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Analysis of quantal synaptic noise in neural networks using iterated function systems

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It is shown how the dynamics of a discrete-time leaky-integrator (time-summating) neural network with quantal synaptic noise may be formulated in terms of a random iterated function system. Conditions are derived for which the limiting behavior of the system is described by an invariant probability measure on the space of membrane potentials. Such an invariant measure typically has a fractal-like structure, which is illustrated by a simple example of a single neuron with inhibitory feedback. The effects of synaptic noise on the response characteristics of the neuron are also considered. Finally, learning in networks with synaptic noise is discussed.

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I. INTRODUCTION

In a previous paper [1] we analyzed the stochastic dynamics of a *time-summating* binary neural network with additive white noise, based upon a discrete-time version of a leaky-integrator shunting network [2]. In such networks the neuron is taken to be a binary threshold element, which is either on or off depending on whether or not its membrane potential (local field) exceeds threshold, and the membrane potential is a slowly decaying function of time with a decay rate of γ, $\gamma < 1$. Thus, each neuron maintains an activity trace of all previous inputs to that neuron. If shunting effects are ignored (as assumed here), then this trace is a linear function of inputs. Time-summating networks are of interest for a number of reasons: (i) They incorporate certain temporal aspects of the process by which real neurons integrate their inputs [2], (ii) they exhibit complex dynamics at both the single-neuron [3,4] and network levels [1], and (iii) they have certain advantages over standard networks in the processing of temporal sequences [5–9]. In Ref. [1] we formulated the stochastic dynamics as a linear Markov process and, using results from the spectral theory of linear Markov operators due to Lasota and Mackey [10], we derived conditions under which the network converges to a unique limiting density on the space of membrane potentials *(asymptotic stability)*. The limiting density was also shown to be a differentiable function of the parameters of the network including the connection weights and decay rates.

However, additive noise does not take into account sources of noise intrinsic to the neuron. The most significant of these is *synaptic* noise arising from random fluctuations in the number of packets or *quanta* of chemical transmitters released into the synaptic cleft on arrival of an action potential [11]. Such (multiplicative) noise can be incorporated into binary networks by taking the connection weights to be independently updated at every time step according to fixed probability distributions [12,13,2]. That is, the connection weight at a given synapse and time t is decomposed as $w(t)=q_0\epsilon u(t)$, where $|q_0|$ is related to the amount of transmitter in a single packet (vesicle), the sign of q_0 indicates whether or not the synapse is excitatory or inhibitory, ϵ is the fixed postsynaptic efficacy (indicating the efficiency with which chemicals bind to receptors in the postsynaptic cell membrane), and $u(t)$ is a random variable representing the number of packets released at time t. If an action potential arrives at the synapse time t, then $u(t)=u$, where u is generated from a fixed distribution p; otherwise, $u(t)=0$. The distribution p is realistically described by a binomial of size L, where L is the maximum number of vesicles released with $L \approx 1-10$ for the central nervous system [14]. Recent evidence [15] suggests that the case $L=1$, the so-called one-vesicle model, may occur in certain cortical neurons.

One consequence of the quantal nature of synaptic transmission is that each random weight only has a finite number of possible values (assuming that p is binomial rather than Poisson, say). If this is combined with the fact that the number of output states of a binary network is itself finite, then it follows that the stochastic dynamics of a time-summating binary network with synaptic noise (and fixed external inputs) is described by a *random iterated function system* (IFS) on the space of membrane potentials $\mathcal{M} \subset \mathbb{R}^N$, where N is the number of neurons. A random IFS [16] consists of a finite set of continuous maps acting on some metric space and a corresponding set of probabilities for choosing one such map per iteration. In the case of the above network model, we can take the metric space to be $\mathcal{M}$ with the Euclidean metric. Each map is affine and is associated with particular configurations of weights and output states, giving a total of $(L+1)^{C(N)}$ maps, where $C(N)$ is the number of weights in the network. [$C(N)=N(N-1)$ for a fully connected network.] Moreover, the maps are contracting (since $\gamma < 1$) and the underlying IFS is said to be *hyperbolic* [16]. The probability of choosing any map at a given time step is generally state dependent, i.e., it is a function of the current values of the network's membrane potentials. The above picture is essentially unaltered if the less biologically significant source of intrinsic noise, threshold noise, is included as well.

In this paper we analyze the asymptotic behavior of time-summating networks in the presence of synaptic (and threshold) noise using results from the theory of hyperbolic random IFS's [16–19]. We begin by constructing the model in Sec. II and showing how quantum synaptic noise leads to the underlying structure of random IFS's. We investigate the limiting behavior of the model in Sec. III following the statistical approach of Norman [19], who carried out a detailed study of IFS's, under the name of distance diminishing models, within the context of mathematical psychology. One of the interesting features of a random IFS is that if the stochastic dynamics, which is described by a Markov process, is ergodic, then the asymptotic behavior of the system is often characterized by a unique invariant measure with a fractal-like structure. This fact underlies more recent interest in IFS's, which has been concerned with the generation of fractal images and their application to data compression [20,21]. It also indicates a major difference between the behavior of networks with synaptic noise, as presented here, and networks with additive noise discussed in Ref. [1]. For the invariant measures of the latter are smooth and allow probability densities on the space of membrane potentials to be defined. This is often no longer possible in the case of synaptic noise. We illustrate the fractal-like nature of the invariant measures in Sec. IV by considering a stochastic version of the Nagumo-Sato model of a single neuron [22]. It turns out that the invariant measure of the resulting IFS belongs to a class of measures studied originally by Erdos [23]. (It is interesting to note that the same IFS has recently arisen in a formulation of associative reinforcement learning in neural networks [24], as well as in a model of stochastically forced oscillators [25]. A review of IFS's and their diverse applications is presented in Ref. [26].) We also consider the effects of synaptic and threshold noise on the response characteristics of the neuron. Finally, in Sec. V we briefly discuss the issue of learning in neural networks with synaptic noise.

II. TIME-SUMMATING NETWORKS WITH SYNAPTIC NOISE

We define a network of N time-summating neurons with synaptic noise as follows [2].

(a) Time is discretized in terms of the smallest unit of time τ, taken to be equal to the absolute refractory period. (For simplicity we set $\tau=1$.)

