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# A SEMICLASSICAL THEORY FOR NERVE EXCITATION BY A LOW INTENSITY ELECTROMAGNETIC FIELD

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A possible mechanism for microwave–neuron interaction, when the nerve is irradiated by a thermally insignificant electromagnetic field, is described. The radiation field is treated classically, but the atomic system which interacts with this field is treated quantum mechanically using the density matrix approach. Attention is given to both homogeneous and inhomogeneous broadening effects, and the degrading influence of inhomogeneous broadening upon the neural membrane's ability to interact with the electromagnetic field is shown.

*1. Introduction.* The pros and cons of the possibility of electromagnetic fields, primarily in the microwave regime, interacting in a non-thermal fashion with the human nervous system has recently received much attention in the literature; for example see Tell (1972), Johnson and Guy (1972), Schwan (1968) or Frey (1971). One motivation behind this growing interest is the government's desire to classify and define biologically hazardous field strengths for the purpose of validating existing safety standards or possibly initiating new standards.

It would be fair to say that the consensus of opinion of most U.S. researchers in this field would be that electromagnetic fields do not interact in a non-thermal fashion with biological systems. There have, however, been some interesting experimental studies apparently yielding results to the contrary. For example, Kamenskii (1964) has studied the effects of continuous and pulsed microwave fields on the functional state of the frog sciatic nerve. The main results of his study reveal that when the nerve is irradiated by a low intensity (12 mW/cm$^2$)

pulsed microwave field, increased rate of conduction of excitation and excitability of the nerve occurred. He suggests these changes in neural behavior are due to non-thermal interaction mechanisms. Also, "hearing" pulsed electromagnetic waves over a limited range of microwave frequencies is well documented (Frey, 1971). Finally, such widely different phenomena as irritability, loss of memory, headache, tremor, hallucinations, autonomic disorders, or disturbed sensory sensitivity as a result of exposure to low intensity microwave fields have been reported by researchers primarily in the Soviet Union (Pressman, 1965).

It is the purpose of this paper to consider plausible non-thermal mechanisms for neuron–electromagnetic field interaction at the molecular level, and to mathematically model the interaction. The theory presented is semiclassical in scope, that is, the electromagnetic field is treated classically and the atomic system which interacts with this field is treated quantum mechanically using the density matrix approach, which has gained wide usage in laser theory. Emphasis is focused on the microwave regime; however, the theory is general in scope.

*2. Double-Dipole Layer Model.* The recently proposed electric dipole theory of nerve excitation and conduction provides the basis from which the neuron–microwave interaction will be modeled. The double-dipole layer membrane model is based upon a widely accepted model proposed by Danielli and Davson (1935). The Danielli–Davson membrane model consists of a bimolecular leaflet of lipid and protein molecules organized and arranged in a highly specific manner. Noting that asymmetries of molecular structures give rise to electrical imbalances or polar properties, Wei proposes a membrane model composed of two layers of dipole molecules arranged in an orderly fashion. Recently, the electrical structure of this model has been extensively developed in a series of papers by Wei (1968, 1969a, b, 1971a, b), to explain molecular mechanisms of nerve excitation and conduction. Also, Arndt and Roper (1972) and Arndt *et al.* (1972) have done steady-state electro-diffusion calculations for a double-dipole layer model to satisfactorily fit axon membrane rectification data.

In essence, the dipole theory considers the nerve membrane to have a dipole layer on both the inner and outer surfaces of the membrane. Within the membrane, there is an excess of mobile negative ions, and in the immediate vicinity of the membrane in the aqueous phase, there is an excess layer of mobile positive ions whose concentration is greater than that in the bulk solution. In the resting state of the nerve, the majority of the outer and inner dipoles have their negative end facing the extracellular and intracellular fluid, respectively. The outer dipole layer plays the key role in whether or not the nerve is stimulated,



and in the resting state, it presents a potential barrier to the extracellular positive ions which keeps them from penetrating the membrane. Therefore, in order for the nerve to be stimulated, it becomes necessary to lower the outer potential barrier. It is apparent that this can be achieved if enough of the outer surface dipoles can be made to flip or rotate 180 deg so as to orient themselves in a direction which has the positive end of the dipole facing outward. Once the population of dipoles with the positive end facing outward becomes large enough, the potential barrier will be lowered sufficiently so as to permit an impulse of positive ions to penetrate the membrane. It goes without saying, that the reorientation of the dipoles can be achieved by application of an electric field.

