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## CONTENTS

15. High Damage Threshold AR-Coating for Nd-YAG Lasers-I: Conventional Systems  
S.M.Javed Akhtar, D.Ristau, J.Ebert and H. Welling .....169
16. High Damage Threshold AC-Coating for Nd:YAG Lasers-II: Inhomogeneous Systems  
S.M.Javed Akhtar, F.Mittendorf, H.Schink, D.Ristau, J.Ebert and H.Welling .....191
17. Parametric Study of RF Capacitative Discharges for the Excitation of CO<sub>2</sub> Gas Lasers  
S.M.A.Durrani, S.M.Nasir, S.M.Raza, M.A.Farooqui, P.Vidaud and D.R.Hall .....202
- D. PLASMA PHYSICS**
18. Some Aspects of Thermonuclear Fusion  
G.Murtaza, F.Nadeem Beg and M.Zakaullah .....217
19. Benjamin-Bona-Mahoney Solitons in a Piezoelectric Semiconductor Plasma  
H.A.Shah, N.Ikram and T. Abdullah .....231
- E. GENERAL TOPICS**
20. Frontiers of Cosmology  
Asghar Qadir .....246
21. Neurophysics: A Beginner's View Point  
K.A.Siddiqui, S.A.Kamal and N.U.Khan ..... 285

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## NEUROPHYSICS: A BEGINNER'S VIEW POINT

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### ABSTRACT

*Brain is a complex system and the brain theory is developing into a central focus among many disciplines. The functioning of the brain has long been prominent among topics of fundamental interests to biologists and physicists alike. After an introduction of brain and the physics involved, a space time representation of the global electrocortical activity has been given. The idea of analyzing EEG signals by mathematical modelling is a topic among scientists and a model based on quantum oscillators is under discussion in our group. A brief outline of the model is given.*

### 1. INTRODUCTION

The working of the brain presents one of the most challenging problems of neurosciences. Quantitative data about the nervous system are much more sparse than is

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desirable and it seems a major difficulty for attempts to build mathematical theories of brain functions. It is an extremely complex structure containing something of the order of  $10^{10}$ - $10^{12}$  nerve cells with perhaps  $10^{15}$  interconnections [1]. Essential to the development of principles capable of relating brain cell activities of psychological processes, will be a more profound understanding of the bio-electrical and neuronal interaction phenomena.

It is beyond our capabilities to attempt any survey of neuroanatomy and we shall refer to a standard text [2]. A knowledge of the stuff of the brain will be fascinating to many participants, hence an introduction is given at the pedestrian level.

### 1.1 Brain

An examination of the human brain will show it to be a large, bilaterally symmetrical organ (Figure 1). The outer surface, or cortex contains billions of nerve cells (neurons) and the processes that connects one neuron to

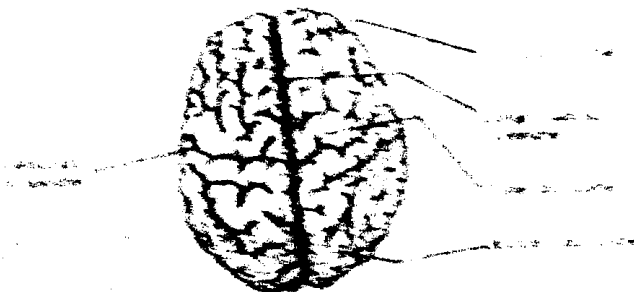


Fig.1(a) The human brain seen from above.

another. Towards the rear of the brain the cerebellum can be seen under the cortex. Underlying the layer of cortical neurons are the tracts of the fibrous axons that extend from and carry messages from cortical neurons to other neurons.

An insulating sheath, termed the myelin sheath, serves to insulate one axon from its immediate surroundings and gives it a whitish appearance. Each hemisphere of the cortex can be divided into four lobes. The frontal, Parietal, temporal and the occipital lobes. These lobes contain two types of zones, The zones that are either sensory or motor and the zones that are associated.