(b) The output of the ith neuron at time m is denoted by $a_i(m)$, with $a_i(m)=1$ if the neuron fires an action potential and $a_i(m)=0$ otherwise. The output state of the network is specified by the vector $\mathbf{a}=(a_1,\ldots,a_N)$.

(c) The arrival of an action potential from the jth neuron causes the release of u_{ij} packets of transmitter chemical into synapse (ij). The efficiency with which these transmitters bind to receptors on the postsynaptic membrane is given by the postsynaptic efficacy ϵ_{ij}. The size of a single packet or vesicle is denoted by q_{ij}, with positive and negative values of q_{ij} corresponding, respectively, to excitatory and inhibitory inputs. More precisely, q_{ij} indicates the change in the membrane potential of the ith neuron induced by the release of a single vesicle when $\epsilon_{ij}=1$. (The synaptic delay between the arrival of an action potential and the resulting change in the membrane potential is taken to be a single time step.) The stochastic nature of synaptic transmission is incorporated by taking each u_{ij} to be independently generated from a fixed probability distribution [12,13]. Following Ref. [14], we shall take this distribution to be a binomial of size L (where, for simplicity, L is assumed to be synapse independent).

(d) The membrane potential V_i of the ith neuron evolves according to the equation

$$V_i(m)=\gamma_i V_i(m-1)+\sum_{j\,(\neq i)} w_{ij}(m-1)a_j(m-1)+I_i\ , \tag{2.1}$$

where I_i is a fixed external input and $\gamma_i<1$ is a decay factor. The connection weights in Eq. (2.1) satisfy

$$w_{ij}(m)=q_{ij}\epsilon_{ij}u_{ij}(m)\ . \tag{2.2}$$

The random variable $u_{ij}(m)$ is equal to the number of vesicles released at the discrete time m. If $a_j(m)=0$, then $u_{ij}(m)=0$, whereas if $a_j(m)=1$, then $u_{ij}(m)$ is generated by a binomial distribution. Hence, for a given output $a_j(m)=a$, the conditional probability that $u_{ij}(m)=u$ is given by

$$\begin{aligned}P_{ij}(u|a)&=\mathrm{Prob}(u_{ij}(m)=u\,|a_j(m)=a)\\&=a\begin{bmatrix}L\\u\end{bmatrix}\lambda_{ij}^{u}(1-\lambda_{ij})^{L-u}+(1-a)\delta_{u,0}\quad u\le L\end{aligned} \tag{2.3}$$

where λ_{ij} are constant parameters.

(e) A neuron fires whenever its membrane potential V_i exceeds a threshold h_i,

$$a_i(m)=\Theta(V_i(m)-h_i(m))\ , \tag{2.4}$$

where each $h_i(m)$ is an independent random variable generated from a fixed probability density ρ_i. In the presence of such threshold noise, the probability of the jth neuron firing when its membrane potential is equal to V_i is

$$\psi_i(V_i)=\int_{-\infty}^{\infty}dh_i\rho_i(h_i)\Theta(V_i-h_i)\ . \tag{2.5}$$

The above model can be interpreted as a discrete-time version of a leaky-integrator network in which details of quantal synaptic transmission have been included [2]. The major difference between a time-summating network and a standard binary network is the presence of the decay term $\gamma_i V_i$ in Eq. (2.1). This term represents, in idealized form, the persistence of cell activity over extended time intervals, with the rate of decay γ_i being determined by the electrical properties of the neuron, such as the leakage capacitance and resistance. Time-summating networks have been studied extensively elsewhere, both from the viewpoint of network dynamics [1,3,4] and temporal-sequence processing [5–9]. In particular, the stochastic dynamics of a time-summating network with additive noise and fixed weights and thresholds was studied in Ref. [1].

Extensions of the model are also possible, although we shall not consider these in any detail here. For example, spontaneous emission of vesicles in the absence of an incoming action potential can be incorporated by replacing the second term on the right-hand side of Eq. (2.1) with

$$\sum_{j\,(\neq j)} q_{ij}\epsilon_{ij}\{u_{ij}^{\rm st}(m-1)a_j(m-1) + u_{ij}^{\rm sp}(m-1)[1-a_j(m-1)]\}\ ,$$

where both stimulated (st) and spontaneous (sp) processes are generated according to binomial distributions so that Eq. (2.3) becomes

$$P_{ij}(u|a)=a\begin{bmatrix}L\\u\end{bmatrix}(\lambda_{ij}^{\rm st})^u(1-\lambda_{ij}^{\rm st})^{L-u} + (1-a)\begin{bmatrix}L\\u\end{bmatrix}(\lambda_{ij}^{\rm sp})^u(1-\lambda_{ij}^{\rm sp})^{L-u}\ ,$$

where $\lambda_{ij}^{\rm st} \gg \lambda_{ij}^{\rm sp}$. Another extension takes into account the fact that changes in the membrane potential V_i induced by a synaptic input from another neuron j depend on the size of deviation of V_i from some local, fixed resting potential S_{ij}. These so-called shunting effects can be incorporated into the time-summating model by multiplying the right-hand side of (2.1) by the exponential factor [2]

$$\exp\left[-\sum_{j\,(\neq i)}\frac{w_{ij}(m-1)}{S_{ij}}a_j(m-1)\right]\ .$$

Finally, some of the details of the geometrical structure of neurons can be included by splitting each neuron into a number of distinct compartments, each with its own local membrane potential [2]. All of these features can be accommodated within the analytical framework of IFS's presented below.

