Effects of local feedback on dispersion of electrical waves in the cerebral cortex

C. J. Rennie,[1,2,*] P. A. Robinson,[1,†] and J. J. Wright[3,‡]
[1]*School of Physics, University of Sydney, Sydney, New South Wales 2006, Australia*
[2]*Department of Medical Physics, Westmead Hospital, Westmead, New South Wales 2145, Australia*
[3]*Mental Health Research Institute, Parkville, Victoria 3052, Australia*
(Received 5 May 1998; revised manuscript received 14 August 1998)

This paper generalizes an existing continuum model of the large-scale electrical activity of the brain by incorporating local feedback. The model is first reformulated by parametrizing neuronal function in more detail and we then show that only four broad classes of local feedback are consistent with general physiological constraints. The corresponding linear dispersion relations are found to contain additional modes as a result of feedback. It is shown that each feedback class can produce lightly damped or undamped oscillatory modes at frequencies similar to those seen in electroencephalography, although not all combinations of type and speed of feedback result in oscillatory behavior. Three kinds of lightly damped resonances are found, each with distinctive frequency characteristics. The effect of feedback on other roots is also described.
[S1063-651X(99)05901-2]

PACS number(s): 87.22.Jb, 87.22.As, 87.10.+e

I. INTRODUCTION

Continuum approximations offer a means of modeling the large-scale electrical activity of the brain and current models incorporate the principal features of the brain's structure and physiology, including distinct neural populations, nonlinearities, and the conduction velocities of impulses [1–10]. However, feedback has not previously been considered in such models, despite being a demonstrable feature of brain physiology and a potential reason for the periodic appearance of electrical signals recorded from the brain. The purpose of this paper is (a) to find a way of categorizing the many potential forms of local feedback and (b) to examine the consequences of each kind of feedback. This is done in the context of a recent continuum model of brain electrical activity [11,12].

Brain activity causes potentials on the scalp and recordings of these potentials, called electroencephalograms (EEGs), are used as a measure of brain function. EEGs contain spatial and temporal features that are sufficiently consistent that correlations can be made with mental state; however, the correlations are based on coarse parametrization of the EEGs so their specificity is poor. If, with the help of a model, EEGs could be quantified more in terms of underlying anatomical and physiological quantities, their value as a probe of brain function would be greatly enhanced.

In modeling EEGs there is an abundance of anatomical and neurophysiological information that can be drawn upon, although it must be understood that the huge number of neurons in the human brain ($\sim 10^{11}$) and their intricate structure and interconnections preclude modeling the whole cortex down to the scale of individual neurons. An alternative is to use continuum equations that average over times and distances much less than are characteristic of EEGs. Early such works were those of Wilson and Cowan [1], Nunez [2], and van Rotterdam *et al.* [3], followed by generalizations by Nunez [4], Jansen and Rit [5], Wright and co-workers [6–8], and Jirsa and Haken [9,10]. Some sought to account for the general characteristics of EEG, while others made a connection with cognition and behavior.

A recent continuum model, proposed by Robinson *et al.* [11], treated the propagation of neuronal activity between neurons using a wave equation. This approach, when combined with a model of transmission within neurons including nonlinear elements, was found to facilitate both analytical and numerical modeling of electrical activity in two dimensions and has been used for estimating two-point correlation functions [12] and in studying global cortical dynamics [13].

This paper extends the model of Robinson *et al.* to incorporate more physiological detail. We are particularly concerned with the many metabolic and biophysical processes involved in chemical transmission between neurons and the resulting changes in membrane properties, each of which has the effect of modulating the responsiveness of neurons. The approach adopted here is to consider them as instances of local feedback. The goal is to find tractable representations of these feedbacks that are sufficiently general to cover the numerous physiological possibilities. A further goal is to characterize the classes of feedback through their linear dispersion relations.

One likely result of feedback in cortical models is to induce resonances that may be of relevance to the modeling of EEGs. This is in contrast to previous versions of our model [11,12] which lacked feedback and showed no spectral peaks typical of EEGs [4] when driven by wideband noise. While the possibility also exists of subcortical generators for EEG components [14], resonances induced by local feedback may also affect the signals recorded from the cortex and scalp. As an application of the classification and characterization of feedbacks, we look at the circumstances in which feedback leads to resonances.

Section II briefly reviews the model of Robinson *et al.* [11]. Section III describes the general approach by which

*Electronic address: rennie@physics.usyd.edu.au
†Electronic address: robinson@physics.usyd.edu.au
‡Electronic address: jjw@mhri.edu.au

feedback is introduced and identifies just four distinct classes of feedback. The dispersive characteristics of the different classes are described in Sec. IV. In Sec. V implications for electrocortical modeling are discussed.

II. BASIC CORTICAL MODEL

This section outlines the model that is the basis of the subsequent sections. First it briefly reviews the model as it appeared in previous work [6–8,11,12], with some notational changes that allow us to incorporate feedback. We then outline the steady state solutions and the dispersion relations that result.

A. Basic equations

Neurons are diverse and intricate in their structure and function; however, their density is such that at scales of greater than a few tenths of a millimeter it is valid to approximate the cortex as a homogeneous continuum. Several continuum models have been proposed [1–12]. The cortex is commonly assumed to be two dimensional, which is justified by its thinness (2–5 mm in humans) compared to its circumference (~ 600 mm).

Another assumption common to continuum models is that the properties at any point are simply the average of those of the population of real neurons in the vicinity. However, one distinction between neurons is maintained: Some release *excitatory* chemical transmitters at their terminals while others release *inhibitory* transmitters. The effect of the former is to depolarize the target neuron and make it more likely to fire, while the latter have the opposite effect.

The operation of neurons consists of four stages: combination of all incoming activities, transmission of summed activity to the main body of the neuron, consequent modulation of the neuron's level of activity, and propagation to neighboring neurons. The first of these processes, sometimes referred to as spatial summation, is the combination of impulses arriving from other locations in the cortex and from deep within the brain. This takes place in the input or *dendritic* part of the neuron. The dendrites are fibers projecting from the main body of the neuron, which serve to increase the neuron's receptive area for connections (*synapses*) from other neurons. We use $\phi_e(\mathbf{r},t)$ and $\phi_i(\mathbf{r},t)$ to represent the excitatory and inhibitory activity reaching a particular point from other points in the cortex, expressed as a rate of impulses or *action potentials* (rather than being normalized as in our previous work [11]). For this reason the ranges of $\phi_{e,i}$ are limited by the maximal rate at which action potentials can be generated $Q_{e,i}^{\max}$, so that $0 \leqslant \phi_{e,i} \leqslant Q_{e,i}^{\max}$. Activity $\phi_s(\mathbf{r},t)$ also reaches the cortex from subcortical sites. The net effects $P_{e,i}$ on excitatory and inhibitory neurons are sums of cortical and subcortical contributions

$$P_e(\mathbf{r},t) = N_{ee}s_e\phi_e(\mathbf{r},t) + N_{ei}s_i\phi_i(\mathbf{r},t) + N_{es}s_s\phi_s(\mathbf{r},t), \tag{1a}$$

$$P_i(\mathbf{r},t) = N_{ie}s_e\phi_e(\mathbf{r},t) + N_{ii}s_i\phi_i(\mathbf{r},t) + N_{is}s_s\phi_s(\mathbf{r},t). \tag{1b}$$

Each of the coupling strengths in Eqs. (1) has two components: N_{qp}, which is anatomical or structural in character, and s_p, which is physiological or functional in nature; N_{qp} is the mean number of connections from neurons of type $p = e,i,s$ on a neuron of type $q = e,i$ and s_p is the size of the impulse response associated with synapses of type $p = e,i,s$. The latter is the time integral of the perturbation to the transmembrane potential, as measured at the synapse. Table I lists representative values of these factors, which are used in later numerical results. Note that s_i is negative in keeping with its inhibitory character and is greater in magnitude than s_e to reflect experimental evidence better [15]. However, the parameters can be considered arbitrary in the present context, as we are principally concerned with general results.