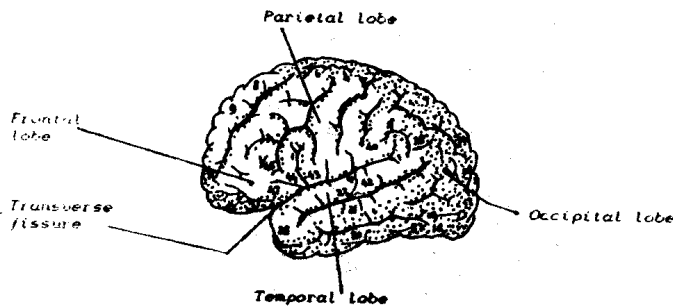
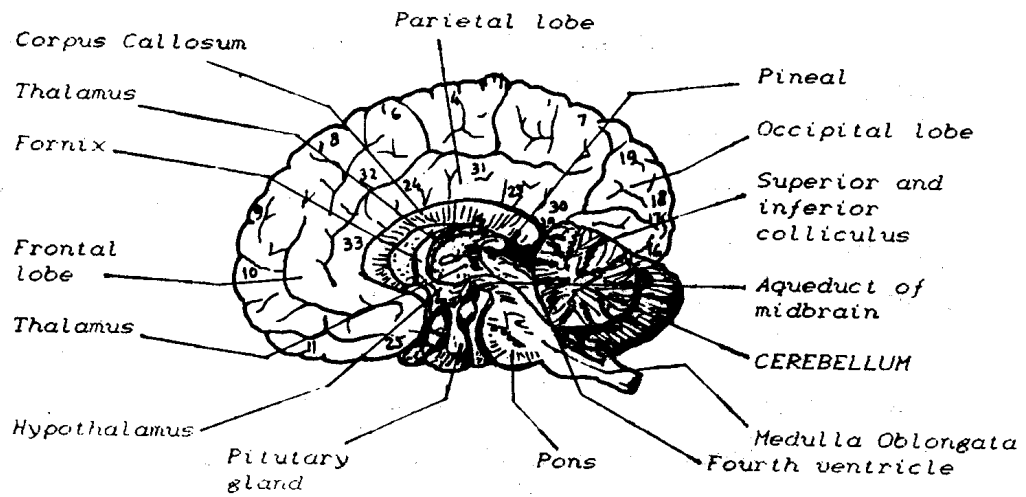
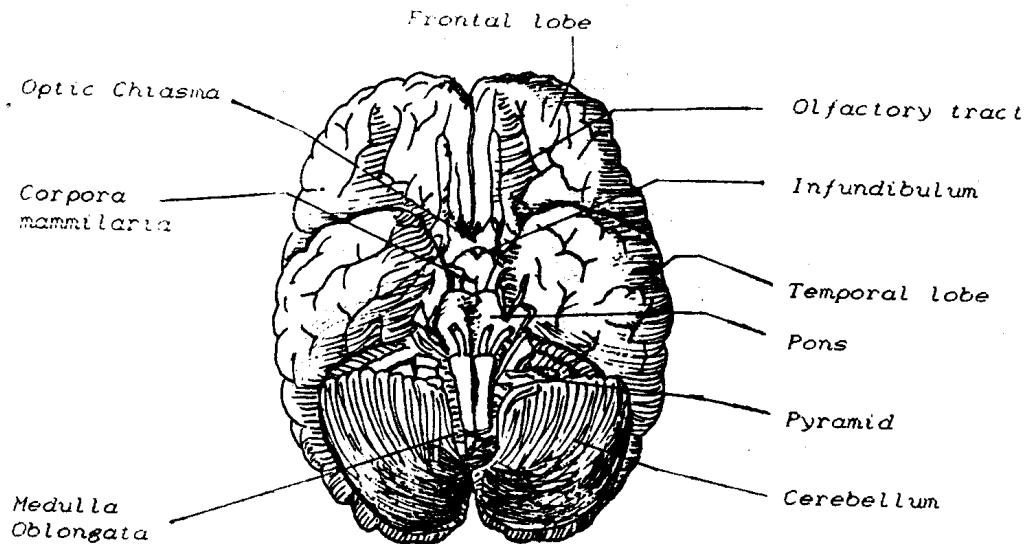


Fig. 1(b) The human brain from a side view.