There have been three experimental discoveries which lend most, but far from definitive, physical support for the dipole theory. They are: negative fixed surface charge (Segal, 1968), birefringence change (Cohen, Keynes and Hille, 1968), and infrared emission (Fraser and Frey, 1968). Wei (1971a) explains in detail how these discoveries relate to the dipole theory, however, a few words of explanation may be in order. The birefringence experiments reveal that it arises from sources arranged in a cylindrical region at the outer edge of the axon, and it occurred during the action potential. This birefringence change is easily explained as dipole flipping to another orientation during the action potential. The emission of infrared radiation, which incidently exceeded the blackbody radiation by two orders of magnitude, must be located at the surface of the nerve, and can be explained by the relaxation of the dipoles from their excited to ground states with the emission of energy quanta in the infrared band. It is obvious how the negative fixed surface charge relates to the dipole theory.

To lend further support for molecular rotation in biological membranes, Brown (1972) and Cone (1972) have shown conclusively that rhodopsin molecules, whose chromophores interact with light to initiate sight, are free to rotate in the rod membrane. Cone shows that the viscosity of the membrane site, in order to account for measured rhodopsin rotational diffusion relaxation times, must be comparable with the viscosity of light oils. This would imply that to describe such a membrane as rigid would be inappropriate, and hence the visual receptor membrane might be best described as highly fluid. He also suggests that the viscosity of this membrane should be comparable to that of nerve axons.

*3. States of a Hypothetical Dipole.* In principle, an electromagnetic field can interact with an atomic system through either the induced or permanent dipole moment associated with the molecule. If the molecule possesses neither, no interaction occurs. Thus, staying within the framework of the dipole theory,

there would appear to be two physical mechanisms available for neuron–microwave interaction. First, if membrane dipole reorientation is due to molecular rotation, it could be expected that the associated rotational spectrum should be in the microwave band, since the molecules involved are large. An external microwave field could therefore couple to the quantum mechanically allowed absorption bands. Secondly, it is reasonable to postulate that dipole flip-flop action can occur in much the same manner as the ammonia molecule in a microwave maser. This would be an inversion type motion in which one end of the dipole is able to push or tunnel its way through an energy barrier causing the dipole moment to be reversed, obviously corresponding to a 180-deg rotation of the dipole. Technically, inversion motion may be classified as vibrational motion. Vibrational frequencies typically occur in the infrared spectrum, however, due to the hindering action of the energy barrier, the inversion motion is slowed down. Consequently, the inversion frequency may exist well below the infrared spectrum. Thus if dipole flip-flop action occurs due to an inversion, the inversion frequency has a reasonable probability of occurring in the microwave spectrum.

In this paper we will consider the second possibility, since it is easier to relate the mathematical model to experimentally observable excitable membrane properties. As to whether or not this model has any bearing to reality, Wei (1971a) stated: "It is well known that a biological membrane is mostly a phospholipid which contains trimethylamine ions $-N^+\equiv(CH_3)_3$ near the surface. From its structure, this ion species must possess a permanent dipole moment. It is very probable that $N(CH_3)_3$ like $NH_3$ belongs to the class of symmetric top molecules and thus has a tetrahedral configuration. The $N^+$ ion sits at the apex and vibrates relative to the base plane of the three $CH_3$ molecules. Every time the $N^+$ inverses from in to out, the dipole moment is reversed corresponding to a 180-deg rotation."

With the above in mind, it will be assumed that the energy associated with each possible state of the dipoles is split into two energy levels. For example, if the energy associated with a particular vibrorotational state is $E_0$, it will be split into levels $E_0 + A$ and $E_0 - A$, where the energy difference of $2A$ is very small, corresponding to a frequency in the microwave band. The lower $(E_0 - A)$ and upper $(E_0 + A)$ energy levels will be assumed to possess a weak permanent dipole moment oriented inward and outward, respectively. Nerve stimulation could then be caused by irradiation from a low intensity source if the frequency of the field is $f = 2A/h$, where $h$ is Planck's constant.

It should be pointed out that due to interaction between the dipolar molecules, it is highly unlikely that discrete absorption lines exist. More likely, broad absorption bands can be expected.