TABLE I. Parameters for the human cortex, based on [11] and the additional assumptions $\Sigma N_{ep} = \Sigma N_{ip} = 5000$, $|s_i| = 4s_e$, $Q_{e,i}^{\max} = 200\ \mathrm{s}^{-1}$, $l_{e,i} = 0.1$, and $\theta_{e,i} = 0.015$ V.

Parameter	Value
N_{ee}	4826
N_{ei}	130
N_{es}	44
N_{ie}	4789
N_{ii}	166
N_{is}	46
s_e	2.0×10^{-6} V s
s_i	-8.0×10^{-6} V s
s_s	2.0×10^{-6} V s
$l_{e,i}$	0.1
α,β	200 s^{-1}
$Q_{e,i}^{\max}$	200 s^{-1}
$\sigma_{e,i}$	0.005 V
$\theta_{e,i}$	0.015 V
v	9 m s^{-1}
r_e	0.084 m
r_i	0.0001 m

Each pulse arriving at a synapse causes a brief localized increase in membrane conductivity, leading to a perturbation of several millivolts in the potential across the membrane. Each perturbation spreads through the dendritic tree and reaches the body of the neuron with some attenuation and lag. Both depend on the distance of the synapse from the cell body; a factor $l_{e,i}$ can be used to represent the average attenuation and convolution of $P_{e,i}$ with a unit-area function $L(t)$ can be used to represent lag. This results in the following expression for the net depolarization at the body of the two classes of neurons:

$$V_{e,i}(\mathbf{r},t) = l_{e,i}\int_{-\infty}^{\infty} L(t-t')P_{e,i}(\mathbf{r},t')dt' \tag{2}$$

$$= l_{e,i}L(t) \otimes P_{e,i}(\mathbf{r},t), \tag{3}$$

where the symbol $\otimes$ indicates convolutions of the kind in Eq. (2) and $L(\tau)$ is a non-negative function that is zero for $\tau < 0$ and has unit area. Experiments have suggested the following approximation for $L(\tau)$:

$$L(\tau)=\begin{cases}\dfrac{\alpha\beta}{\beta-\alpha}(e^{-\alpha\tau}-e^{-\beta\tau}), & \alpha\neq\beta \quad (4)\\ \alpha^2\tau e^{-\alpha\tau}, & \alpha=\beta, \quad (5)\end{cases}$$

for $\tau\geqslant 0$, and $L(\tau)=0$ otherwise [16]. The time constants $1/\alpha$ and $1/\beta$ are of order 5 ms, which is greater than the duration of an action potential, so the process (2) is sometimes described as temporal summation, in contrast to the spatial summation in Eqs. (1).

In the absence of synaptic input, the transmembrane potential of a neuron tends to a resting value of about -70 mV with respect to the exterior of the cell. For simplicity, this offset is omitted from Eq. (3) and from all subsequent expressions involving potentials.

The transmembrane potential $V_{e,i}$ at the body of the neuron determines the neuron's output firing rate. An outgoing pulse is triggered whenever $V_{e,i}$ reaches a threshold, so the response probability has the form of a step function. However, as we are considering population behavior and using a continuous variable to describe neuronal output, we take the response function to have a smooth transition from 0 to a maximum $Q_{e,i}^{\max}$, with a width that reflects the variation of threshold levels encountered in real populations of neurons. We could assume the distribution of thresholds to be normal with a mean of $\theta_{e,i}$ and a standard deviation of $\sigma_{e,i}$, so the response function would be an error function; however, it is more convenient and consistent with previous work [1,5–12] to adopt the following relation between $V_{e,i}$ and the output firing rate $Q_{e,i}$:

$$Q_{e,i}(\mathbf{r},t)=\frac{Q_{e,i}^{\max}}{1+\exp[-C(V_{e,i}-\theta_{e,i})/\sigma_{e,i}]}, \quad (6)$$

which closely approximates an error function. The derivative of Eq. (6) with respect to $V_{e,i}$, required below, is

$$\frac{dQ_{e,i}}{dV_{e,i}}\equiv\rho_{e,i}=(C/\sigma_{e,i})Q_{e,i}(1-Q_{e,i}/Q_{e,i}^{\max}), \quad (7)$$

which has a maximum $\rho_{e,i}^{\max}=CQ_{e,i}^{\max}/4\sigma_{e,i}$ at $V_{e,i}=\theta_{e,i}$. We choose $C=\pi/\sqrt{3}\approx 1.81$ so that $dQ_{e,i}/dV_{e,i}$ approximates a Gaussian and has standard deviation $\sigma_{e,i}$.

The last stage in the operation of neurons is the transmission of impulses along output fibers (*axons*) to other synapses. In the continuum approximation this is usually assumed to be isotropic. It has been shown [11] that this propagation of activity through the cortex can be described by the damped wave equations

$$D_{e,i}\phi_{e,i}(\mathbf{r},t)=Q_{e,i}(\mathbf{r},t), \quad (8)$$

$$D_{e,i}=\left[\frac{1}{\gamma_{e,i}^2}\frac{\partial^2}{\partial t^2}+\frac{2}{\gamma_{e,i}}\frac{\partial}{\partial t}+1-r_e^2\nabla^2\right], \quad (9)$$

where v is the axonal conduction velocity, $r_{e,i}$ are the characteristic ranges of axons, and $\gamma_{e,i}=v/r_{e,i}$.

Together, Eqs. (1), (3), (6), and (8) represent a complete set of equations for the dynamics of the variables $P_{e,i}, V_{e,i}, Q_{e,i}$, and $\phi_{e,i}$, of which only Eq. (6) is nonlinear.

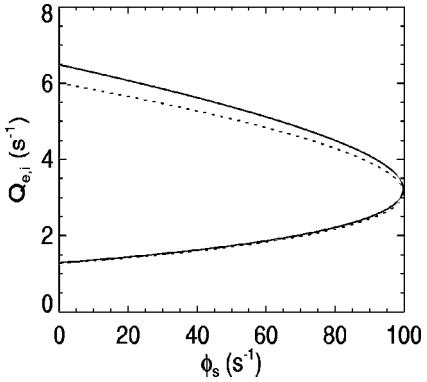

FIG. 1. Steady state solutions for Q_e (solid line) and Q_i (dotted line) for the parameters in Table I and $\phi_s(\mathbf{r},t)=\phi_s=$const. The corresponding results for the other variables are qualitatively similar.

From these, steady state solutions can also be obtained, as can linear dispersion and stability properties [11].