We now show how the dynamics of a time-summating network with synaptic noise, as expressed by Eq. (2.1), may be formulated as a random IFS on the space of membrane potentials $\mathcal{M}\subset\mathbb{R}^N$. (Since the dynamics is bounded, we may take $\mathcal{M}$ to be a compact subset of $\mathbb{R}^N$.) Associate with each synapse (ij) an integer $u_{ij}\in(0,1,\ldots,L)$ and denote the set of such integers by

$$\alpha=\{u_{ij}, i,j=1,\ldots,N, i\neq j\}\ .$$

Introduce the index set

$$\Omega=\{\alpha|\alpha\in\{0,\ldots,L\}^{C(N)}\}\ ,$$

where $C(N)$ is the number of connections in the network. [In the case of a fully connected network without self-interactions, $C(N)=N(N-1)$.] For each set α, define an affine mapping $F_\alpha:\mathcal{M}\to\mathcal{M}$ by

$$[F_\alpha(\mathbf{V})]_i=\gamma_i V_i+\sum_{j\,(\neq i)}\epsilon_{ij}q_{ij}u_{ij}+I_i\ . \tag{2.6}$$

We identify $\alpha(m)\in\Omega$ as the event that $u_{ij}(m)$ vesicles are released into synapse (ij), for $i\neq j$, at the mth time step. From Eqs. (2.3) and (2.5), the probability of an event α, for a given state $\mathbf{V}$, is

$$\Phi_\alpha(\mathbf{V})=\text{Prob}[\alpha(m)=\alpha|\mathbf{V}] = \sum_{\mathbf{a}\in\{0,1\}^N} P(\alpha|\mathbf{a})\tilde{P}(\mathbf{a}|\mathbf{V})\ , \tag{2.7}$$

where

$$P(\alpha|\mathbf{a})=\prod_{i,j(i\neq j)} P_{ij}(u_{ij}|a_j) \tag{2.8}$$

is the probability that the event α occurs given the output $\mathbf{a}$, and

$$\tilde{P}(\mathbf{a}|\mathbf{V})=\prod_{i=1}^{N}\psi_i(V_i)^{a_i}[1-\psi_i(V_i)]^{1-a_i} \tag{2.9}$$

is the probability that the output of the network is $\mathbf{a}$ given the state $\mathbf{V}$. The distributions Φ_α satisfy $\Phi_\alpha(\mathbf{V})\geq 0$ and $\sum_{\alpha\in\Omega}\Phi_\alpha(\mathbf{V})=1$ for all $\mathbf{V}\in\mathcal{M}$.

The set $\{(F_\alpha,\Phi_\alpha)|\alpha\in\Omega\}$ determines a random IFS $\mathcal{F}$ on the space of membrane potentials $\mathcal{M}$, endowed with, say, the Euclidean metric. That is, $\mathcal{F}$ consists of a finite, indexed set of continuous mappings on some metric space, together with a corresponding set of probabilities for selecting one such map at random per iteration. It is clear from the definitions of F_α and Φ_α, Eqs. (2.6) and (2.7), that the dynamical evolution of the membrane potentials, as described by Eq. (2.1), corresponds to an orbit of the IFS $\mathcal{F}$. In other words, we may rewrite Eq. (2.1) as

$$\mathbf{V}(m)=F_{\alpha(m-1)}[\mathbf{V}(m-1)]\ , \tag{2.10}$$

where $F_{\alpha(m-1)}=F_\alpha$ with probability $\Phi_\alpha[\mathbf{V}(m-1)]$, so that a particular trajectory of the dynamics is specified by a particular sequence of events

$$\{\alpha(m); m=0,1,\ldots|\alpha(m)\in\Omega\}\ ,$$

together with the initial point $\mathbf{V}(0)$.

It is important to note that $\mathcal{F}$ is in fact an *hyperbolic* IFS (using Barnsley's terminology [16]); the affine maps F_α of Eq. (2.6) are contraction mappings on $\mathcal{M}$, i.e., the contraction ratio λ_α of F_α, defined by

$$\lambda_\alpha=\sup_{\mathbf{V}\neq\mathbf{V}'}\frac{|F_\alpha(\mathbf{V})-F_\alpha(\mathbf{V}')|}{|\mathbf{V}-\mathbf{V}'|} \tag{2.11}$$

(assuming the Euclidean metric), satisfies $\lambda_\alpha<1$ for all $\alpha\in\Omega$. This follows from the fact that the decay factors in Eq. (2.6) satisfy $\gamma_i<1$ and $\lambda_\alpha=\gamma\equiv\max_i(\gamma_i)$. Then by the contraction mapping theorem [16], there exists a unique fixed point $\mathbf{V}^\alpha$ of F_α such that $\lim_{m\to\infty}(F_\alpha)^m(\mathbf{V})=\mathbf{V}^\alpha$ for all $\mathbf{V}\in\mathcal{M}$. This may trivially be seen from Eq. (2.6) with

$$V_i^\alpha=\left[I_i+\sum_{j\,(\neq i)}q_{ij}u_{ij}\epsilon_{ij}\right]\Big/(1-\gamma_i)\ .$$

The fact that $\mathcal{F}$ is hyperbolic will allow us to apply a number of known results concerning the limiting behavior of random IFS's in Sec. III.

Two features emerge from the formulation of the dynamics in terms of random IFS's. First, the neurons couple dynamically through the probabilities Φ_α and not through the mappings F_α which are diagonal [see Eq (2.6)]. Second, the weights $w_{ij}(m)$, Eq. (2.2), decompose into the product of a fixed postsynaptic term ϵ_{ij} and a random presynaptic term $u_{ij}(m)$. These two factors enter the IFS in different ways. The former parametrizes the mappings F_α, whereas the distribution of the latter contributes to the probabilities Φ_α. An important question is, where does learning take place? That is, do modifications occur postsynaptically in terms of the efficacies ϵ_{ij}, leading to changes in F_α, or do they occur presynaptically in terms of the parameters λ_{ij} of the distributions (2.3), leading to changes in Φ_α? (It is assumed that the vesicle size q_{ij} and the maximum possible number of vesicles L are not adaptive parameters.) We shall consider the issue of learning in Sec. V.

III. ANALYSIS OF DYNAMICS USING IFS'S

In this section we apply some general results concerning the properties of random hyperbolic IFS's on compact metric spaces to determine the limiting behavior of the neural network model introduced in Sec. II. Consider an ensemble of such networks describing different orbits of the same IFS $\mathcal{F}$, Eq. (2.10). The dynamics of this ensemble may be formulated in terms of the time evolution of probability distributions on the space of membrane potentials $\mathcal{M}$. Let $\mu_m(B)$ be the probability that an orbit of the ensemble passes through the subset $B\subset\mathcal{M}$ at time m, i.e., $\mathbf{V}(m)\in B$. More formally, let $\mathcal{B}(\mathcal{M})$ be the Borel σ field of $\mathcal{M}$ [27,28] and $\mathcal{P}(\mathcal{M})$ the space of probability measures on $\mathcal{B}(\mathcal{M})$. Then we have $\mu_m\in\mathcal{P}(\mathcal{M})$ such that

$$\mu_m(B)\equiv\int_B d\mu_m(\mathbf{V})=\text{Prob}[\mathbf{V}(m)\in B]\ ,\quad B\in\mathcal{B}(\mathcal{M})\ . \tag{3.1}$$

Note that, in contrast to the analysis of additive noise in Ref. [1], we cannot assume that the measures μ_m are Lebesgue and introduce smooth probability densities on $\mathcal{M}$ accordingly. For, as will be made clear below, there is the possibility of fractal-like structures emerging.