*1. Dipole–Microwave Resonance Interaction.* The mathematical procedure used to describe the interaction of the dipolar molecules with the microwave field will be the density matrix approach. It has gained wide usage in the laser field, and the development of the necessary concepts and mathematical methods for various types of applications is aptly provided by such sources as Lamb (1964), Hopf and Scully (1969), Icsevgi and Lamb (1969), Maitland and Dunn (1969) and Stenholm and Lamb (1969). Consequently, we will proceed directly to the pertinent equations, and the reader is referred to the mentioned references for background information.

The field which is irradiating a nerve axon is a pulsed microwave signal. The pulse width, $\Delta t_0$, is long enough so that the signal is essentially a monochromatic wave and can be described by

$$\bar{E} = \bar{E}_0 \cos(\omega t - \bar{k}\cdot\bar{r} + \phi). \tag{1}$$

Since the dimensions of the nerve axom are much smaller than both the wavelength and spatial extent of the pulsed field, it is possible to neglect the $\bar{k}\cdot\bar{r}$ term. This allows the density matrix equations to become uncoupled from Maxwell's equations, resulting in a much simpler treatment of the problem. Removal of the spatial dependent $\bar{k}\cdot\bar{r}$ term, results in a perturbing field of the form

$$\bar{E} = \bar{E}_0 \cos(\omega t + \phi), \tag{2}$$

where $\bar{E}_0$, $\omega/2\pi$ and $\phi$ are the amplitude, frequency and phase, respectively, of the wave.

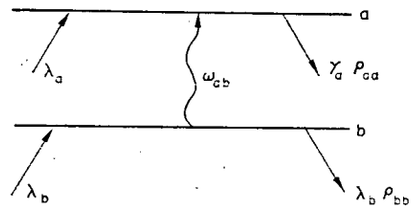


Figure 1. Energy level diagram for the two-level approximation of the active medium

We will assume the active medium of the nerve axon (outer dipole layer) to consist of $M$ two-level molecules per unit volume embedded in a homogeneous dielectric whose properties are constant over the microwave spectrum of interest. Figure 1 illustrates the atomic system under consideration. The lower energy level, $b$, and upper energy level, $a$, correspond to the physical states of the dipolar molecules in which the negative end is facing outward and inward,

respectively. Nerve stimulation occurs when enough dipoles make quantum transitions to state $a$. The resonance frequency of the two-level atomic system is denoted by $\omega_{ab}$. The quantities, $\gamma_a$ and $\gamma_b$, are the decay rates for the $a$ and $b$ energy levels, respectively. They account for the effects of finite atomic lifetime. The excitation rates, $\lambda_a$ and $\lambda_b$, account for the fact that the neuronal metabolic heat production probably interacts thermally with the dipolar molecules by "pumping" them up to states $a$ and $b$. It is also conceivable that the radiation field may also contribute to $\lambda_a$ and $\lambda_b$ due to the generation of heat by interaction of the field with tissues and fluids surrounding the neuronal membrane.

The density matrix for the two-level system as described by Figure 1 takes the form

$$\rho(t) = \begin{bmatrix} \rho_{aa}(t) & \rho_{ab}(t) \\ \rho_{ba}(t) & \rho_{bb}(t) \end{bmatrix}. \tag{3}$$

The diagonal terms, $\rho_{aa}(t)$ and $\rho_{bb}(t)$, are the populations, per unit volume, of the $a$ and $b$ energy levels, respectively. The off-diagonal terms, $\rho_{ab}(t)$ and $\rho_{ba}(t)$, have physical relevance in that the induced partial polarization of the active medium is defined by

$$\bar{P} = \bar{p}(\rho_{ab} + \rho_{ba}), \tag{4}$$

where $\bar{p}$ is the average dipole moment of the active molecules.

The equation of motion of the density matrix is produced by taking the time derivative of (3). It can be shown (for instance, see Maitland and Dunn, 1969) that the elements of the density matrix obey the following differential equations:

$$\frac{\mathrm{d}}{\mathrm{d}t}\rho_{aa}(t) = \gamma_a\left(\frac{\lambda_a}{\gamma_a} - \rho_{aa}\right) + i\frac{V}{\bar{h}}(\rho_{ab} - \rho_{bc}) \tag{5}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}\rho_{ab}(t) = i\frac{V}{\bar{h}}(\rho_{aa} - \rho_{bb}) - (i\omega_{ab} + \gamma_{ab})\rho_{ab} \tag{6}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}\rho_{bb}(t) = \gamma_b\left(\frac{\lambda_b}{\gamma_b} - \rho_{bb}\right) + i\frac{V}{\bar{h}}(\rho_{ba} - \rho_{ab}) \tag{7}$$

$$\rho_{ba} = \rho_{ab}^*, \tag{8}$$

where

$\gamma_{ab}$ = decay constant for the dipole moment of the active molecules,
$V = -\bar{p}\cdot\bar{E}$ = perturbation term for the interaction with the radiation field which induces transitions between the two levels,
$\bar{p} = e\bar{r}_{ab}$ = average dipole moment of the active molecules,