B. Steady state solutions

When all derivatives are set to zero and $\phi_s(\mathbf{r},t)=\phi_s=$const in Eqs. (1), (3), (6), and (8), we find $V_{e,i}=l_{e,i}P_{e,i}$ and $\phi_{e,i}=Q_{e,i}$, giving

$$V_e=l_e(N_{ee}s_eQ_e+N_{ei}s_iQ_i+N_{es}s_s\phi_s), \quad (10)$$

$$V_i=l_i(N_{ie}s_eQ_e+N_{ii}s_iQ_i+N_{is}s_s\phi_s), \quad (11)$$

$$Q_{e,i}=\frac{Q_{e,i}^{\max}}{1+\exp[-C(V_{e,i}-\theta_{e,i})/\sigma_{e,i}]}. \quad (12)$$

Figure 1 shows $Q_{e,i}$ as a function of ϕ_s for a typical case. For given ϕ_s there are two solutions for either Q_e or Q_i with low firing rates and one high firing rate solution with $Q_{e,i}\approx Q_{e,i}^{\max}$ (not shown) [11], but as the control parameter ϕ_s is increased a point is reached where the two low firing rate solutions merge and vanish [11]. The high firing rate solutions are not of concern here as we are only interested here in the linear dynamics of the system at the low firing rates more typically observed in the brain. It was established previously [11] that only the lower of the two low firing rate solutions is stable, so that is the one we will be concerned with below.

C. Basic linear dispersion relation

The linear dispersion relation is a useful starting point for the investigation of the dynamics of our model and its derivation is straightforward. First Eq. (6) is linearized about a fixed point $Q_{e,i}^{(0)}$, giving

$$Q_{e,i}(\mathbf{r},t)=Q_{e,i}^{(0)}+\rho_{e,i}(V_{e,i}(\mathbf{r},t)-V_{e,i}^{(0)}), \quad (13)$$

where $\rho_{e,i}$ is defined by Eq. (7). Fourier transformation of Eqs. (1), (3), (8), and (13) then gives

$$P_q(\mathbf{k},\omega)=N_{qe}s_e\phi_e+N_{qi}s_i\phi_i+N_{qs}s_s\phi_s, \quad (14)$$

$$V_q(\mathbf{k},\omega)=l_qL(\omega)P_q(\mathbf{k},\omega), \quad (15)$$

$$Q_q(\mathbf{k},\omega)=\rho_qV_q(\mathbf{k},\omega), \quad (16)$$

$$D_q\phi_q(\mathbf{k},\omega)=Q_q(\mathbf{k},\omega), \tag{17}$$

with

$$D_q=(1-i\omega/\gamma_q)^2+k^2v^2/\gamma_q^2, \tag{18}$$

$$L(\omega)=[(1-i\omega/\alpha)(1-i\omega/\beta)]^{-1}, \tag{19}$$

and $q=e,i$. Here and subsequently the component at $(\mathbf{k},\omega)=(\mathbf{0},0)$ is excluded, as steady state values are treated separately.

Equations (14)–(17) reduce to

$$(D_e-\rho_e l_e N_{ee}s_e L)\phi_e-\rho_e l_e N_{ei}s_i L\phi_i=\rho_e l_e N_{es}s_s L\phi_s, \tag{20}$$

$$(D_i-\rho_i l_i N_{ii}s_i L)\phi_i-\rho_i l_i N_{ie}s_e L\phi_e=\rho_i l_i N_{is}s_s L\phi_s \tag{21}$$

by elimination of $P_{e,i}$, $V_{e,i}$, and $Q_{e,i}$. The transfer function ϕ_e/ϕ_s is then

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{D_e(D_i-G_{ii}L)-G_{ee}L(D_i-I_{ie}G_{ii}L)}, \tag{22}$$

where we have introduced dimensionless variables

$$G_{ee}=\rho_e l_e N_{ee}s_e, \tag{23}$$

$$G_{ii}=\rho_i l_i N_{ii}s_i, \tag{24}$$

$$G_{es}=\rho_e l_e N_{es}s_s, \tag{25}$$

$$I_{ie}=1-N_{ei}N_{ie}/N_{ee}N_{ii}, \tag{26}$$

$$I_{is}=1-N_{ei}N_{is}/N_{es}N_{ii} \tag{27}$$

to characterize components of gain and the asymmetry between connectivities of excitatory and inhibitory neurons.

The dispersion relation is obtained by setting the denominator of the transfer function to zero, giving

$$D_iD_e-G_{ii}LD_e-G_{ee}LD_i+I_{ie}G_{ee}G_{ii}L^2=0. \tag{28}$$

Equation (28) is a rational function of ω and although numerical solution is straightforward, some approximations are possible that aid in the analytical interpretation of dispersion relations. One relates to a constraint on connectivities: We can consider that there are fractional densities $f_p(p=e,i,s)$ associated each of the three possible types of axonal terminals in the cortex, which depend both on the density of neurons of each type and on the average number of synapses on the axons of each. We then assume that connections made with dendrites are random, in the sense that $N_{qp}=N_q^{\mathrm{tot}}f_p$ with $p=e,i,s$ and $q=e,i$, where $N_{e,i}^{\mathrm{tot}}$ are the mean total numbers of dendritic synapses on excitatory and inhibitory neurons. This definition of N_{qp} with Eqs. (26) and (27) implies that I_{ie} and I_{is} are identically zero.

Another useful approximation relates to the term D_i. Since $r_i \ll r_e$ in humans, $\gamma_i \gg \gamma_e$ so $D_i(\mathbf{k},\omega)\approx 1$ whenever $\omega, kv \ll \gamma_i$ [11]. This is termed the *local inhibition approximation*.

When both random connectivity and the local inhibition approximation are assumed, the transfer function reduces to

$$\phi_e/\phi_s=G_{es}/[D_e(L^{-1}-G_{ii})-G_{ee}]. \tag{29}$$

This can be further simplified when $G_{ii}\approx 0$ to give

$$\phi_e/\phi_s=G_{es}/[L^{-1}(\omega)D_e(\mathbf{k},\omega)-G_{ee}]. \tag{30}$$

The above approximations will be used as appropriate in later sections, where we present dispersion relations for various forms of feedback and emphasize the effects of feedback by comparison with the case where there is no feedback. To that end we next outline the frequency domain characteristics of the model without feedback.

D. Properties of basic linear dispersion relation

The dispersion relation corresponding to Eq. (30) is [11]

$$\left(1-i\frac{\omega}{\alpha}\right)\left(1-i\frac{\omega}{\beta}\right)\left[\left(1-i\frac{\omega}{\gamma_e}\right)^2+(kr_e)^2\right]-G_{ee}=0, \tag{31}$$

in which the linear dynamics is determined only by excitatory neurons. Solutions to Eq. (31) have a simple form when $\alpha=\beta$ and $k=0$, with

$$\omega=\begin{cases}-i\left(\dfrac{\alpha+\gamma_e}{2}\right)\pm i\sqrt{\alpha\gamma_e\sqrt{G_{ee}}+\left(\dfrac{\alpha-\gamma_e}{2}\right)^2}\\[2ex] -i\left(\dfrac{\alpha+\gamma_e}{2}\right)\pm\sqrt{\alpha\gamma_e\sqrt{G_{ee}}-\left(\dfrac{\alpha-\gamma_e}{2}\right)^2}.\end{cases} \tag{32}$$

Note that (i) the first pair of solutions predicts one purely imaginary root that can be unstable, yielding the stability criterion $(\alpha+\gamma_e/2)>\sqrt{\alpha\gamma_e\sqrt{G_{ee}}+[(\alpha-\gamma_e)/2]^2}$ (or, more simply, $G_{ee}<1$), (ii) the other member of the first pair has a value ranging from $\omega=-i\alpha$ for $G_{ee}=0$ to $\omega=-i(\alpha+\gamma_e)/2$ for $G_{ee}=1$, and (iii) the second pair yields a condition $\alpha\gamma_e\sqrt{G_{ee}}>[(\alpha-\gamma_e)/2]^2$ for there to be oscillatory roots. Equation (31) is easily solvable for $kr_e \gg G_{ee}$, in which case G_{ee} can be neglected and the solutions are damped nondispersive traveling waves with $\omega=\pm kv-i\gamma_e$ and two purely damped roots at $\omega=-i\alpha$ and $-i\beta$.