The sequence of measures $\{\mu_m\}$ describes a Markov process. Introduce the time-independent transition probability $\mathcal{K}(\mathbf{V},B)$ that given $\mathbf{V}(m)=\mathbf{V}$ at time m, then $\mathbf{V}(m+1)\in B$. This is equal to the probability of choosing a map $F\in\{F_\alpha,\alpha\in\Omega\}$ such that $F(\mathbf{V})\in B$. Thus,

$$\mathcal{K}(\mathbf{V},B)=\sum_{\alpha\in\Omega}\Phi_\alpha(\mathbf{V})\chi_B(F_\alpha(\mathbf{V}))\ ,\quad B\in\mathcal{B}(\mathcal{M}) \tag{3.2}$$

where χ_B is the indicator function

$$\chi_B(\mathbf{V})=\begin{cases}1 & \text{if } \mathbf{V}\in B\\ 0 & \text{otherwise}\ .\end{cases} \tag{3.3}$$

Given an initial probability measure $\mu_0\in\mathcal{P}(\mathcal{M})$, the following sequence of measures is induced by $\mathcal{K}$:

$$\mu_m(B)=\int_{\mathcal{M}}\mathcal{K}(\mathbf{V},B)d\mu_{m-1}(\mathbf{V})\ , \tag{3.4a}$$

which, from Eq. (3.2) can be rewritten as

$$\mu_m(B)=\sum_{\alpha\in\Omega}\int_{F_\alpha^{-1}(B)}\Phi_\alpha(\mathbf{V})d\mu_{m-1}(\mathbf{V})\ , \tag{3.4b}$$

where the inverse set

$$F_\alpha^{-1}(B)=\{V\in\mathcal{M}|F_\alpha(\mathbf{V})\in B\}\ .$$

Hence, the long-term dynamics of a network of the ensemble can be characterized by the asymptotic behavior of the sequence of measures $\{\mu_m\}$ generated according to Eq. (3.4).

In the following we shall take $\mathcal{F}$ to be an arbitrary hyperbolic random IFS on some metric space $(\mathcal{M},d)$ and summarize certain results concerning the limit of the sequence $\{\mu_m\}$ (see also Refs. [24,26]). Note that the dynamics of an IFS is independent of the particular metric d chosen; the introduction of a metric structure allows certain mathematical results to be proven and is useful in characterizing geometrical aspects of the system's attractor. We begin by discussing the simplest case, that of an IFS with probabilities $\Phi_\alpha>0$ which are independent of the state variables $\mathbf{V}$. (An explicit example of this will be given in Sec. IV in terms of a single-neuron model.) Under such a simplification, Eq. (3.4b) becomes

$$\mu_m(B)=K[\mu_{m-1}](B)=\sum_{\alpha\in\Omega}\Phi_\alpha\mu_{m-1}[F_\alpha^{-1}(B)]\ , \tag{3.5}$$

where K is a linear Markov operator on $\mathcal{P}(\mathcal{M})$ [28]. That is, K: $\mathcal{P}(\mathcal{M})\to\mathcal{P}(\mathcal{M})$ with (i) $K[\mu]\geq 0$ and (ii) $\int_{\mathcal{M}}dK[\mu]=1$ for all $\mu\in\mathcal{P}(\mathcal{M})$. Since the space $\mathcal{M}$ is a compact metric space, it can be shown that $\mathcal{P}(\mathcal{M})$ is a complete metric space with respect to the Hutchinson metric d_H defined by [29],

$$d_H(\mu,\nu)=\sup_{\varphi\in C}\left\{\int_{\mathcal{M}}\varphi\,d\mu-\int_{\mathcal{M}}\varphi\,d\nu\right\}\ ,\quad \mu,\nu\in\mathcal{P}(\mathcal{M})\ , \tag{3.6}$$

where

$$C=\{\varphi:\mathcal{M}\to\mathbb{R}\,\|\varphi(\mathbf{V})-\varphi(\mathbf{V}')|\leq d(\mathbf{V}-\mathbf{V}')\quad\text{for all } \mathbf{V},\mathbf{V}'\in\mathcal{M}\}\ .$$

Moreover, it turns out that since the mappings F_α are contracting with respect to d on $\mathcal{M}$, the operator K is contracting with respect to d_H on $\mathcal{P}(\mathcal{M})$. (In the case of the time-summating network of Sec. II, with d Euclidean,

$$d_H[K(\mu),K(\nu)]\leq\gamma d_H(\mu,\nu)$$

for $\gamma=\max_i\{\gamma_i\}$.) It can then be deduced from the contraction mapping theorem [16] that the limit of the sequence $\{\mu_m\}$ is the unique fixed point $\mu_{\mathcal{F}}$ of K, i.e.,

$$K[\mu_{\mathcal{F}}]=\mu_{\mathcal{F}}\ ,\quad \lim_{m\to\infty}K^m[\mu]=\mu_{\mathcal{F}}\quad\text{for all } \mu\in\mathcal{P}(\mathcal{M})\ . \tag{3.7}$$

The fixed point $\mu_{\mathcal{F}}$ is said to be the *unique invariant measure* of $\mathcal{F}$ and the dynamics is said to be *asymptotically stable*.