$\bar{r}_{ab} = \int \psi_a \bar{r} \psi_b^* \, \mathrm{d}^3r$ = strength of the transition,
$\psi_a, \psi_b$ = normalized quantum states corresponding to the upper and lower energy levels,
$\bar{h} = h/2\pi$,
$e$ = electronic charge,
$i = \sqrt{-1}$,
$*$ = complex conjugate.

The decay constant, $\gamma_{ab}$, accounts for homogeneous broadening effects such as dipole–dipole and thermal interaction mechanisms. In general, $\gamma_{ab}$, is greater than $1/2(\gamma_a + \gamma_b)$. The assumed weak permanent dipole moments associated with states $a$ and $b$ were neglected in the above equations.

To account for inhomogeneous broadening mechanisms, such as different active molecules having different resonant frequencies due to variations in the immediate surroundings of the molecules, one must average the solutions to (5)–(8). For instance, the total induced polarization would be given by multiplying (4) by an appropriate distribution function, $F(\omega_{ab})$, and integrating over the limits of the distribution, i.e.

$$\bar{P} = \bar{p}\int_{-\infty}^{\infty} [\rho_{ab}(\omega_{ab}) + \rho_{ba}(\omega_{ab})]F(\omega_{ab})\,\mathrm{d}\omega_{ab}. \tag{9}$$

In this paper, because no information exists regarding the functional form for $F(\omega_{ab})$, we will either neglect inhomogeneous effects by assuming

$$F(\omega_{ab}) = \delta(\omega_{ab} - \bar{\omega}_{ab}), \tag{10}$$

where $\delta$ is the delta function; or assume that inhomogeneous effects are realistically modeled by a Gaussian curve which is given by

$$F(\omega_{ab}) = \frac{1}{\sigma\sqrt{2\pi}} \exp -(\omega_{ab} - \bar{\omega}_{ab})^2/2\sigma^2, \tag{11}$$

where $\sigma^2$ and $\bar{\omega}_{ab}$ are the variance and average value, respectively, of the random variable, $\omega_{ab}$.

*5. Solution by Long Pulse Approximation.* A general analytic solution for (5)–(8) is a fairly difficult proposition, and consequently one must resort to approximations which are valid for the problem under consideration. For our case, it is reasonable to stipulate that the pulse duration, $\Delta t_0$, is much longer than the decay constant for the dipole moment of the active molecules, $1/\gamma_{ab}$, i.e.

$$\frac{1}{\gamma_{ab}} \ll \Delta t_0.$$

Also, it is expedient to remove the fundamental frequency from the off-diagonal elements of the density matrix by writing

$$\rho_{ab} = \rho_1 e^{-i\omega t} \tag{12}$$

$$\rho_{ba} = \rho_1^* e^{+i\omega t}, \tag{13}$$

and to define the following terms:

$$N = \rho_{bb} - \rho_{aa}, \qquad S = \rho_1 - \rho_1^*$$

$$M = \rho_{bb} + \rho_{aa}$$

$$C = \rho_1 + \rho_1^*, \qquad I = \frac{\bar{E}_0 \cdot \bar{p}}{\hbar}.$$

Substitution of the above definitions into (5)–(8) allow the basic equations to be recast into a form more amenable for solution. The result is:

$$\frac{dN(t)}{dt} = (\lambda_b - \lambda_a) - \gamma N(t) - iIS(t) \tag{14}$$

$$\frac{dM(t)}{dt} = (\lambda_b + \lambda_a) - \gamma M(t) \tag{15}$$

$$\frac{dC(t)}{dt} = -\gamma_{ab} C(t) + i(\omega - \omega_{ab}) S(t) \tag{16}$$

$$\frac{dS(t)}{dt} = -\gamma_{ab} S(t) + i(\omega - \omega_{ab}) C(t) + iIN(t), \tag{17}$$