In conclusion, the fourth-order approximation (31) to the full dispersion relation has either two or four purely damped roots, one of which may be unstable; if there are oscillatory roots they have a damping rate $\sim\gamma_e$. These general characteristics also apply to the full eighth-order dispersion relation (28), as is illustrated in Fig. 2, and will be contrasted in Sec. IV with those that occur when the model is augmented with feedback.

III. FEEDBACK

There are numerous metabolic and biophysical processes whose effect is to modulate neuronal responsiveness, dependent on current or recent levels of activity. Each is considered here as a form of feedback, whereby some measure of neuronal activity modulates parameters of the model that had previously been considered constant. We aim to represent feedback in a sufficiently general way that our results can be

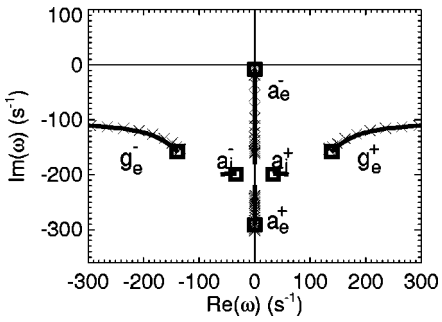

FIG. 2. Solutions of the dispersion relation (28) with the parameters as in Table I and $\phi_s=90\ \mathrm{s}^{-1}$ for $0\leqslant kr_e\leqslant 5$. For each mode, $kr_e=0$ is marked with a square. Two sets of roots are shown. One set (solid lines) consists of solutions to the eighth-order dispersion relation (28), of which six roots are shown. An additional pair of roots with $\omega\approx\pm kv-i\gamma_i$ is not shown since $\gamma_i\approx 10^5\ \mathrm{s}^{-1}$. Superimposed on these roots are the solutions (shown with crosses) of the fourth-order dispersion relation derived from Eq. (30). In this figure and subsequently we use the labels a and g to suggest the asymptotic damping rates $-i\alpha$ or $-i\gamma_{e,i}$. The subscripts e and i indicate whether the root concerned is most sensitive to excitatory or inhibitory parameters. Roots occur in pairs, so each member is arbitrarily assigned a superscript $+$ or $-$. The range of the abscissa is $\pm 300\ \mathrm{s}^{-1}$ or ± 48 Hz, to match that of EEGs.

applied to a wide variety of specific mechanisms. The simplest such formulation is

$$x(\mathbf{r},t)=x^{(0)}+x^{(1)}H(t)\otimes[\chi(\mathbf{r},t)-\chi^{(0)}], \tag{33}$$

where x represents a parameter of the model, χ is one of the model variables, $x^{(0)}$ and $\chi^{(0)}$ are steady state values, $x^{(1)}$ is a constant describing the strength of feedback, and $H(t)$ is a function that describes the temporal form of the feedback, with $\int_{-\infty}^{\infty}H(t)dt=1$ and $H(t)=0$ for $t<0$ to enforce causality. Some assumptions are implied by Eq. (33): (i) the perturbations are small enough that a linear equation is adequate, (ii) modulation is local in space, and (iii) $x^{(1)}$ and $H(t)$ do not vary with position or time. These assumptions may need to be revised in the future, but in the absence of compelling reasons to do otherwise we consider only modulations of the form (33).

Classes of feedback

When x represents one of the parameters listed in Table I and χ one of $P_{e,i}$, $Q_{e,i}$, $V_{e,i}$, or $\phi_{e,i}$, one can substitute Eq.(33) into Eqs. (14)–(17) to derive a linear dispersion relation. A potential difficulty is that there are many combinations of parameter and variable. However, it is not necessary to consider every one: For both mathematical and physiological reasons the number of cases needing to be examined is actually far less, as we show next.

Concerning which parameters may be affected by feedback, the majority are either inherently constant (such as N_{ee} and N_{ei}) or effectively constant in the normal brain (such as $\alpha, Q_{e,i}^{\max}$, and v). The only parameters that are certainly modulated are $s_{e,i,s}$ (due to biophysical and biochemical effects [17]) and $\theta_{e,i}$ (e.g., due to long-acting neurotransmitters and reduced excitability immediately following firing [18]). Of these, s_s is associated with the subcortical driving signal and so does not appear in dispersion relations. Also, s_i and θ_i do not need to be considered explicitly due to the symmetry between excitatory and inhibitory quantities. Consequently, only modulations of s_e and θ_e need to be investigated in what follows.

As to which variables drive modulations, there are again mathematical and physiological reasons for limiting the possibilities. First, Eqs. (14)–(17) are similar with respect to excitatory and inhibitory neurons. This means that we need to derive dispersion relations for driving variables belonging to only one class of neuron, as the others can be obtained by symmetry. This means that feedback mediated by P_i, Q_i, V_i, and ϕ_i need not be explicitly considered here. Second, P_e is an intermediate construct that is not directly identifiable with any physiological quantity, so it is not an appropriate driving variable. Third, Eq. (16) shows that small-amplitude perturbations of Q_e and V_e are proportional to each other. For this reason we do not consider Q_e in what follows. That leaves ϕ_e and V_e as the driving variables to be considered below.

The final matter is to decide the form of $H(t)$ in Eq. (33). For $n=1,2$ we use $H(t)=\eta^n t^{n-1}\exp(-\eta t)$ when $t\geqslant 0$ and zero otherwise. The rate constant η characterizing feedback is assumed positive. This form of $H(t)$ implies a simple differential equivalent of Eq. (33):

$$\left(\frac{1}{\eta}\frac{d}{dt}+1\right)^n[x(\mathbf{r},t)-x^{(0)}]=x^{(1)}[\chi(\mathbf{r},t)-\chi^{(0)}]. \tag{34}$$

Substituting s_e and θ_e for x, and ϕ_e and V_e for χ in Eq. (33) gives

$$s_e(\mathbf{r},t)=s_e^{(0)}+s_e^{(1)}H(t)\otimes[\phi_e(\mathbf{r},t)-\phi_e^{(0)}], \tag{35a}$$

$$s_e(\mathbf{r},t)=s_e^{(0)}+s_e^{(1)}H(t)\otimes[V_e(\mathbf{r},t)-V_e^{(0)}], \tag{35b}$$

$$\theta_e(\mathbf{r},t)=\theta_e^{(0)}+\theta_e^{(1)}H(t)\otimes[\phi_e(\mathbf{r},t)-\phi_e^{(0)}], \tag{35c}$$

$$\theta_e(\mathbf{r},t)=\theta_e^{(0)}+\theta_e^{(1)}H(t)\otimes[V_e(\mathbf{r},t)-V_e^{(0)}] \tag{35d}$$

as the four cases to be considered, which we refer to as types $A-D$, respectively. The reference values $\phi_e^{(0)}$ and $V_e^{(0)}$ are always chosen to be steady state values of the corresponding variables, which is not strictly necessary but has the practical attraction that $s_e^{(0)}$ and $\theta_e^{(0)}$ have the nominal values in Table I.

