
- [13] - *Stanciu Nicolae Stelian* / Analiza de corelatie de semnal PEV in cazuri normale si patologice / Vol 2 / Editura Esfalia .Bucuresti / 2021. sub tipar în 2025
- [13*] - *Dan Mihai Psatta, Mircea Olaru, Mihaela Matei* – Visual inflow traveling in the brain highlighted by photic evoked potentials mapping a derivated model of cognitive processing ./ Rom.J.Neurol.Nr 40./ 2002.
- [14] - *Dan Mihai Psatta , Mihaela Matei* - Mapping of VEPs and ERG responses to punctual stimulations in the visual field. / Rev.Roum.Neurol. Psychiat. NR.30 / Ed. Academiei Romane./ 1992.
- [15] - *Paul Borza, Ioan Matlac, Mihail Micu* – Instrumente Biomedicale / Editura Tehnică, 1996
- [16] - *Steven W.Smith* - Digital Signal Processing - California Technical Publishing - San Diego California / 1999
- [17] - *Ing Joarg Hansmann, Andreas G* / nod EEGdigital.vi,θ / Modular EEG / [http // openeeg.net](http://openeeg.net) / 01.05.2003
- [18] - *Stanciu Nicolae Stelian* - Aspecte Biofizice ale propagarii influxului nervos in sistemul visual – Ed Estfalia. / 2014
- [19] - *Stanciu Nicolae Stelian* - Referat Stiintific nr 5 Legea lui Adrian E.D. și legătura ei cu PEV. S.D.I.E / 2021
- [20] - *Stanciu Nicolae Stelian* - Referat Stiintific nr 2 Sistemul și tehnica de măsurare a PEV și EEG , S.D.I.E / 2020
- [21] - *Stanciu Nicolae Stelian* - Referat Stiintific nr 1 Elemente caracteristice producerii, morfologiei și înregistrării semnalului Potențial Evocat Vizual S.D.I.E / 2019
- [22] - *Stanciu Nicolae Stelian* - Referat Stiintific nr 4. Analiza de corelație de semnal PEV în cazuri normale și patologice. S.D.I.E / 2020
- [23] - *Stanciu Nicolae Stelian* - Referat Stiintific nr 3 Analiza de corelație de semnal EEG în cazuri normale și patologice. S.D.I.E / 2020
- [24] - *Nicolae Stanciu, Mihaela Matei, Dan M.Psatta* – Coherence Between Neocortical Areas Electrical Activity Recorded in Monopolar and Source derivation – Editura Academiei Romane / Vol.44.Nos.1-2 / 2006
- [25] - *Keith H.Chiappa* - M.D-Assistant Professor of Neurology, Harvard Medical School, Director EEG and Evoked Potential Unit, Clinical Neurophysiology Laboratory, Massachusetts General Hospital Boston,

Massachusetts, U.S.A – *Evoked Potentials in Clinical Medicine, Third Edition* – Lippincott – Raven Publishers Philadelphia, New York / 1997

[26] – *Constantin Antonescu, Tudorel Andrei, Liviu Stelian Begu* – Bazele teoretice ale Statisticii – Ed. Fundatia “Romania de Maine” / 2000

[27] – *Markus Werkle – Bergner, Yee Lee Shing, Viktor Muller, Sho-Chen, Ulman Lindenberger* (Germany) - EEG gamma band synchronization in visual coding from childhood to old age; Evidence from evoked power and inter-trial phase locking – *Clinical Neurophysiology* / Elsevier / 29 May 2009

[28] – *B. Demidovich* – Problems in mathematical analysis / MIR Publishers / Moscow / 1976

[29] – *Mihoc Ghe, Urseanu V.* – Matematici aplicate in statistica / Colectia Teoria Probabilitatilor / Ed. Academiei Romane / 1962.