**Fig. 1(c) Vertical cross section of the human brain**



**Fig.1(d) The under surface of the human brain**

A large bundle of fibres, the corpus callosum, serves to connect the two cortical hemispheres. The thalamus, a forebrain tissue, acts to process and relay visual information to the cortex. A complex structure located at the base of the forebrain, immediately below the thalamus is the hypothalamus. It is composed of many neurons and is in direct contact with the master endocrine gland called pituitary. It has been involved in wide variety of behaviours and processes.

## 1.2 Neurons

The basic unit of the brain is neuron. Neurons come in a variety of shapes and sizes, some are with one or two processes extending from the cell body, others can have many branchings (Figure 2). A neuron is like other cells in that it possesses a cell membrane enclosing Neurons have cell

cytoplasm.

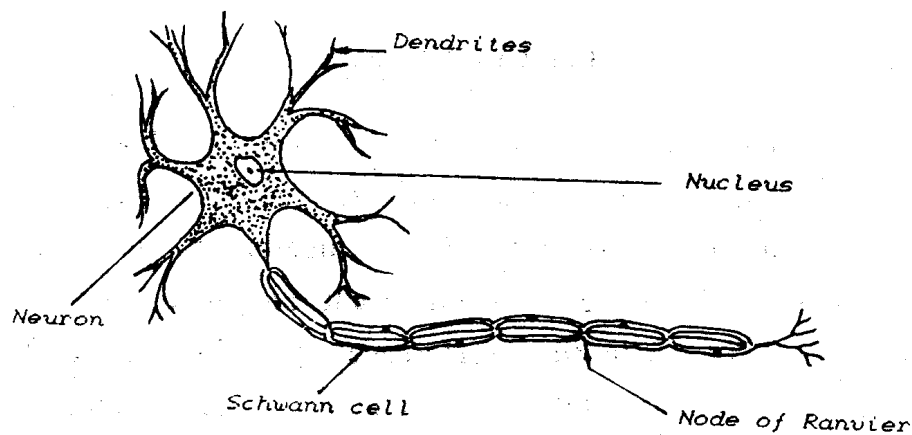


Fig.(2a) Elementary structure of a neuron

nucleus and the necessary metabolic machinery. They are specialized for the integration and transmission of information. The short branching processes extending from the cell body are dendrites. Dendrites receive information from other cells. The long process is the axon. The synapse refers to the region of communication between two neurons. The axon of one neuron does not touch the dendrite of the other, instead, there is a gap-the synaptic gap-between them.

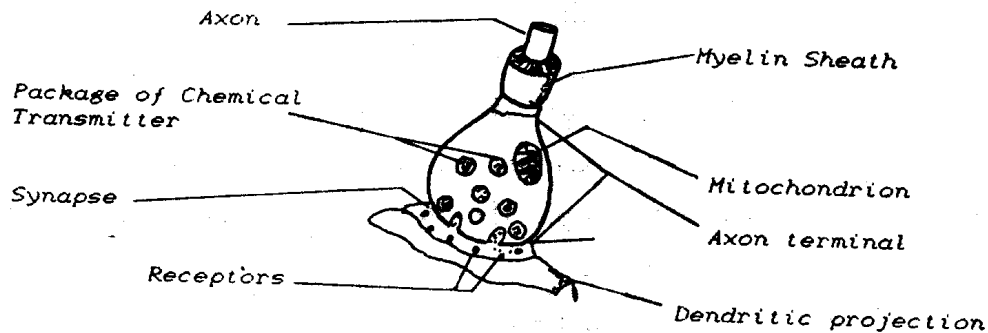


Fig.2(b) The Synapse

The entire surface of the cell is bounded by a membrane and the interior of the cell is negative with respect to the exterior. Cell membrane is selectively permeable to ions, the most important of which are  $\text{Na}^+$ ,  $\text{K}^+$  and  $\text{Cl}^-$ . The potential difference can be given by the Nernst equation, in terms of relative concentrations  $C_o$  and  $C_i$  [3], as