in which it was assumed that $\gamma_a = \gamma_b = \gamma$. Terms which contained $e^{\pm i2\omega t}$ were neglected in all the equations. The equation for the total density of active molecules is solved immediately, with the result

$$M(t) = \left(M_0 - \frac{\lambda_b + \lambda_a}{\gamma}\right) e^{-\gamma t} + \frac{\lambda_b + \lambda_a}{\gamma}, \tag{18}$$

where $M_0$ is the initial value or the resting state value of the population density. If $M_0 = (\lambda_b + \lambda_a)/\gamma$, then

$$M(t) = \frac{\lambda_b + \lambda_a}{\gamma}, \tag{19}$$

which implies that $M(t)$ is a constant. Since we will take the total number of molecules available for interaction with the external field to be fixed, it will henceforth be assumed that $M(t)$ is given by (19).

Since $C(t)$ and $S(t)$ vary slowly in a time $1/\gamma_{ab}$, it is possible to set

$$\frac{dC(t)}{dt} = \frac{dS(t)}{dt} = 0.$$



Thus the solutions to (16) and (17) are given by

$$S(t) = \frac{iI\gamma_{ab} N(t)}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2} \tag{20}$$

$$C(t) = \frac{(\omega - \omega_{ab}) IN(t)}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2}. \tag{21}$$

Now if (20) is substituted into (14), it is straightforward to show that

$$N(t) = N_{ss} + [N_0 - N_{ss}] \exp - \left[\gamma + \frac{I^2 \gamma_{ab}}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2}\right] t, \tag{22}$$

where

$$N_{ss} = (\lambda_b - \lambda_a)\left[\gamma + \frac{I^2 \gamma_{ab}}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2}\right]^{-1}. \tag{23}$$

The quantity $N_0$ is the initial value or the resting state value of the population difference between the energy levels $b$ and $a$ when no radiation field is present. Substitution of (22) into (20) and (21) completes the solution for (14)–(17).

For completeness, we also desire an expression for the induced partial polarization (Equation 4). From (12) and (13) it is evident that

$$\bar{P} = \bar{p}[C(t) \cos \omega t - iS(t) \sin \omega t]. \tag{24}$$

The insertion of (20) and (21) into (24) yields the desired result

$$\bar{P} = \frac{\bar{p} IN(t)}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2} [(\omega - \omega_{ab}) \cos \omega t + \gamma_{ab} \sin \omega t]. \tag{25}$$

With the exception of averaging for inhomogeneous broadening effects, the complete solution (within our approximations) for the problem of microwave–dipole resonant interaction has been specified. We now turn our attention to how these expressions relate to some physically observable excitable membrane properties. We will also briefly consider inhomogeneous effects.

*6. Quantum Description of Excitable Membrane Properties*

(a) *Homogeneously Broadened System.* As previously mentioned, a homogeneously broadened system is mathematically represented by the distribution function given by (10). Although a homogeneously broadened model cannot completely describe the true physical interaction mechanisms existing between the dipolar molecules, the analysis of nerve properties using this model still should yield information as to the general tendencies of the neuron–microwave interaction. It will be shown how the previously presented theory can account

for such well-known excitable membrane properties as the all-or-none response, the strength–duration relation, no-threshold excitation at elevated temperatures, refractoriness and refractory period, and birefringence change.

According to the dipole theory, nerve excitability is primarily dependent on the outer surface potential barrier, and not on membrane behavior *per se*. The equation for the barrier potential is given by

$$V_0 = N(t)\frac{qs}{\varepsilon}, \tag{26}$$

where $N(t)$ is the population difference density between states $a$ and $b$, as given by (22). The other quantities $q$, $s$ and $\varepsilon$ are the magnitude of charge of a single pole, barrier width and permittivity, respectively. It is obvious that lowering the potential barrier amounts to reducing the population difference between the two states. Nerve excitation occurs when $V_0$ is reduced below a critical value of say $V_T$. This in turn implies that $N(t)$ must be reduced to a value below a threshold value, $N_T$. Thus (22) is of paramount importance when discussing the all-or-none response phenomenon, and in fact most of the properties considered here. In considering this equation, it is evident that the external signal is attempting to drive $N(t)$ exponentially to the steady-state value, $N_{ss}$. Nerve stimulation could occur if $N_{ss} < N_T$. The speed at which the threshold condition is achieved is controlled by such factors as the intensity, $E_0^2$, and frequency, $\omega/2\pi$, of the radiation field, the strength of the allowed quantum transition, $\bar{r}_{ab}$, and the decay rates, $\gamma_{ab}$ and $\gamma$. Whether or not $N(t)$ is driven below the threshold value, $N_T$, is also dependent upon the duration of the pulse, $\Delta t_0$, and the resting state population difference, $N_0$. It would appear reasonable to postulate that a suitably chosen combination of the external quantities $E_0^2$, $\omega/2\pi$, and $\Delta t_0$ could drive the population difference below threshold, thereby inducing nerve stimulation. This fact leads us to the so-called strength–duration relationship.

