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Alpha, delta and theta rhythms in a neural net model. Comparison with MEG data

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HIGHLIGHTS

- The epileptic patients had high MEG amplitudes whereas the normal subjects had low.
- The epileptic MEG had Poisson distribution and absence of α-rhythm.
- The normal MEG had Gauss distribution and presence of α-rhythm.
- TMS to epileptic patients changed the distribution of MEG from Poisson to Gauss.
- The findings from the theoretical neural model were comparable to the MEG data.

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ABSTRACT

The aim of this study is to provide information regarding the comparison of a neural model to MEG measurements. Our study population consisted of 10 epileptic patients and 10 normal subjects. The epileptic patients had high MEG amplitudes characterized with θ (4–7 Hz) or δ (2–3 Hz) rhythms and absence of α-rhythm (8–13 Hz). The statistical analysis of such activities corresponded to Poisson distribution. Conversely, the MEG from normal subjects had low amplitudes, higher frequencies and presence of α-rhythm (8–13 Hz). Such activities were not synchronized and their distributions were Gauss. These findings were in agreement with our theoretical neural model. The comparison of the neural network with MEG data provides information about the status of brain function in epileptic and normal states.

1. Introduction

1.1. *The Neural Net Model*

The neural nets (Anninos et al., 1970; Anninos and Kokkinidis, 1984) are supposed to be created of discrete sets of randomly interconnected neurons of related organization and function. The neural connections are set up by means of chemical markers carried by the individual cells, in accordance to the hypothesis of neural specificity (Sperry 1943, 1963; Prestige and Willshaw, 1975). These are established only if both pre and postsynaptic neurons have the same marker. Consequently, the neural population of the netlet is treated as a set of subpopulations of neurons, each of them described by a particular chemical marker. McCulloch and Pitts (1943) suggested that the neuron is a bistable element and can be either in a resting or in an active state. The transition from the resting to the active state happened when the sum of postsynaptic potentials (PSPs) arriving at the cell surpasses a definite critical value, the threshold (θ) of the neuron. PSPs might be excitatory (EPSPs) or inhibitory (IPSPs) shifting the membrane potential closer to or further away from the threshold.

In this model, a neural net with N markers (neurotrasmitters) is presumed to be assembled of A neurons. A part h ($0<h<1$) of them are inhibitory neurons while the rest are excitatory. Each neuron receives, on average, μ^+ EPSPs and μ^- IPSPs. K^+ (K^-) is the size of the PSP produced by an excitatory (inhibitory) component. The neurons are also characterized by the absolute refractory period (r) and the synaptic delay (τ) ($\tau<r<2\tau$). If a number of neurons fire concurrently at time t, then all neural activity resulting from this primary activity will be restricted to times $t+\tau$, $t+2\tau$,…

If a neuron fires at time t, it produces the appropriate PSP after a synaptic delay τ. PSPs arriving at a neuron are summed immediately, and if this sum is greater or equal to θ, then the neuron will fire at once, or else it will be inactive. PSPs (if below θ) will continue with or without decrement for a period called the summation time, which is assumed to be less than the synaptic delay.

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Firing is momentary and causes the neuron to be insensible to further stimulation for the time of a refractory period (Fournou et al., 1991, 1993, 1995, 1997, 2005; Kotini and Anninos, 1997; Anninos et al., 1984; Anninos and Kokkinidis, 1984).