$$V = \pm (RT/F) \log (C_e/C_i) \quad (1)$$

There is less sodium and more potassium inside than the expected value. This disequilibrium defines a steady state, called the resting state of the nerve cell and the potential difference of  $V = -70$  mv is called the resting potential. According to Hodgkin and Huxley [4], the total current into the cell is given by

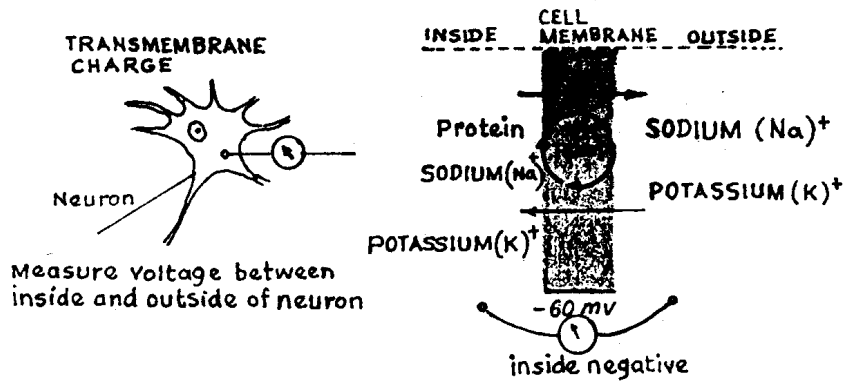


Fig.2(c) Features of a resting neuron

$$i = \sigma (\partial V/\partial t) + g_{Na} (V - V_{Na}) + g_K (V - V_K) \quad (2)$$

where  $V$  is the membrane potential,  $g_{Na}$  and  $g_K$  are conductivities for  $Na^+$  and  $K^+$  and  $\sigma$  is the capacity of the membrane. In resting state  $i = (\partial V/\partial t) = 0$ , so

$$V = (g_{Na} V_{Na} + g_K V_K) / (g_{Na} + g_K) \quad (3)$$

Above equation shows the  $g_{Na}$  and  $g_K$  are themselves dependent upon the potential difference  $V$ . A threshold value  $V_0$  (-60 mv to -50 mv) exist in normal circumstances, above which

the mutual interdependence of  $g_{Na}$  and  $V$  leads to increase of  $g_{Na}$  relative to  $g_K$ .  $g_K$  increases also but more slowly. Ultimately  $g_K$  again exceeds  $g_{Na}$  pulling  $V$  back to its resting value. The observed potential changes are called action potential. Let the axon be arbitrarily split up into a sequence of patches having resistance  $r$  between them. Then when  $g_{Na}$  is increased in one patch so that  $V$  across it rises from resting value this causes a current flow from through the next patch and depolarize it so that the action potential moves from one patch to another does the axon. This is the method of propagation of the action potential along axons and the dendrites.

When an action potential has passed down an axon and reached a synapse, it normally stops there and does not jump immediately across to other neuron. It stimulates the release of a neurotransmitter (Acetylcholine, ACh). It diffuses across the synaptic gap and alters the conductances of  $N^+$  and  $K^+$  which alters  $V$ . Immediately after the generation of the new signal, the Ach molecules are inactivated by the enzyme, Acholinesterase, hence the potential change due to an impulse arriving at a synapse does not persist indefinitely, but decays exponentially towards the resting potential.