For our problem, a simple strength–duration relationship is not very informative. It would be more meaningful to speak in terms of a strength–duration–frequency relation. For instance, from (22) with $N(t) = N_T$, it is a simple matter to show that a minimum pulse length of

$$\Delta t_0 = \left[\gamma + \frac{I^2\gamma_{ab}}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2}\right]^{-1} \log\left[\frac{N_0 - N_{ss}}{N_T - N_{ss}}\right] \tag{27}$$

would be required for stimulation. As the pulse length is decreased, the efficacy of the stimulus can be maintained by either a corresponding increase in the strength of the signal or by adjusting the signal frequency closer to resonant line center.



The refractory period is determined by a consideration of (14) with no external stimulus present, i.e. $I = 0$. The basic equation we must solve is then

$$\frac{d}{dt}N(t) = (\lambda_b - \lambda_a) - \gamma N(t), \tag{28}$$

with a solution represented by

$$N(t) = \frac{\lambda_b - \lambda_a}{\gamma} + \left[N(\Delta t_0) - \frac{\lambda_b - \lambda_a}{\gamma}\right] e^{-\gamma(t-\Delta t_0)}, \tag{29}$$

where $N(\Delta t_0)$ is the value of $N(t)$ at the end of the first stimulus. It is seen that in the interval between two stimuli, $N(t)$ is relaxing back to its resting state value of

$$N_0 = \frac{\lambda_b - \lambda_a}{\gamma}, \tag{30}$$

with a time constant given by $1/\gamma$. If it is assumed that $N(t)$ is driven below threshold at the end of the first stimulus, for the nerve to respond to the second stimulus, it is necessary for $N(t)$ to have relaxed to a value greater than $N_T$ in the interval between the two stimuli. From (29), this would require a minimum time given by

$$\Delta t_r = \frac{1}{\gamma}\log\left[\frac{N_0 - N(\Delta t_0)}{N_0 - N_T}\right]. \tag{31}$$

The refractory period is consequently defined by (31).

This brings us to the subject of no-threshold excitation mechanisms. This effect is manifested in the pumping rate difference, $(\lambda_b - \lambda_a)$. As previously mentioned, it appears logical to assume the pumping rates, $\lambda_a$ and $\lambda_b$, are primarily thermally controlled. For thermally insignificant field strengths the pumping rates would be dictated by neuronal metabolic heat production. On the other hand, if the external signal contributed significantly to the pumping rates, it would be reasonable to speak in terms of a thermal steady-state population difference (see (23) with $I = 0$) given by

$$N_{ss} = \frac{\lambda_b - \lambda_a}{\gamma}. \tag{32}$$

If the thermal steady-state value is barely larger than the threshold value, $N_T$, this condition would make the field threshold essentially imperceptible.

The birefringence change property can probably be related to the induced polarization, as given by (25). No attempt will be made to do so in this paper.

(*b*) *Inhomogeneously Broadened System.* To achieve a model which more

closely approximates reality, it is necessary to incorporate inhomogeneous broadening mechanisms into the analysis. To include these effects, one must average $N(t)$ over the Gaussian distribution function, $F(\omega_{ab})$ of (11), as

$$N(t) = \int_{-\infty}^{\infty} N(t, \omega_{ab})F(\omega_{ab})\, d\omega_{ab}. \tag{33}$$