An important property of $\mu_{\mathcal{F}}$, as proven originally by Norman [19] and later independently by Elton [18], is that for almost all event sequences $\{\alpha(m),m=1,2,\ldots\}$, time averages are equal to space averages. In other words,

$$\lim_{M\to\infty}\frac{1}{M}\sum_{m=0}^{M-1}f(\mathbf{V}(m))=\int_{\mathcal{M}}f(\mathbf{V})d\mu_{\mathcal{I}}(\mathbf{V}) \tag{3.8a}$$

for all continuous functions f: $\mathcal{M}\to\mathbb{R}$ and all initial conditions $\mathbf{V}(0)$. Equation (3.8a) is a stronger condition than that of ergodicity, since the latter simply states that the two averages are equal for almost all initial conditions with respect to the measure $\mu_{\mathcal{I}}$. An equivalent result is that for almost all event sequences [18], the frequency with which an orbit $\{\mathbf{V}(m)\}$ visits a subset B is $\mu_{\mathcal{I}}(B)$,

$$\lim_{M\to\infty}\frac{\mathfrak{N}\{\mathbf{V}(m)\in B:\ 1\le m\le M\}}{M}=\mu_{\mathcal{I}}(B)\ ,\quad B\in\mathcal{B}(\mathcal{M})\ . \tag{3.8b}$$

It is interesting to note that the support of $\mu_{\mathcal{I}}$, which is a closed invariant subset $A_{\mathcal{I}}\subset\mathcal{M}$, called the *attractor* of $\mathcal{I}$, can be generated by a purely deterministic process independently of the probabilities Φ_α [16]. Let $\mathcal{H}(\mathcal{M})$ be the space of closed subsets of $\mathcal{M}$. Then it can be shown that this is a complete metric space with respect to the Hausdorff metric d_h where [30]

$$d_h(A,B)=\max[d_s(A,B),d_s(B,A)]\ ,\quad A,B\in\mathcal{H}(\mathcal{M}) \tag{3.9a}$$

and

$$d_s(A,B)=\max_{\mathbf{V}\in A}\min_{\mathbf{V}'\in B}d(\mathbf{V},\mathbf{V}')\ . \tag{3.9b}$$

Define the operator T on $\mathcal{H}(M)$ by

$$T(A)=F_1(A)\cup\cdots\cup F_q(A)\ ,\quad A\in\mathcal{H}(\mathcal{M})\ , \tag{3.10}$$

where q is the cardinality of the index set Ω. [Recall that $q=(L+1)^{C(N)}$ for the IFS of a time-summating neural network.] It can be proven that if the F_α are contractions on $\mathcal{M}$, then T is a contraction on $\mathcal{H}(\mathcal{M})$ [16] (with

$$d_h[T(A),T(B)]\le\gamma d_h(A,B)$$

in the case of a time-summating neural network). Hence, the contraction mapping theorem may be used to deduce that T has a unique fixed point $A_{\mathcal{I}}$ such that $\lim_{m\to\infty}T^m(A)=A_{\mathcal{I}}$ for all $A\in\mathcal{H}(\mathcal{M})$. The fixed point $A_{\mathcal{I}}$ is also the attractor of the random IFS $\mathcal{I}$ in the sense that for every orbit $\{\mathbf{V}(m),m=0,1,\dots\}$ of $\mathcal{I}$, $\mathbf{V}(m)\to A_{\mathcal{I}}$ as $m\to\infty$.

A major reason why IFS's have attracted interest recently, within the context of image generation and data compression [20,21], is that both the attractor $A_{\mathcal{I}}$ and the invariant measure $\mu_{\mathcal{I}}$ typically have a rich fractal-like structure. We shall illustrate this from the viewpoint of neural-network dynamics in Sec. IV. However, it is first necessary to see how the above picture extends to the more general case of state-dependent probabilities. We shall find that an extra nonergodic feature emerges—that of an absorbing state.

We shall follow the approach of Norman [19], who considered random IFS's (distance-diminishing models), in the context of mathematical learning theory. (Random IFS's with state-dependent probabilities have also been discussed by Barnsley *et al.* [17] and by Elton [18].) To proceed, introduce the m-step transition probability that $\mathbf{V}(m)\in B$ given $\mathbf{V}(0)$, which has the explicit form

$$\mathcal{K}^{(m)}(\mathbf{V},B)=\sum_{\alpha(1),\dots,\alpha(m)}\Phi_{\alpha(1)}(\mathbf{V})\Phi_{\alpha(2)}(F_{\alpha(1)}(\mathbf{V}))\cdots\Phi_{\alpha(m)}(F_{\alpha(m-1)}\circ\cdots\circ F_{\alpha(1)}(\mathbf{V}))\chi_B(F_{\alpha(m)}\circ\cdots\circ F_{\alpha(1)}(\mathbf{V}))\ . \tag{3.11}$$

Then Eq. (3.4) may be rewritten as

$$\mu_m=\int_{\mathcal{M}}\mathcal{K}^{(m)}(\mathbf{V},.)d\mu_0(\mathbf{V}) \tag{3.12}$$

and the asymptotic behavior of $\mathcal{I}$ may be formulated in terms of the sequence $\{\mathcal{K}^{(m)},m=1,2,\dots\}$. Such a sequence will be said to converge uniformly to some $\mathcal{K}_\infty$ if for any (Borel) subset $B\subset\mathcal{M}$ and any $\epsilon>0$, there is an integer M for which

$$\mathcal{K}_\infty(\mathbf{V},B^0)-\epsilon\le\mathcal{K}^{(m)}(\mathbf{V},B)\le\mathcal{K}_\infty(\mathbf{V},\bar{B})+\epsilon$$

for all $m\ge M$ and $V\in\mathcal{M}$. Here B^0 is the interior and $\bar{B}$ the closure of B [27]. In particular, Norman distinguishes two important types of limit for the sequence $\{\mathcal{K}^{(m)}\}$. To describe these, it is useful to introduce some extra notation. Let $T_m(\mathbf{V})$ be the set of values that $\mathbf{V}(m)$ has with probability greater than zero for a given $\mathbf{V}(0)=\mathbf{V}$. Thus,

$$T_m(\mathbf{V})=\{\mathbf{V}'|\mathcal{K}^{(m)}(\mathbf{V},\{\mathbf{V}'\})>0\}\ . \tag{3.13}$$

Next, define an *absorbing state* as one which satisfies $T_1(\mathbf{V})=\{\mathbf{V}\}$. In other words, once such a state is occupied, it remains so with probability 1. Finally, set $\bar{d}(A,B)$ to be the minimum distance between any two subsets A and B of $\mathcal{M}$,

$$\bar{d}(A,B)=\min_{\{\mathbf{V}\in A,\mathbf{V}'\in B\}}d(\mathbf{V},\mathbf{V}')\ . \tag{3.14}$$

Norman has shown the following [19]:

(a) If a random hyperbolic IFS satisfies

$$\lim_{m\to\infty}\bar{d}(T_m(\mathbf{V}),T_m(\mathbf{V}'))=0\quad\text{for all }\mathbf{V},\mathbf{V}'\in\mathcal{M}\ , \tag{3.15}$$

then the sequence $\{\mathcal{K}^{(m)},m=1,2,\dots\}$ converges uniformly to $\mathcal{K}_\infty$ such that $\mathcal{K}_\infty(\mathbf{V},.)$ is independent of the initial state $\mathbf{V}$. Thus, the sequence of measures $\{\mu_m,m=0,1,\dots\}$ converges to a unique invariant measure $\mu_{\mathcal{I}}$, where $\mu_{\mathcal{I}}\equiv\mathcal{K}_\infty(\mathbf{V},.)$ for any $\mathbf{V}$. Moreover, $\mu_{\mathcal{I}}$ satisfies Eq. (3.8) so that time averages are equal to space averages.