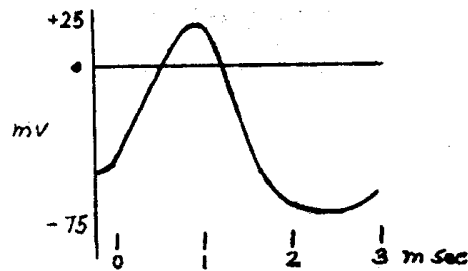


Fig. 2(d) A general nerve impulse as observed in axon.

### 1.3 Electrical Activity of the Brain

The constant flow of electrical signals along with nerve fibres suggest that some electrical activity might be observable in the brain [5]. The measurement of the brain activity is a limiting factor in our understanding of its functions. The limitations of the procedure and to the intrusive effects of the recording device on the normal operation of the brain. Electrical activity can be recorded by simply attaching electrodes directly to the scalp. The recording called an electroencephalogram or EEG, must be made with proper electrical filtering in order to avoid confusing the record of the brain's electrical signals with signals produced by muscle movement etc. (Figure 3). The neurons are synchronously active, the resultant record shows rhythmic values of various frequencies. This is called spontaneous activity and is usually marked by the presence of a dominant frequency which can be used to characterize

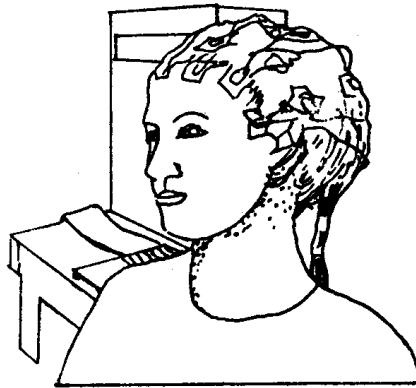


Fig.3(a) The electroencephalogram (EEG).  
the EEG pattern [6]. An important aspect of EEG is its close correlation with cognition and behaviour [7].

## 2. ELECTROCORTICAL ACTIVITY

If cortical cells are viewed as information processing networks, then electrocortical waves might be viewed as statistical shifts in average local depolarization. Determination of the laws of motion of these waves and their internal mechanisms of controls is a step towards an understanding of the brain. A major difficulty arises, because of the complexity and the non-linearity of the neural interactions [8].

Wright and Kydd [9] developed a linear model for the gross waves. They showed that the gross electrocortical activity will exhibit resonant modes of fixed natural frequency with specific dispersion relations. they assumed that the telencephalon is a system with modes of constant

natural frequency damped by input from the lateral hypothalamus and driven by the noise. starting with the differential equation for the system as

$$X_n + D_n \dot{X}_n + M_n^2 = X_0 \quad (4)$$

Where  $X_0(t)$  represents all processes driving the surface activity and surface signal is  $\Sigma^n X_i(t)$ ,  $M_n$  and  $D_n$  are the natural frequency and damping coefficient of a mode. They arrived at a theoretical expression for the EEG power spectrum given by

$$V^2(\omega) = |X_0|^2(\omega) |A(\omega)|^2 \left\{ \left[ \Sigma^n \frac{M_i - \omega^2}{(M_i^2 - \omega^2)^2 + D_i^2 \omega^2} \right]^2 + \left[ \Sigma^n \frac{-D_i \omega}{(M_i^2 - \omega^2)^2 + D_i^2 \omega^2} \right]^2 \right\} \quad (5)$$

If this is true description, then a unilateral hypothalamus electrolytic lesion will give an asymmetry in the damping coefficients. Considering the left and right average power spectra obtained before and after lesion, one can get the relative squared gain as

$$G^2(\omega) = K \frac{[\Sigma^n (M_i^2 - \omega^2) / \{(M_i^2 - \omega^2)^2 + D_{iLA}^2 \omega^2\}]^2}{[\Sigma^n (M_i^2 - \omega^2) / \{(M_i^2 - \omega^2)^2 + D_{iLX}^2 \omega^2\}]^2} \\ \frac{+ [\Sigma^n (-D_{iLA} \omega) / \{(M_i^2 - \omega^2)^2 + D_{iLA}^2 \omega^2\}]^2}{+ [\Sigma^n (-D_{iLX} \omega) / \{(M_i^2 - \omega^2)^2 + D_{iLX}^2 \omega^2\}]^2} \quad (6)$$

This model does not take into account of the magnetic field which are generated when there is a motion of charges. We write the equation for the potential of a segment of dendritic tree in the comoving frame of the signal. When this equation is transformed into the laboratory frame a magnetic vector potential appears along with electrostatic potential.