A. *Poisson distribution*

The expectation value of the neural activity α_{n+1} (the fractional number of active neurons in the net at time $t=(n+1)\,\tau$) in an isolated net is given by:

$$<\alpha_{n+1}> = (1-\alpha_n)\, exp\,(-\alpha_n h\,\mu^-)\sum_{m=0}^{m_{max}}(\alpha_n\, h\mu^-)^{\mathrm{m}}/m!$$
$$\left\{1-exp\,(-\alpha_n\,(1-h)\,\mu^+)\sum_{l=0}^{\eta'-1}(-\alpha_{\mathrm{n}}\,(1-h)\,\mu^+)^{\mathrm{l}}/l!\right\}$$

where

$$\eta' = u((\theta - mK^-)/K^+)$$
$$m_{\max} = A\,\alpha_n h\,\mu^- \qquad (1)$$
$$l_{\max} = A\,\alpha_n(1-h)\,\mu^+$$

If the neural net has N chemical markers m_j $(j=1,\ldots N)$ the expectation value of the neural activity α_{n+1} at time $t=(n+1)\,\tau$ is given by:

$$<a_{n+1}> = (1-\alpha_n)\left[\sum_{j=1}^{N-1} m_j \sum_{I=0}^{I_{\max}}(1-\sum_{L=0}^{L=\eta_j-1} P_L)Q_I\right.$$
$$\left.+(1-\sum_{j=1}^{N-1} m_j)\sum_{I'=0}^{I'_{\max}}(1-\sum_{L'=0}^{L'=\eta_j-1} P_{L'})Q_{I'}\right]$$

where

$$\eta_{\mathrm{j}} = u[(\theta_j + IK_j^- \pm MK_{\mathrm{o}}^+)/K_j^+]$$
$$I_{\max} = A\,\alpha_n\,\mu_j^-\, h_j\, m_j \qquad (2)$$
$$I'_{\max} = A\,\alpha_n\mu_j^-\, h_j(1-m_j)$$

and

$$P_{\mathrm{L}} = exp(-\alpha_{\mathrm{n}}\,(1-h_j)\mu_j^+ m_j)\,(\alpha_{\mathrm{n}}(1-h_j)\mu_{\mathrm{j}}^+ m_j)^{\mathrm{L}}/\,\mathrm{L}!$$
$$Q_{\mathrm{I}} = exp(-\alpha_n h_j\mu_j^-\, m_j)\,(\alpha_n h_j\mu_j^-\, m_j)^{\mathrm{I}}/\,\mathrm{I}!$$
$$P_{\mathrm{L}'} = exp(-\alpha_n(1-h_j)\mu_j^+\,(1-m_j))\,(\alpha_n(1-h_j)\mu_{\mathrm{j}}^+\,(1-m_j))^{L'}/\,\mathrm{L}'!$$
$$Q_{\mathrm{I}'} = exp(-\alpha_n h_j\mu_{\mathrm{j}}^-\,(1-m_j))\,(\alpha_n h_j\mu_{\mathrm{j}}^-\,(1-m_j))^{\mathrm{I}'}/\,\mathrm{I}'! \qquad (3)$$

B. *Gauss distribution*

The expectation value of the neural activity $\alpha_{\mathrm{n}+1}$ at time $t=(n+1)\,\tau$ in an isolated net is given by:

$$\alpha_{n+1} = (1-\alpha_n)\frac{1}{\sqrt{2\pi}}\int_{X_{n+1}}^{\infty} \exp\left(-\frac{X^2}{2}\right)\mathrm{dx}$$

where:

$$\mathrm{X}_{n+1} = (\theta - \overline{\mathrm{e}_{n+1}})/\delta_{n+1}$$
$$\overline{\mathrm{e}_{n+1}} = \alpha_{\mathrm{n}}\left[\,\mu^+(1-\mathrm{h})\,\mathrm{K}^+ + \mu^-\mathrm{h}\,\mathrm{K}^-\right]$$
$$\delta_{n+1}^2 = \alpha_{\mathrm{n}}\left[\,\mu^+(1-\mathrm{h})\,(\mathrm{K}^+)^2 + \mu^-\mathrm{h}\,(\mathrm{K}^-)^2\right] \qquad (4)$$

If the neural net has N chemical markers m_j $(j=1,\ldots N)$ the prospect value of the neural activity α_{n+1} at time $t=(n+1)\,\tau$ is given by:

$\alpha_{n+1}=(1\text{-}\alpha_{\mathrm{n}})\,\frac{1}{\sqrt{2\pi}}\int_{x_{j,n+1}}^{\infty} e^{-\frac{x^2}{2}}dx$ where:

$$x_{j,n+1} = \frac{\theta_j - \overline{e}_{j,n+1}}{\delta_{j,n+1}}$$
$$\overline{e}_{j,n+1} = a_n m_j[\mu_j^+(1-h_j)K^+ + \mu_j^-\, h_j K^-]$$
$$\delta^2{}_{j,n+1} = a_n m_j[\mu_j^+(1-h_j)(K^+)^2 + \mu_j^-\, h_j(K^-)^2] \qquad (5)$$

If a neural net of neurons is activated, it will either returned to an inactive state or settle into a periodic form of activity, which is referred to as "cyclic" mode. We examined the circumstances and reasons under which such cyclic modes come out in simulated neural nets. A quantitative evaluation of the status of the net is provided for this purpose by a matrix, which specifies the particular set of neurons active at any given instant of time, and describes the instantaneous state of the net. This is called the "net state vector". This matrix can be defined as:

$$\{\alpha_n\} = ({}^1\alpha_n\,{}^2\alpha_n : {}^A\alpha_n) \qquad (6)$$

Where A is the total number of neurons in the net and ${}^i\alpha_n$ equals 1 or 0, according to whether or not the ith neuron fires at time $t=n\tau$.

2. Methods

We compared the theoretical results with experimental findings obtained using MEG data from 10 epileptic patients and 10 healthy volunteers. Informed consent for the methodology and the aim of the study was obtained from all participants prior to the procedure. The research program was approved by the Research Committee of the Democritus University of Thrace.

Magnetoencephalography (MEG) measurements were performed using a second order gradiometer SQUID (Superconducting Quantum Interference Device) (Model 601, Biomagnetic Technologies Inc.), located in a magnetically shielded room with low magnetic noise. The MEG recordings were performed after positioning the SQUID sensor 3 mm above the scalp of each subject using a reference system based on the International 10–20 Electrode Placement System. This system uses any one of the standard EEG recording positions as its origin (we used the P3, P4, T3, T4, F3, and F4 recording positions). Around the origin (T3 or T4 for temporal lobes) a rectangular 32-point matrix was used (4 rowsx8 columns, equidistantly spaced in a 4.5 cmx10.5 cm rectangle) for positioning of the SQUID. The MEG was recorded from each temporal lobe at each of the 32 matrix points of the scalp for 32 s and was band-pass filtered with cut-off frequencies of 0.1 and 60 Hz. The MEG recordings were digitized using a 12 bit precision analog-to-digital converter with a sampling frequency of 256 Hz, and stored in a PC peripheral memory for off-line Fourier statistical analysis. The method, with temporal and spatial averaging, eliminates short-term abnormal artifacts in any cortical area, while it retains long-lasting localized activation phenomena (Anninos et al., 1997, 1999, 2000, 2003). External transcranial magnetic stimulation (TMS) in the order of pico Tesla was applied in the frontal, occipital and temporal lobes using an electronic device and the emitted magnetic activity was recorded again (Anninos and Tsagas, 1995; Anninos et al., 1991, 2000, 2003, 2007, 2008). The coils of this device were placed on the patient's scalp and weak magnetic fields, were applied for total 6 min. The electronic device consists of a low voltage generator, which can produce low frequencies from 2–13 Hz to a group of coils of 1 cm diameter. The 32 coils are enclosed between two parallel plane surfaces in such a way that the axis of the coils is situated perpendicular to these surfaces and on the 32-point matrix. The applied TMS carried similar field characteristics with the ones emitted from the patient's brain prior to the application of TMS (intensity: 1–7.5pT; frequency: 8–13 Hz).

We used the x^2 – fitting method to analyze the MEG data (Kotini et al., 2005).