(b) Suppose that a random hyperbolic IFS has $D>1$ absorbing states $\mathbf{U}_1,\dots,\mathbf{U}_D$ such that for any $\mathbf{V}\in\mathcal{M}$ there exists some $\mathbf{U}_{j(\mathbf{V})}$ for which

$$\lim_{m\to\infty} \bar{d}(T_m(\mathbf{V}),\{\mathbf{U}_{j(\mathbf{V})}\})=0 . \tag{3.16}$$

Then the sequence of states $\{\mathbf{V}(m), m=0,1,\dots\}$ converges with probability 1 to a random absorbing state $\mathbf{V}_\infty$. This means that

$$\text{Prob}\{\lim_{m\to\infty}\mathbf{V}(m)=\mathbf{V}_\infty, \mathbf{V}_\infty\in\{\mathbf{U}_1,\dots,\mathbf{U}_D\}\}=1 . \tag{3.17}$$

Moreover, given an initial state $\mathbf{V}$, $\mathcal{K}^{(m)}$ converges uniformly to $\mathcal{K}_\infty$, where, for any subset B,

$$\mathcal{K}_\infty(\mathbf{V},B)=\sum_{j=1}^{D}\Gamma_j(\mathbf{V})\delta_{\mathbf{U}_j}(B) . \tag{3.18}$$

In Eq. (3.18), $\delta_{\mathbf{U}_j}$ is the pointlike measure defined by $\delta_{\mathbf{U}_j}(B)=1$ if $B\ni\mathbf{U}_j$ and 0 otherwise, and $\Gamma_j(\mathbf{V})$ is the nonzero probability of arriving at the absorbing state $\mathbf{U}_j$ from $\mathbf{V}$,

$$\Gamma_j(\mathbf{V})=\text{Prob}\{\lim_{m\to\infty}\mathbf{V}(m)=\mathbf{U}_j|\,\mathbf{V}(0)=\mathbf{V}\} . \tag{3.19}$$

We end this section by relating the above results to the dynamics of an ensemble of time-summating neural networks, expressed in the space of binary outputs $\{0,1\}^N$. Let $P_m(\mathbf{a})$ be the probability that the output of a network at time m is $\mathbf{a}$. Then $P_m(\mathbf{a})$ may be obtained from the measure μ_m by the projection

$$P_m(\mathbf{a})=\int_{\mathcal{M}}\tilde{P}(\mathbf{a}|\mathbf{V})d\mu_m(\mathbf{V}) , \tag{3.20}$$

where $\tilde{P}(\mathbf{a}|\mathbf{V})$ is defined by Eq. (2.9). Thus the sequence of densities $\{\mu_m\}$ on $\mathcal{M}$ induces, according to Eq. (3.20), the sequence of probability distributions $\{P_m\}$ on $\{0,1\}^N$. Furthermore, if the dynamics of the underlying IFS $\mathcal{J}$, defined by Eqs. (2.6) and (2.7), is ergodic [case (a)], then the sequence $\{P_m\}$ converges to a unique distribution $P_{\mathcal{J}}$ with

$$P_{\mathcal{J}}(\mathbf{a})=\lim_{m\to\infty}P_m(\mathbf{a})=\int_{\mathcal{M}}\tilde{P}(\mathbf{a}|\mathbf{V})d\mu_{\mathcal{J}}(\mathbf{V}) \tag{3.21}$$

independently of the initial distribution. On the other hand, if the dynamics is nonergodic or absorbing [case (b)] and each network of the ensemble has the initial state $\mathbf{V}(0)$ with probability 1, then

$$\lim_{m\to\infty}P_m(\mathbf{a})=\sum_{j=1}^{D}\Gamma_j(\mathbf{V}(0))\tilde{P}(\mathbf{a}|\mathbf{U}_j) . \tag{3.22}$$

In the ergodic case, it can be shown that the invariant distribution $P_{\mathcal{J}}$ of Eq. (3.21) is generally a differentiable function of parameters such as the decay rates γ_i and external inputs I_i. This follows from a theorem due to Withers [31]: Suppose that the mappings F_α and probabilities Φ_α of an hyperbolic IFS $\mathcal{J}$ on a compact metric space $\mathcal{M}$ are smooth functions of $\mathbf{V}\in\mathcal{M}$ and a parameter ξ. Also assume that the system converges to a unique invariant measure $\mu_{\mathcal{J}}$. (Both $\mu_{\mathcal{J}}$ and its support $A_{\mathcal{J}}$ are functions of ξ.) Then [31], for any smooth function f: $\mathcal{M}\to\mathbb{R}$ the average

$$\bar{f}=\int_{\mathcal{M}}f\,d\mu_{\mathcal{J}} \tag{3.23}$$

is a differentiable function of the parameter ξ. In the particular case of our neural-network model, the theorem holds for each $P_{\mathcal{J}}(\mathbf{a})$, $\mathbf{a}\in\{0,1\}^N$, as a function of γ_i and I_i, provided that $\tilde{P}(\mathbf{a}|\mathbf{V})$, Eq. (2.9), is a differentiable function of $\mathbf{V}$, i.e., the probability density ρ_i associated with threshold noise is differentiable. (Thus, one situation in which the result fails is the absence of threshold noise.) We shall illustrate this in Sec. IV using a single-neuron model.