Let us introduce 4-potential  $A^\mu$ ;  $\mu=0, 1,2,3$  which can be written as

$$A^\mu = \begin{bmatrix} \Phi \\ A \end{bmatrix} \quad (7)$$

In the subsequent discussion the superscript  $\mu$  is dropped. In the comoving frame of the signal passing through a segment of the dendritic tree, the electrical potential can be represented as

$$\Phi_i + D_i(\tau) \Phi_i + N_i^2(\tau) \Phi_i = 0 \quad (8)$$

The Roman index  $i$  which runs from 1 to  $n$  denotes the number of dendritic trees considered in the model.  $\tau$  is time in the comoving frame of the signal. There is no summation over repeated Roman indices. However, repeated Greek indices denote summation. The parameters  $D_i(\tau)$  and  $N_i(\tau)$  are free parameters analogous to a damping coefficient and a natural frequency. In matrix form the above equation can be written as

$$\Phi_i + D_i(\tau) \Phi_i + N_i^2(\tau) \Phi_i = 0 \quad (9)$$

where

$$\Phi_i = \begin{bmatrix} \Phi_i \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \dot{\Phi}_i = d\Phi_i/d\tau, \quad \ddot{\Phi}_i = d^2\Phi_i/d\tau^2$$

etc. and

$$D_i(\tau) = \begin{bmatrix} D_i(\tau) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (10a)$$

$$N_i(\tau) = \begin{bmatrix} N_i(\tau) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (10b)$$

Now we transform equation (3) in the laboratory frame. Under the Lorentz transformation  $\lambda_i$  the four vectors and matrices transform as

$$\Phi_i \quad A_i = \lambda_i \Phi_i \quad (11a)$$

$$D_i(\tau) \quad \Delta_i(\tau) = \lambda_i D_i(\tau) \quad \lambda_i^T \quad (11b)$$

$$N_i(\tau) \quad \eta_i(\tau) = \lambda_i N_i(\tau) \quad \lambda_i^T \quad (11c)$$

Where  $\lambda^T$  denotes the transpose of matrix  $\lambda$ . Therefore equation (3) transform into the laboratory frame as

$$A_i + \Delta_i(\tau) A_i + \eta_i^2(\tau) A_i = 0 \quad (12)$$

$A_i$ ,  $A_i$  and  $A_i$  are all four vectors,  $\Delta_i(\tau)$  and  $\eta_i(\tau)$  are 4x4 matrices.

A mass of unit sources coupled to each other may be similarly represented by (j runs from 1 to n subject to the condition that  $i \neq j$ ).

$$A_i + \Delta_i(\tau) A_i + \eta_i^2(\tau) A_i = \sum_j K_j^i(\tau) A_j \quad (13)$$

Where  $\Delta_i^2(\tau)$ ,  $\eta_i(\tau)$ ,  $K_j^i(\tau)$  are free parameters (4x4 matrices).