IV. DISPERSION RELATIONS WITH FEEDBACK

Here we examine the general characteristics of the four classes of feedback through the corresponding dispersion relations and contrast them with the feedback-free results of Sec. II. The transfer functions resulting from Eqs. (35a)–(35d) are

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{D_e(D_i-G_{ii}L)-G_{ee}L(D_i-I_{ie}G_{ii}L)F_A}, \tag{36a}$$

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{D_e(D_i-G_{ii}L)F_B-G_{ee}L(D_i-I_{ie}G_{ii}L)}, \tag{36b}$$

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{(D_e-F_C)(D_i-G_{ii}L)-G_{ee}L(D_i-I_{ie}G_{ii}L)}, \tag{36c}$$

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{D_e(D_i-G_{ii}L)/F_D-G_{ee}L(D_i-I_{ie}G_{ii}L)}, \tag{36d}$$

respectively, where $D_{e,i}(\mathbf{k},\omega)$ and $L(\omega)$ are given by Eqs. (18) and (19). [The Appendix contains a derivation of Eq. (36a) and the other transfer functions can be derived similarly.] The present transfer functions differ from the feedback-free case (22) only in the factors F_A-F_D,

$$F_A(\omega)=1+h_AH(\omega), \tag{37a}$$

$$F_B(\omega)=1-h_BH(\omega)L(\omega), \tag{37b}$$

$$F_C(\omega)=h_CH(\omega), \tag{37c}$$

$$F_D(\omega)=1+h_DH(\omega), \tag{37d}$$

where

$$h_A=(s_e^{(1)}/s_e^{(0)})\phi_e^{(0)}, \tag{38a}$$

$$h_B=l_eN_{ee}s_e^{(1)}\phi_e^{(0)}, \tag{38b}$$

$$h_C=-\rho_e\theta_e^{(1)}, \tag{38c}$$

$$h_D=-\theta_e^{(1)} \tag{38d}$$

are the dimensionless feedback strengths. The quantities $s_e^{(1)}$ and $\theta_e^{(1)}$, introduced in Eqs. (35), can be evaluated when specific physiological feedback mechanisms are chosen. However, in the present context, which aims to be independent of such choices, it is more convenient to present results in terms of h_A-h_D. These values will be referred to generically as h_M in situations where no confusion should result and will be considered to be free parameters of either sign. The definitions of h_A-h_D have been chosen so that $h_M>0$ corresponds to positive feedback and $h_M<0$ corresponds to negative feedback. The remaining feedback quantity is $H(\omega)$: According to the choice for $H(t)$ made in Sec. III,

$$H(\omega)=(1-i\omega/\eta)^{-n}. \tag{39}$$

.

For $h_M=0$, Eqs. (36a)–(36d) each reduces to Eq. (22), but for $h_M\neq 0$, Eqs. (36a)–(36d) are less convenient for analytical investigation. However, the simplification $G_{ii}=0$ has been found to result in adequate approximations to the exact solutions for parameters in physiologically reasonable ranges and gives

$$\phi_e/\phi_s=G_{es}H^{-1}/[H^{-1}(L^{-1}D_e-G_{ee})-h_AG_{ee}], \tag{40a}$$

$$\phi_e/\phi_s=G_{es}H^{-1}/[H^{-1}(L^{-1}D_e-G_{ee})-h_BD_e], \tag{40b}$$

$$\phi_e/\phi_s=G_{es}H^{-1}/[H^{-1}(L^{-1}D_e-G_{ee})-h_CL^{-1}], \tag{40c}$$

$$\phi_e/\phi_s=\frac{G_{es}(H^{-1}+h_D)}{[H^{-1}(L^{-1}D_e-G_{ee})-h_DG_{ee}]}, \tag{40d}$$

where $H^{-1}=(1-i\omega/\eta)^n$, $L^{-1}=(1-i\omega/\alpha)^2$, and $D_e=(1-i\omega/\gamma_e)^2+k^2r_e^2$. A further simplification has been made here and henceforth by setting $\alpha=\beta$, which is consistent with physiology [16]. In order to understand the case $h_M\neq 0$, we consider separately two extremes of η: $\eta\gg\alpha,\gamma_e$ and $0\lesssim\eta\ll\alpha,\gamma_e$.

A. Fast feedback: $\eta\gg\alpha,\gamma_e$

In examining fast feedback, it is convenient to examine types *A* and *D* first and then types *B* and *C*.

1. Types A and D feedback

The transfer functions (40a) and (40d) both imply normal modes given by the roots of

$$H^{-1}(L^{-1}D_e-G_{ee})-h_AG_{ee}=0, \tag{41}$$

so the two cases can be dealt with as one. If we substitute Eqs. (18), (19), and (39) into Eq. (41) we find

$$(1-i\omega/\eta)^n\{(1-i\omega/\alpha)^2[(1-i\omega/\gamma_e)^2+k^2r_e^2]-G_{ee}\}-h_AG_{ee}=0. \tag{42}$$

This is easily solved in the trivial case $G_{ee}=0$, resulting in roots at $-i\eta,-i\alpha$, and $\pm kv-i\gamma_e$. However, in practice, G_{ee} may be ~ 1, which is of the same order as all other terms in Eq. (42), so the effect of G_{ee} and h_A must be considered in more detail.

One can show that the roots near $\omega=-i\eta$ are only weakly affected by G_{ee} and h_A by substituting $\omega=-i\eta+\epsilon$ into Eq. (42) and retaining only the lowest order of ϵ/η. We find $\epsilon/\eta\approx i(h_AG_{ee}\alpha^2\gamma_e^2/\eta^4)^{1/n}$ and for $\eta\gg\alpha,\gamma_e$ these are rapidly damped modes of little relevance to EEGs. The remaining roots all have $|\omega|\ll\eta$ so the term $H^{-1}=(1-\omega/\eta)^n\approx 1$ and a much simplified dispersion relation results. Then, in the limit $k\to\infty$, the solutions are simply $\omega=\pm kv-i\gamma_e,-i\alpha$ (as in the feedback-free case) and when $k=0$ the solutions are

$$\omega=-i\left(\frac{\alpha+\gamma_e}{2}\right)\pm\sqrt{\pm\alpha\gamma_e\sqrt{(1+h_A)G_{ee}}-\left(\frac{\alpha-\gamma_e}{2}\right)^2}, \tag{43}$$

where all four combinations of signs are implied. Only the latter limit depends on h_A. While $h_A\geqslant -1$ results in four solutions that are similar to those described in Sec. II D in being of the form $-i(\alpha+\gamma_e)/2\pm$(purely real values) or $-i(\alpha+\gamma_e)/2\pm$(purely imaginary values), the alternative $h_A<-1$ gives qualitatively different solutions of the form $-i(\alpha+\gamma_e)/2\pm\sqrt{\text{complex values}}$ and $-i(\alpha+\gamma_e)/2\pm\sqrt{(\text{complex values})^*}$. This has an important consequence for the modeling of electrocortical activity: Purely damped modes of the basic model are transformed into modes that are guaranteed to be oscillatory and that may be lightly damped.