The evaluation of this integral in the most general sense would be a rather formidable task. Therefore, we will only consider how inhomogeneous broadening mechanisms affect the steady-state population difference. This allows (33) to be written as

$$N_{ss} = \int_{-\infty}^{\infty} N_{ss}(\omega_{ab})F(\omega_{ab})\, d\omega_{ab}, \tag{34}$$

and upon the insertion of (11) and (23), we arrive at

$$N_{ss} = \frac{\lambda_b - \lambda_a}{\sigma\sqrt{2\pi}} \int_{-\infty}^{\infty} \left[\gamma + \frac{I^2\gamma_{ab}}{\gamma_{ab}^2 + (\omega - \omega_{ab})^2}\right]^{-1} \exp -[(\omega_{ab} - \bar{\omega}_{ab})^2/2\sigma^2]\, d\omega_{ab}. \tag{35}$$

To make matters even simpler, the external pulse frequency is adjusted to $\bar{\omega}_{ab}$. The integral can then be transformed into

$$N_{ss} = \frac{(\lambda_b - \lambda_a)}{\sigma\sqrt{2\pi}} \int_0^{\infty} \frac{\gamma_{ab}^2 + y}{(y + \beta)y^{1/2}}\, e^{-ky}\, dy, \tag{36}$$

where

$$k = \frac{1}{2\sigma^2}$$

$$\beta = \gamma_{ab}\left(\gamma_{ab} + \frac{I^2}{\gamma}\right)$$

$$y = (\omega_{ab} - \bar{\omega}_{ab})^2.$$

This integral is tabulated in the Bateman Series (1954), so that (36) becomes

$$N_{ss} = \frac{(\lambda_b - \lambda_a)}{\sigma\gamma(2\pi)^{1/2}} \left\{\frac{\gamma_{ab}^2\pi\, e^{\beta k}}{\beta^{1/2}}[1 - \text{erf}\,(\sqrt{k\beta})] + \left(\frac{\pi}{k}\right)^{1/2} - \pi\beta^{1/2}\, e^{\beta k}[1 - \text{erf}\,(\sqrt{k\beta})]\right\}, \tag{37}$$

where erf is the error function.

It will now be shown that the steady-state inhomogeneous population difference as given by (37), and henceforth denoted as $N_{ss}^{(i)}$, is larger than the corresponding homogeneous population difference, $N_{ss}^{(h)}$. This requires the consideration of (37) under two limiting conditions. First, if the inhomogeneous linewidth approaches infinity ($k \to 0$), this condition causes erf $\to 0$, so that (37) reduces to

$$N_{ss}^{(i)} \to N_0, \tag{38}$$

where

$$N_0 = \text{resting state population difference} = \frac{\lambda_b - \lambda_a}{\gamma}.$$

The other condition, $k \to \infty$ (homogeneous broadening mechanisms dominate), causes $1 - \text{erf}\,(\sqrt{k\beta}) \to e^{-k\beta}/\sqrt{\pi k\beta}$, resulting in

$$N_{ss} \to N_{ss}^{(h)}, \tag{39}$$

where

$$N_{ss}^{(h)} = \frac{N_0}{1 + I^2/\gamma\gamma_{ab}}. \tag{40}$$

From (40) it is clear that $N_0 > N_{ss}^{(h)}$ and thus the inequality

$$N_{ss}^{(h)} < N_{ss}^{(i)} < N_0 \tag{41}$$

must be satisfied.

The degrading influence inhomogeneous broadening has on nerve stimulation by microwaves is very apparent. For example, if the external stimulus had driven the population difference to just the threshold value for a homogeneously broadened system, the same stimulus would not drive an inhomogeneously broadened system to threshold since $N_{ss}^{(i)}$ would not be less than $N_T$. It is a relatively simple matter to consider the effects of inhomogeneous broadening on the other excitable membrane properties. In all cases, it has a degrading influence upon the membrane's ability to interact strongly with the microwave signal.

*7. Discussion and Conclusion.* A possible mechanism for direct microwave stimulation of nerves has been discussed and mathematically presented in rather complete fashion. No numerical illustrations were presented since it was felt that applicable information regarding the necessary quantities was virtually nil. In this regard, it is hoped that this effort stimulates some experimental ventures along these lines. For example, it would appear that microwave spectroscopy techniques could prove useful in determining such parameters as the dipole moment and relaxation time of the surface dipoles. If a microwave absorption spectrum can be measured, this would be direct proof that microwaves and neurons interact in a non-thermal fashion. Analysis of the absorption spectrum, coupled with the theory presented here, could potentially provide

the necessary insight for understanding neural membrane biophysical mechanisms at the molecular level. Lastly, it is felt the density matrix approach itself should be of intrinsic interest, since the method is general in scope, and could have other applications when studying radiation effects on biological systems.

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