Let  $\Omega_1 = A_1$ ,  $\Omega_2 = \Omega_1 = A_1$ ,  $\Omega_3 = A_2$ ,  $\Omega_4 = \Omega_3 = A_2$  to  $\Omega_{m-1} = A_n$ ,  $\Omega_m = \Omega_{m-1}$ ;  $m = 2n$ . In matrix representation,

$$dZ / d\tau = AZ \quad (14)$$

where

$$Z = \begin{bmatrix} \Omega_1 \\ \Omega_2 \\ \Omega_3 \\ \Omega_4 \\ \Omega_5 \\ \Omega_6 \\ \dots \\ \Omega_m \end{bmatrix} ; A = \begin{bmatrix} 0 & 1 & 0 & 0 \dots 0 & 0 \\ -\eta_1^2 & -\Delta_1 & K_2^1 & 0 \dots K_m^1 & 0 \\ 0 & 0 & 0 & 1 \dots 0 & 0 \\ K_1^2 & 0 & -\eta_2^2 & -\Delta_2 \dots K_n^2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ K_1^3 & 0 & K_2^3 & 0 \dots K_m^3 & 0 \\ \dots & \dots & \dots & \dots & \dots & \dots \\ 0 & 0 & 0 & 0 & 0 & 1 \\ K_1^n & 0 & K_2^n & 0 \dots \eta_n^2 & -\Delta_n \end{bmatrix} \quad (15a,b)$$

The matrix  $A$  is a  $4m \times 4m$  matrix and it will have  $4m$  eigenvalues. If  $\alpha$  represents an eigenvalue of the matrix  $A$ , the secular equation can be written as

$$\alpha I - A = 0 \quad (16)$$

where  $I$  is a  $4m \times 4m$  unit matrix. In the subsequent discussion we are dropping the subscript  $i$  which denotes the number of dendritic trees considered in the mode. Let us take  $\lambda$  in the form

$$\lambda_{oo} = \gamma, \quad \lambda_{op} = \lambda_{po} = \gamma v_p, \quad (p,q=1,2,3) \quad (17a,b)$$

$$\lambda_{pq} = \delta_{pq} + v_p v_q (\gamma - 1) / v^2, \quad (c = 1) \quad (17c)$$

where  $\delta_{pq}$  is kronecker delta given by

$$\delta_{pq} = 1 \quad \text{if } p = q \quad (18)$$

= 0 otherwise

and

$$\gamma = (1 - v^2)^{-1/2} \quad (19)$$

The form of  $\lambda$  chosen in equations (11a-c) gives

$$\Delta = \gamma^2 D \Xi \quad (20)$$

where  $\Xi$  is a 4x4 matrix whose elements are given by  $\Xi_{oo} = 1$ ,

$$\Xi_{po} = \Xi_{op} = v_p, \quad \Xi_{pq} = \frac{v_p v_q}{c^2}$$

Taking a boost in the z-direction and calculating the eigenvalues gives the following relationship between the laboratory frame and comoving frame damping coefficients

$$D_{lab} = D_{comoving} (1+v^2)/(1-v^2) \quad (21)$$

A similar calculation gives

$$N_{lab} = N_{comoving} (1+v^2)/(1-v^2) \quad (22)$$

Note that in the limit  $v \rightarrow 0$ ,  $D_{lab} \rightarrow D_{comoving}$  and

$$N_{lab} \rightarrow N_{comoving}$$

Now we discuss the cases when  $v \rightarrow 0$ ,  $v \rightarrow 1$  and finally  $v \ll 1$ .

Case 1:  $v \rightarrow 0$

In this case  $D_{lab} \rightarrow D_{comoving}$ ,  $N_{lab} \rightarrow N_{comoving}$  and our results are identical with the results of Wright and Kydd [9].

Case 2:  $v \rightarrow 1$

$D_{lab} \rightarrow \infty$ ,  $N_{lab} \rightarrow \infty$ . The left and right average power spectrum obtained before and after unilateral hypothalamic lesion in as close as possible to steady-state conditions can be written as [9].

$$G^2(\omega) = \frac{v_{LA}^2}{v_{LB}^2} : \frac{v_{CA}^2}{v_{CB}^2}(\omega) \quad (23)$$

$\omega$  is the frequency. The subscripts LA, LB, CA, CB indicate the lesion and control sides, after and before lesion. Substituting the theoretical expression of  $v$ 's and taking the limit  $v \rightarrow 1$ , we have

$$G^2(\omega) = 1 \quad (24)$$

This shows that there is no effect of lesion on EEG recordings. However, in reality this situation is rare because the velocities of signals are very much less than the velocity of light [10].