[30] – *Jan Raethjen, R.B. Govindan, M. Muthuraman, Florian Kopper, Jens Volkmann, Gunther Deuschl* – Cortical correlates of the basic and first harmonic frequency of Parkinsonian tremor - *Clinical Neurophysiology* / Elsevier / 12 September 2009.

[31] – *Andrei Novac* – Statistica Socială aplicată și Memorator Statistic / Ed. Hiperion XXI / 1995

[32] – *O. Onicescu, Gh. Mihoc* – Lectii de Statistica Matematica – Ed. Tehnica / 1958

[33] – *Dan Mihai Psatta, Mihaela Matei* – Neural Generators of Visual Evoked Potentials Components / Rom. J. Neurol. Nr 35 / 1997

[34] – *Dan Mihai Psatta, Mircea Olaru, Mihaela Matei* – Visual inflow traveling in the brain highlighted by photic evoked potentials mapping a derived model of cognitive processing. / Rom. J. Neurol. Nr 40. / 2002.

[35] – *Paul Mc Fedries* – VBA pentru începători / Teora QUE / 2007

[36] – *Stanciu Nicolae Stelian* - Correlation Analysis of the EEG Signals in Normal and Pathological Cases- 2021 IEEE – 2021 12th International Symposium on Advanced Topics in Electrical Engineering (ATEE) / 2021 / Proceedings Paper – the 12th International Symposium of Advanced Topics in Electrical Engineering ATEE 2021, Bucharest, Romania, March 2021, published by IEEE, ISBN: 978-1-6654-1878-2, ISSN: 1843-8571

[37] – *Stanculescu N, Bobulescu N* – Circuite și Dispozitive electronice / Curs Facultatea de Fizică, București, Măgurele / 1991

[38] – *B.P. Lathi* – Modern Digital and Analog Communication Systems – New York – Oxford, OXFORD UNIVERSITY PRESS / 1998

[39] – *Landau and Lifchitz* - THEORIE DES CHAMPS – PHYSIQUE THEORIQUE – EDITION MIR / MOSCOU 1970

[40] – *Frank S Crawford Jr.* – WAVES – Berkeley Course of Physics – Volume III - University of Berkeley USA / UNDE – Cursul de Fizică Berkeley – Volum III – Ed. Didactică și Pedagogică – București / 1983

[41] – *David M. Cook* – THE THEORY OF THE ELECTROMAGNETIC FIELD – Lawrence University, Appleton, Wisconsin – PRINTICE - HALL INC. - Englewood Cliffs, New Jersey, USA / 1975

[42] – *Stanciu Nicolae Stelian* - Biofizica Excitației Neuronale – Ed. Estfalia. / 2017 / 2018

[43] – *Stanciu Nicolae Stelian* - Correlation of electric EEG and VEP Signals in Normal Neuro - Physiological Brain Activity - Ed, Springer – Nature IFMBE Proceedings. 2022 - . Meditech / 2020 / . în volumul: Vlad S., Roman N.M. (eds) 7th International Conference on Advancements of Medicine and Health Care through Technology - MEDITECH 2020, IFMBE Proceedings, vol 88, pp. 18-26, Springer Nature Switzerland AG 2022, Print ISBN 978-3-030-93563-4, online ISBN 978-3-030-93564-1

[44] – *Stanciu Nicolae Stelian* - Correlation Analysis of the EEG Signals in Normal and Pathological Cases- 2021 - Ed. IEEE – 2021 12th International Symposium on Advanced Topics in Electrical Engineering (ATEE) / 2021

[45] – *Stanciu Nicolae Stelian* - Features of the Electrical Cortical Signal in Steady State and in White Flash Stimulation.- 2019 – Ed. IEEE – 2019 11th International Symposium on Advanced Topics in Electrical Engineering (ATEE) / 2019 published by IEEE, ISBN: 978-1-4799-7514-3, ISSN: 1843-8571

[46] – Circuit Globe.com / RLC Series Circuits

[47] – <https://platform.ginamed.ro/cursuri/biologie-barrons/organizarea-sistemului-nervos> / Fig. 11.2