Another feature of the ergodic case, which follows from Eqs. (3.8) and (3.21), is that time averages are independent of initial conditions and may be replaced by ensemble averages over the limiting distribution $P_{\mathcal{J}}$. Thus, for any state variable X: $\{0,1\}^N\to\mathbb{R}$,

$$\lim_{M\to\infty}\sum_{m=0}^{M-1}\frac{X(\mathbf{a}(m))}{M}=\sum_{\mathbf{a}\in\{0,1\}^N}P_{\mathcal{J}}(\mathbf{a})X(\mathbf{a}) . \tag{3.24}$$

In Ref. [1] we considered the behavior of a time-summating network in the thermodynamic limit $N\to\infty$. In particular, we derived dynamical mean-field equations for a time-summating neural network with threshold noise and fixed weights and external inputs. These mean-field equations had periodic and chaotic solutions, implying a breaking of asymptotic stability, as expressed by Eq. (3.21), in the large-N limit. [Note, however, that in Ref. [1] it was incorrectly assumed that threshold noise was effectively equivalent to additive noise. In fact, the former, like synaptic noise, is described by a stochastic process involving a finite set of maps (IFS), whereas the latter involves an infinite set (continuum) of maps. The techniques used to study the two cases differ considerably. Moreover, IFS's are distinguished by the possibility of fractal-like structures emerging.] The emergence of complex behavior in the thermodynamic limit is well known in the spin-glass approach to associative memory in Hopfield-Little networks [32].

Finally, observe that, in general, the sequence $\{P_m\}$ of Eq. (3.20) does not evolve according to a Markov chain, but is described by a non-Markovian process. This reflects the fact that the membrane potential of a neuron is a function of all previous inputs to that neuron [as may be seen by iterating equation (2.1)]. An exception occurs in the limit $\gamma_i\to 0$. To see this, set $\gamma_i=0$ in Eqs. (2.6) and (3.2) (so that F_α is now $\mathbf{V}$ independent) and use equations (3.4a), (2.7)–(2.9), and (3.20) to obtain the Markov chain

$$P_m(\mathbf{b})=\sum_{\mathbf{a}}Q_{\mathbf{ba}}P_{m-1}(\mathbf{a}) , \tag{3.25}$$

where the transition matrix $\mathbf{Q}$ satisfies

$$\begin{gathered}\mathbf{Q}_{\mathbf{ba}}=\sum_{\alpha\in\Omega}[\tilde{P}(\mathbf{b}|\mathbf{V}(\alpha))P(\alpha|\mathbf{a})] ,\\ V_i(\alpha)=\sum_{j\,(\neq i)}q_{ij}u_{ij}\epsilon_{ij}+I_i .\end{gathered} \tag{3.26}$$

Equation (3.26) may be rewritten in the more familiar form [12,13]

$$\mathbf{Q}_{\mathbf{ba}}=\prod_{i=1}^{N}p(i|\mathbf{a})^{b_i}[1-p(i|\mathbf{a})]^{1-b_i} , \tag{3.27}$$

where

$$p(i|\mathbf{a})=\sum_{\{u_{ij}\le L, j\neq i\}}\left[\prod_{j\,(\neq i)}\begin{bmatrix}L\\u_{ij}\end{bmatrix}\lambda_{ij}^{u_{ij}}(1-\lambda_{ij})^{L-u_{ij}}\right]\int_{-\infty}^{\infty}\rho_i(h_i)\Theta\left[\sum_{j\,(\neq i)}q_{ij}\epsilon_{ij}u_{ij}a_j+I_i-h_i\right]dh_i \tag{3.28}$$

is the conditional probability of neuron i firing given the output state $\mathbf{a}$ at the previous time step. In the absence of synaptic noise, with $u_{ij}\equiv 1$, say, and if the density ρ_i is defined by

$$\rho_i(h_i)=\frac{d}{dh_i}\frac{1}{1+e^{\beta h_i}}\ , \tag{3.29}$$

then Eq. (3.29) reduces to the firing probability of the Little model [33]. For a detailed analysis of the stochastic dynamics of the Little model, see Ref. [34].

IV. EXAMPLE: SINGLE-NEURON MODEL

To illustrate the analysis of Sec. III, we consider a simple example consisting of a single time-summating neuron with inhibitory feedback. Thus,

$$V(m)=\gamma V(m-1)$$
$$-w(m-1)\Theta[V(m-1)-h(m-1)]+I\ , \tag{4.1}$$

where $w(m)=q\epsilon u(m)$, $q,\epsilon>0$, and $u(m)$ is the random number of vesicles released at time m. For a given output

$$a(m)\equiv\Theta[V(m)-h(m)]=a\ ,$$

$u(m)$ is generated according to

$$P(u|a)=\text{Prob}(u(m)=u|a(m)=a)$$
$$=a\begin{bmatrix}L\\u\end{bmatrix}\lambda^u(1-\lambda)^{L-u}+(1-a)\delta_{u,0}\quad u\le L\ . \tag{4.2}$$

The random threshold $h(m)$ is generated from the density (3.29) so that

$$\psi(V)=\text{Prob}(a=1|V)=\frac{1}{1+e^{-\beta V}}\ . \tag{4.3}$$

There are two possible interpretations of the inhibitory feedback.

(a) An inhibitory interneuron mediates the feedback so that whenever the excitatory neuron represented by Eq. (4.1) fires, it excites the inhibitory neuron, which in turn fires, leading to the excitatory neuron receiving a negative feedback.

(b) The feedback represents the effects of the relative refractory period [22]—the time interval, after the absolute refractory period t_R, over which a neuron is capable of firing but its likelihood of firing is suppressed. Such a phenomenon is often described (in continuous-time models) in terms of a time-dependent threshold $\kappa(\Delta t)$, where Δt is the time after emission of an action potential. In particular, $\kappa(\Delta t)=\infty$ for $0<\Delta t\le t_R$ and $\kappa(\Delta t)$ is continuous and monotonically decreasing for $\Delta t>t_R$. An alternative way of implementing such an effect is to assume that a neuron receives a negative feedback a time t_R after firing, which then decays due to leakage [2]. In Eq. (4.1), this corresponds to the presence of the term

$$-w\Theta[V(m-1)-h(m-1)]$$

with w fixed. [In contrast to case (a), the feedback does not involve synaptic processing and, hence, w is not a random variable.]