Figure 3 shows solutions of the type *A* dispersion relation for several values of h_A and a range of *k*. Solutions of the *full*

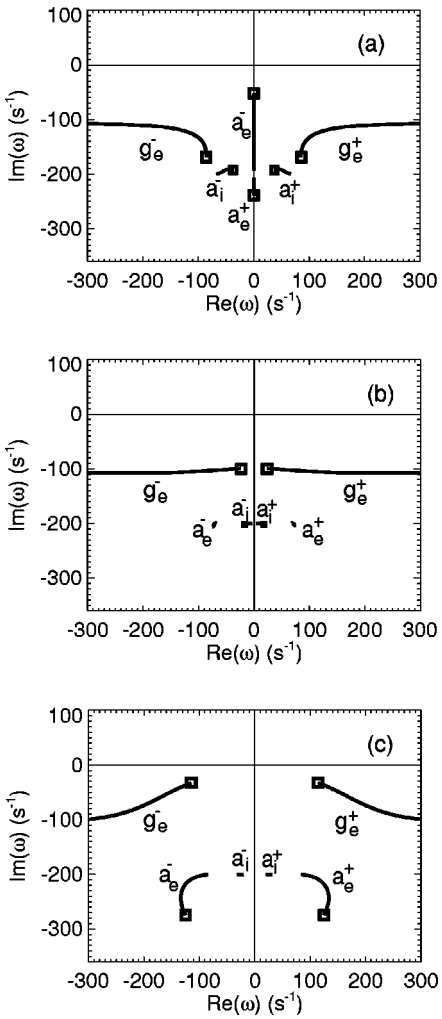

FIG. 3. Effect of fast ($\eta = 5000\ s^{-1}$), first order type *A* feedback. The roots are solutions to the full dispersion relation obtained from Eq. (36a). The parameter values are as in Table I and $\phi_s = 90\ s^{-1}$, for $0 \leqslant k r_e \leqslant 5$; $k=0$ is indicated by a square unless there is little dependence on *k*. One root with $\mathrm{Im}(\omega) \sim -\eta$ and a pair of roots with $\mathrm{Im}(\omega) \sim -\gamma_i$ are not shown as they are too rapidly damped to be of relevance. (a) $h_A = -0.8$, (b) $h_A = -1$, and (c) $h_A = -3$.

dispersion relation are shown, rather than those of the approximation (42), to demonstrate that conclusions based on the approximate form also apply to the exact dispersion relation. Figure 3(a), in which feedback is weak, has the same general appearance as Fig. 2, while Figs. 3(b) and 3(c) show solutions for stronger feedback. As suggested by Eq. (43), the latter are characterized by the absence of purely damped roots. Of particular interest is the progressively increasing damping of the nearly unstable root labeled a_e^- as h_A becomes more negative and the simultaneous destabilization of the roots labeled $g_e^\pm$. The latter roots, identified as propagating wave modes by their asymptotically constant group velocities, eventually become unstable when they enter the upper half of the complex plane.

The feedback parameters η and *n* have only a weak effect on solutions. The reason for this is most evident from Eq. (42): For $\omega \ll \eta$ the term $(1 - i\omega/\eta)^n \approx 1$ whatever the values of η and *n*. In physical terms, the high rate of feedback means that s_e is modulated proportionately to, and in phase with ϕ_e, altering the total gain from G_{ee} to $(1+h_A)G_{ee}$ and all modal changes flow from this effective alteration of the gain.

2. Types B and C feedback

The approximate dispersion relations for fast feedback of types B and C are obtained from the denominators of Eqs. (36b) and (36c) if we set $I_{ie}=0$, $G_{ii}=0$, $D_i=1$, and $H^{-1}=1$ as before. This gives, respectively,

$$[(1 - i\omega/\alpha)^2 - h_B][(1 - i\omega/\gamma_e)^2 + k^2 r_e^2] - G_{ee} = 0, \tag{44}$$

$$(1 - i\omega/\alpha)^2[(1 - i\omega/\gamma_e)^2 + k^2 r_e^2 - h_C] - G_{ee} = 0. \tag{45}$$

Although Eqs. (44) and (45) are similar to Eq. (42) in being independent of η and *n*, they have different dependences on h_M. For $h_M = 0$, solutions are as for the basic model without feedback, but as $|h_M|$ increases, G_{ee} becomes negligible and the equations simplify accordingly. The solutions of Eq. (44) are then $\omega = -i\alpha(1 \pm \sqrt{h_B})$ and $\omega = \pm kv - i\gamma_e$. Similarly, the solutions of Eq. (45) approach $\omega = -i\alpha$ and $\omega = -i\gamma_e \pm \gamma_e \sqrt{k^2 r_e^2 - h_C}$. They are marked $a_e^\pm$ and $g_e^\pm$, respectively, in Fig. 4. The exact dispersion relations also have a pair of roots $a_i^\pm$ near $\omega = -i\alpha$, a pair $g_i^\pm$ that is symmetrically placed around $-i\gamma_i$, and one or two roots (depending on *n*) near $-i\eta$. All but the first pair are too heavily damped to appear in Fig. 4 and are not of relevance to EEG for the same reason.

Figure 4(a) and 4(b) shows roots of the dispersion relations for feedback of types *B* and *C*. In these examples, $|h_M| > G_{ee}$ so that h_M has a particularly strong influence on the roots labeled $a_{e,i}^\pm$ in Fig. 4(a) and on the roots labeled $g_e^\pm$ in Fig. 4(b). In both cases, varying h_M moves the affected roots laterally while not greatly affecting their imaginary parts. The rate constants α and γ_e are the other determinants of the root locations. Figure 4(c) illustrates this point by showing the roots for the same system as in Fig. 4(a) but with α reduced from 200 s^{-1} to 50 s^{-1} in excitatory neurons, thereby scaling the roots $a_e^\pm$ by the same factor: Roots at real frequencies of $\pm 350 s^{-1}$ (± 55 Hz) move to $\pm 90\ s^{-1}$ (± 14 Hz). The roots labeled $g_e^\pm$ can likewise be scaled though γ_e.

B. Slow feedback: $\eta \ll \alpha, \gamma_e$

Here we are mainly concerned with roots lying near the origin, as the remaining roots prove to be scarcely affected by slow feedback. The reason for this is that $|\omega| \sim \alpha$ and $\gamma_e \gg \eta$ for all roots far from the origin, so the magnitude of $H^{-1} = (1 - i\omega/\eta)^n \sim (-i\omega/\eta)^n$ is large compared to the final terms in the denominators of Eqs. (40a)–(40d). Thus the final terms can be dropped and the dispersion relations for the four types of feedback become identical, with $(-i\omega/\eta)^n (L^{-1} D_e - G_{ee}) = 0$, which leads to the same modes as in the feedback-free case (31).

Of the roots near the origin, one is expected from the feedback-free case (the root labeled a_e^- in Fig. 2) and *n* others occur as a result of feedback ($n = 1,2$). The location of each is simply inferred only in the limit $h_M \to 0$. In this case the last term in the denominator of Eqs. (40a)–(40d)