Case 3:  $v \ll 1$

In this case we can obtain first order approximation for the expression of the damping coefficients and the natural frequencies.

$$D_{lab} = (1+2v^2)D_{comoving}; N_{lab} = (1+2v^2)N_{comoving} \quad (25)$$

We have noticed that the state transition matrix and its eigenvalues are modified because of the appearance of a magnetic vector potential. Once we have four potential, different gauges (coulomb or Lorentz) could be used to simplify the resulting wave equations.

During the last few years there have been important discoveries in the application of biomagnetic methods to the investigation of cerebral physiology. In particular, the study of brain evoked responses has allowed a better understanding of the cortical area [11,12,13] and the initiation of the investigations of higher levels of brain functions. We hope that our ongoing calculations of the effects of external magnetic fields on the defined magnetic field will show some pronounced observable effects on magnetoencephalogram, MEG.

### 3. NEURON-NEURON INTERACTIONS

To describe brain's electrical activity, one has to consider many neurons, each interacting with many others at the synapses and with its environments. The important variables for a given neuron are the numbers of the ion-pairs within the neuron and its environment [14,15].

Let,  $a_i^*$  is an operator, which creates a pair of ions and  $a_i$  is the operator, which annihilates a pair of ion within a neuron. The number of ion pairs of opposite charge within the  $i$ th neuron can be written as  $n_i = a_i^* a_i$ . Similarly the number of pairs of ions in the environment of the  $i$ th neuron can be given as  $N_i = b_i^* b_i$ . These operators obey the boson commutation relations.

$$a_i a_j^* - a_j^* a_i = b_i b_j^* - b_j^* b_i = \delta_{ij}$$

The energy of the ions within  $i$ th neuron can be defined as

a sum of mean dissociation energy of the ion pair and the electrostatic energy of the ions. The mean dissociation energy is proportional to  $n_i$  and the electrostatic energy gets the form  $-n_i \exp[(n_i - m_i)/c]$ , where  $m_i$  and  $c$  are the numerical constants. The energy of the ions within the neurons gets the form

$$H_1 = \sum_i [e a_i^* a_i + E b_i^* b_i - a_i^* a_i \exp\{(a_i^* a_i - m_i)/c\}] \quad (26)$$

Besides the energy of the ions within each neuron and its environment, it is necessary to take into account interactions at the synaptic connection and at the membrane. These are proportional to  $a_i^* a_j$  and  $a_j^* a_i$  and  $a_i^* b_i + b_i^* a_i$ . It gives the following contribution to the energy.

$$H_2 = \sum_i [\lambda_i (a_i^* b_i + b_i^* a_i) + \sum_j V_{ij} a_i^* a_j] \quad (27)$$

Additional terms, representing the interaction of the system of neurons and its environment with the external world can be expressed as follows

$$H_3 = \sum_i [\delta E_i (a_i^* a_i - b_i^* b_i) + \delta \lambda_j (a_i^* b_i + b_i^* a_i)] \quad (28)$$

where  $\delta E_i$  and  $\delta \lambda_i$  are time dependent coefficients. The total hamiltonian  $H = H_1 + H_2 + H_3$  determines the dynamic properties of the system. The equations for the change of the system  $(\partial H / \partial a_i^*)$  and  $(\partial H / \partial b_i^*)$  can be written. It seems that the system of neurons then functions as a set of systems of coupled harmonic oscillators. An analysis of the normal modes will give the behaviour of the system. Neuron potentials can be simulated and a comparison with the EEG

recordings determines the coefficients.

#### 4. CONCLUSIONS

There are many theoretical as well as technical problems encountered in studying and characterizing the possible dynamics of the EEG. We have adopted and attacked two of the many approaches as described above [16]. Much remains to be done to understand the electrical activity of the brain and further work is in progress in our group.

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