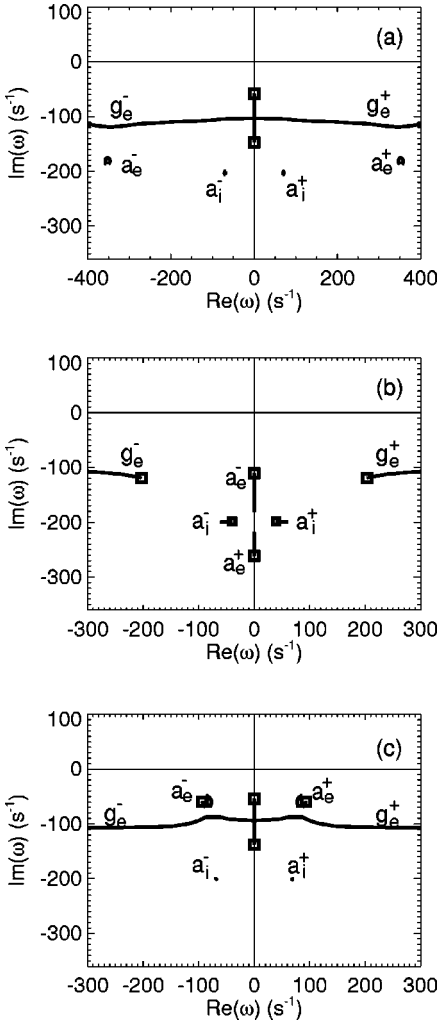

FIG. 4. Fast ($\eta=5000\ \mathrm{s}^{-1}$), first-order feedback of types *B* and *C*. The roots below are solutions to the full dispersion relations obtained from Eqs. (36b) and (36c). The parameter values are as in Table I, $\phi_s=90\ \mathrm{s}^{-1}$, $h_B=h_C=-3$, and $0\leqslant kr_e\leqslant 5$. Squares indicate $k=0$ unless the mode has little dependence on *k*. (a) Type *B* feedback, in which two roots labeled $g_e^{\pm}$ first converge as *k* increases and then diverge. (b) Type *C* feedback. (c) Type *B* feedback as in (a), but with $\alpha=\beta=50\ \mathrm{s}^{-1}$ in excitatory neurons.

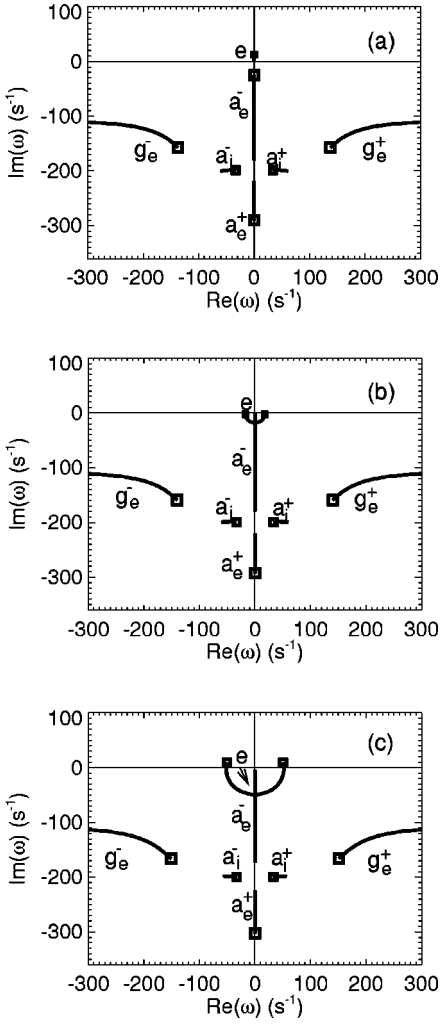

FIG. 5. Effect of slow ($\eta=1\ \mathrm{s}^{-1}$), first-order feedback of type *A* or *D*. The values of other parameters are as in Table I and $\phi_s=90\ \mathrm{s}^{-1}$. The roots below are solutions of the dispersion relation implied by Eq. (36a). Solutions are shown for the range $0\leqslant kr_e\leqslant 5$. In those cases where there are lightly damped oscillatory modes, these merge and become purely damped modes as *k* increases and approach $-i\eta$ and $-i\alpha$ in the limit $k\to\infty$. (a) $h_M=10$, (b) $h_M=-10$, and (c) $h_M=-100$.

vanishes, giving $H^{-1}(L^{-1}D_e-G_{ee})=0$ as the dispersion relation. Then the factor $H^{-1}=0$ implies a root with $\omega=-i\eta$. (If $n=2$ it will be a double root.)

Turning now to $h_M\neq 0$, feedbacks of types *A* and *D* will be considered first and then the results for types *B* and *C* feedback will be summarized.

1. Types A and D feedback

By assuming $H^{-1}=1-i\omega/\eta$ and setting $L^{-1}=(1-i\omega/\alpha)^2$, as in Sec. IV A, the dispersion relation implied by Eq. (40a) becomes

$$(1-i\omega/\eta)[(1-i\omega/\alpha)^2D_e-G_{ee}]-h_MG_{ee}=0, \quad (46)$$

where $D_e=(1-i\omega/\gamma_e)^2+k^2r_e^2$ and $h_M=h_A$ or h_D in the present context. Since $|\omega|\ll\alpha,\gamma_e$, Eqs. (46) becomes

$$0=1+k^2r_e^2-(1+h_M)G_{ee}-i\left[1+k^2r_e^2-G_{ee}+(1+k^2r_e^2)\times\frac{2\eta}{\alpha}+\frac{2\eta}{\gamma_e}\right]\frac{\omega}{\eta}-\left[(1+k^2r_e^2)\frac{2\eta}{\alpha}+\frac{2\eta}{\gamma_e}\right]\frac{\omega^2}{\eta^2}, \quad (47)$$

after discarding terms that are second order or higher in ω/α, ω/γ_e, η/α, and η/γ_e. Figure 5 demonstrates that as kr_e increases, a pair of oscillatory roots approach each other along opposite arcs, merge, and then separate as purely damped roots, the one labeled *e* tending asymptotically to $-i\eta$ and the other labeled a_e^- tending to $-i\alpha$. The latter is similar to the root of the same name in previous figures, while the locus of roots labeled *e* and the unlabeled arcs arise only because of feedback.

The case $n=2$ is more difficult to deal with analytically; however, numerical results show similar behavior to the $n=1$ case, namely, oscillatory roots when h_M is sufficiently

negative and purely damped roots otherwise. The two roots arising from the feedback when $n=2$ tend to cluster closer to the origin than when $n=1$, but otherwise the results are similar.

2. Types B and C feedback

Starting with the transfer functions (40b) and (40c), we obtain the dispersion relations $H^{-1}(L^{-1}D_e-G_{ee})-h_BD_e=0$ and $H^{-1}(L^{-1}D_e-G_{ee})-h_CL^{-1}=0$ for types B and C feedback, respectively. These are similar to the case of types A and D feedback, $H^{-1}(L^{-1}D_e-G_{ee})-h_AG_{ee}=0$, both when $\omega\sim\eta$ (because $D_e\approx L^{-1}\approx 1\sim G_{ee}$) and when $\omega\gg\eta$ (because h_AG_{ee}, h_BD_e, and h_CL^{-1} are negligible). As a result, roots of the dispersion relations are similar to those found for types A and D feedback and Fig. 5 gives an adequate illustration of most aspects of the dispersion for types B and C feedback.

V. SUMMARY

Feedback is likely to be a requirement of models that attempt to model EEGs. In this paper we investigate the role of local feedback by generalizing our previous continuum model of cortical electrical activity [11].

In Sec. II we derive transfer functions that are expressed in terms of basic physiological quantities, such as firing threshold, synaptic response sizes, firing rates, and membrane potentials. This allows local feedback to be classified into just four fundamental classes identified in Sec. III. These four classes describe linear modulation of synaptic strengths and firing thresholds by incoming firing rate or transmembrane potential. It is argued that other possibilities either are not indicated physiologically or are redundant mathematically in the context of our model. The four feedback classes are thus fundamental.

Section IV examines the properties of the four transfer functions to investigate the effects of these feedbacks on the system's characteristics. Several results emerge: Classes A and D of feedback have identical dispersion relations, feedback with a rate constant η introduces roots in the vicinity of $-i\eta$, and as $k\to\infty$ other modes tend to values $\omega=\pm kv-i\gamma_e,-i\alpha$, which are independent of feedback and determined instead by α the dendritic damping rate and γ_e the damping rate of long-range propagation.

In view of the possible significance of feedback-induced modes to the generation or modulation of EEGs, we also looked for modes that were lightly damped. They were found to occur in three situations.

(i) Fast feedback of types A and D (incoming firing rate modulating synaptic efficacy and outgoing firing rate modulating thresholds) caused existing oscillatory roots to become lightly damped (see Fig. 3). A condition for such lightly damped modes to exist is that the strength of feedback is sufficiently *negative*. This could occur if an increase in incoming firing rate causes a decrease in the synaptic efficacy or if an increase in outgoing firing rate causes an increase in firing threshold.

(ii) When there is fast negative feedback of type B and the dendritic rate constant is less than the damping rate of propagating waves, a lightly damped mode arises whose frequency increases with the strength of negative feedback and has little dependence on wave number [see Fig. 4(c)].

(iii) With slow feedback of any type $A-D$ there are additional modes, all of which are lightly damped. Again, negative feedback is a condition that they be oscillatory and the frequency increases with the strength of negative feedback (see Fig. 5).

These three kinds of lightly damped modes are distinguished by their dispersion characteristics: As k increases, ω increases in the first kind of resonance, remains almost constant in the second, and decreases in the third.

All the above results are based in the presumption made in Eqs. (35) that feedback affects either s_e or θ_e; however, the same methods can be applied to infer the effects of modulation of inhibitory parameters. Results are presented in terms of dimensionless feedback strengths h_M; however, Eqs. (38) provide the means to derive numerical results for particular physiological processes.

Having identified and characterized several forms of feedback that are potentially significant in the large scale activity of the cortex, it is now possible to combine the expressions presented here with the rate constants and strengths of specific physiological mechanisms and thereby obtain physiologically based transfer functions and dispersion relations. Then numerical estimates for the frequencies of the most lightly damped modes will follow and identifications may be possible with characteristic EEG rhythms, such as those near 10 Hz (known as α), 15–30 Hz (β), and 40 Hz (γ). It may also be possible to obtain experimental evidence for frequency–wave-number relations resembling those in the present paper. In any case, the present work helps to make a connection between macroscopic quantities and cellular level quantities, which can guide the development of more accurate models of EEGs, and eventually lead to more thorough and clinically relevant analysis of EEGs.

ACKNOWLEDGMENT

This work was supported by the Ross Trust, Melbourne.

APPENDIX: MODULATION OF s_e BY ϕ_e

The transfer equation for type A feedback is obtained from Eqs. (1), (15)–(17), and (35a), as shown below. Transfer equations for cases $B-D$ can be derived similarly.

For linear modulation of s_e by $\phi_e(\mathbf{r},t)$ we begin by assuming, in accordance with Eq. (35a), that

$$s_e(\mathbf{r},t)\approx s_e^{(0)}+s_e^{(1)}H(t)\otimes[\phi_e(\mathbf{r},t)-\phi_e^{(0)}], \tag{A1}$$

so that Eq. (1a) becomes

$$P_e(\mathbf{r},t)\approx N_{ee}s_e^{(0)}\phi_e(\mathbf{r},t)+N_{ee}s_e^{(1)}\phi_e^{(0)}H(t)\otimes[\phi_e(\mathbf{r},t)-\phi_e^{(0)}]+N_{ei}s_i\phi_i(\mathbf{r},t)+N_{es}s_s\phi_s(\mathbf{r},t) \tag{A2}$$

to first order in deviations of the variables from their steady state values. The Fourier transform of Eq. (A2) is

$$P_e(\mathbf{k},\omega)=N_{ee}[s_e^{(0)}+s_e^{(1)}\phi_e^{(0)}H(\omega)]\phi_e(\mathbf{k},\omega)+N_{ei}s_i\phi_i(\mathbf{k},\omega)+N_{es}s_s\phi_s(\mathbf{k},\omega), \quad \text{(A3)}$$

and similarly for $P_i(\mathbf{k},\omega)$,

$$P_i(\mathbf{k},\omega)=N_{ie}[s_e^{(0)}+s_e^{(1)}\phi_e^{(0)}H(\omega)]\phi_e(\mathbf{k},\omega)+N_{ii}s_i\phi_i(\mathbf{k},\omega)+N_{is}s_s\phi_s(\mathbf{k},\omega). \quad \text{(A4)}$$

Equations (A3), (A4), and (15)–(17) can then be used to derive a transfer function. As the only effect of modulation of s_e by ϕ_e is to multiply s_e by factor $1+(s_e^{(1)}/s_e^{(0)})\phi_e^{(0)}H(\omega)$ in (14), we immediately see from Eq. (22) that the transfer function is

$$\frac{\phi_e}{\phi_s}=\frac{G_{es}L(D_i-I_{is}G_{ii}L)}{D_e(D_i-G_{ii}L)-G_{ee}L(D_i-I_{ie}G_{ii}L)F_A}, \quad \text{(A5)}$$

where we define the feedback factor F_A by

$$F_A(\omega)=1+(s_e^{(1)}/s_e^{(0)})\phi_e^{(0)}H(\omega). \quad \text{(A6)}$$

[1] H. R. Wilson and J. D. Cowan, Kybernetik **13**, 55 (1973).
[2] P. L. Nunez, Math. Biosci. **21**, 279 (1974).
[3] A. van Rotterdam, F. H. Lopes da Silva, J. van den Ende, M. A. Viergever, and A. J. Hermans, Bull. Math. Biol. **44**, 283 (1982).
[4] P. L. Nunez, in *Neocortical Dynamics and Human EEG Rhythms*, edited by P. L. Nunez (Oxford University Press, Oxford, 1995), Chaps. 2, 9, and 11.
[5] B. H. Jansen and V. G. Rit, Biol. Cybern. **73**, 357 (1995).
[6] J. J. Wright and D. T. J. Liley, Behav. Brain Sci. **19**, 285 (1996).
[7] J. J. Wright, Biol. Cybern. **76**, 181 (1997).
[8] J. J. Wright, Biol. Cybern. (to be published).
[9] V. K. Jirsa and H. Haken, Phys. Rev. Lett. **77**, 960 (1996).
[10] V. K. Jirsa and H. Haken, Physica D **99**, 503 (1997).
[11] P. A. Robinson, C. J. Rennie, and J. J. Wright, Phys. Rev. E **56**, 826 (1997).
[12] P. A. Robinson, J. J. Wright, and C. J. Rennie, Phys. Rev. E **57**, 4578 (1998).
[13] P. A. Robinson, C. J. Rennie, and J. J. Wright, Phys. Rev. E **58**, 3557 (1998).
[14] M. Steriade, P. Gloor, R. R. Llinás, F. H. Lopes da Silva, and M.-M. Mesulam, Electroencephalogr. Clin. Neurophysiol. **76**, 481 (1990).
[15] I. Segev, in *The Handbook of Brain Theory and Neural Networks*, edited by M. A. Arbib (MIT Press, Cambridge, MA, 1995).
[16] W. J. Freeman, in *Induced Rhythms of the Brain*, edited by E. Başar and T.H. Bullock (Birkhauser, Basel, 1991).
[17] K. L. Magleby, in *Synaptic Function*, edited by G. M. Edelman, W. E. Gall, and W. M. Cowan (Wiley, New York, 1987).
[18] J. Koester, in *Principles of Neural Science*, 3rd ed., edited by E. R. Kandel, J. H. Schwartz, and T. M. Jessell (Prentice-Hall, Englewood Cliffs, NJ, 1991).