We shall use interpretation (a) in the following and assume that synaptic noise is described by a one-vesicle model [14], which corresponds to setting $L=1$ in Eq. (4.2). Equation (4.1) then determines a random IFS $\mathcal{F}$ consisting of the two maps F_0,F_1: $[V_1,V_0]\to[V_1,V_0]$ with associated probabilities Φ_0,Φ_1, where

$$F_0(V)=\gamma V+I\ ,\quad \Phi_0(V)=1-\lambda\psi(V)$$
$$F_1(V)=\gamma V-q\epsilon+I\ ,\quad \Phi_1(V)=\lambda\psi(V)\ ,$$
$$\gamma<1,\quad \lambda\le 1 \tag{4.4}$$

and $V_0=I/(1-\gamma)$, $V_1=(I-q\epsilon)/(1-\gamma)$ are the fixed points of F_0 and F_1. [For nonzero temperatures $(T>0)$ the membrane potential $V(m)$ converges to the interval $[V_1,V_0]$ for all initial conditions $V(0)$.] The dynamics of this system will be described for various parameter regimes.

First, consider the high-temperature limit $T\to\infty$ $(\beta\to 0)$ in which $\psi(V)\to\frac{1}{2}$ for all V. Without loss of generality, we set $q\epsilon=I$ and $I=1-\gamma$, so that $F_{0,1}$ are mappings on the unit interval [0,1] and Eq. (4.4) reduces to

$$F_0(V)=\gamma V+1-\gamma\ ,\quad \Phi_0=1-\lambda/2\ ,$$
$$F_1(V)=\gamma V\ ,\quad \Phi_1=\lambda/2\ . \tag{4.5}$$

Since Eq. (4.5) describes an hyperbolic IFS $\mathcal{F}$ with state-independent probabilities, there exists a unique invariant measure $\mu_{\mathcal{F}}$ (see Sec. III). It is interesting to note that the same IFS or, rather, family of IFS's has arisen in a number of different contexts, including a learning rule for a stochastic learning automaton [24] and a model of stochastically forced oscillators [25]. Moreover, the invariant measure $\mu_{\mathcal{F}}$ in the case $\lambda=1$ has been an object of considerable mathematical interest for at least 50 years [23]. Although many of its properties are still not very well understood, its behavior as a function of γ can be characterized as follows [23,24,26].

(i) For $\gamma<\frac{1}{2}$ the support $A_{\mathcal{F}}$ of $\mu_{\mathcal{F}}$ is a Cantor set whose Hausdorff dimension is $-\log 2/\log\gamma$. In particular, for $\gamma=1/3$ one obtains the middle thirds Cantor set and $\mu_{\mathcal{F}}$ is the uniform measure.

(ii) For $\gamma\ge 1/2$ the support of $\mu_{\mathcal{F}}$ is the whole unit interval and for many values of γ, $\mu_{\mathcal{F}}$ exhibits a rich fractal structure. This is illustrated in Figs. 1(a)–1(c) for the values

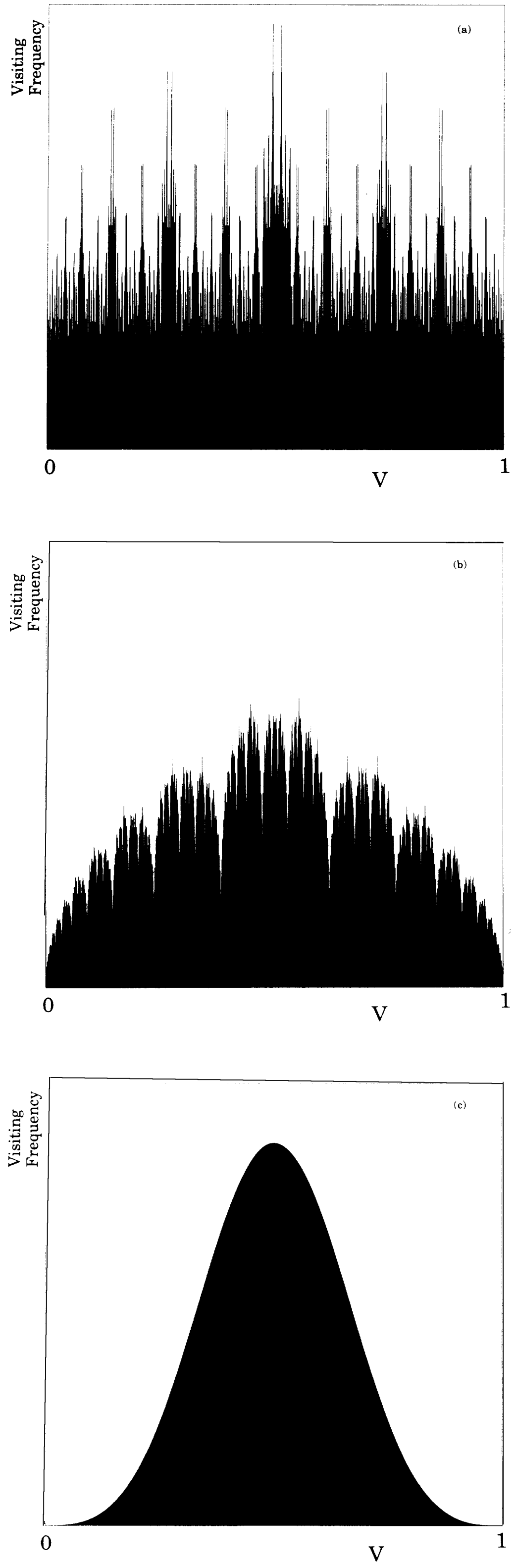

FIG. 1. The invariant measure of the random IFS, Eq. (4.5), is shown for $\lambda=1$. The unit interval is partitioned into 2100 equal subintervals and the frequency histograms displayed for different values of the decay rate γ. (a) $\gamma=0.52$; (b) $\gamma=(\sqrt{5}-1)/2$; (c) $\gamma=2^{-1/4}$.

(a) $\gamma=0.52$,

(b) $\gamma=(\sqrt{5}-1)/2\approx 0.618$,

which is the inverse of the golden mean, and

(c) $\gamma=2^{-1/4}\approx 0.841$.

For each value of γ, a frequency histogram is plotted displaying how often an orbit $\{V(m)\}$ visits a particular subinterval of [0,1]. The fact that such a histogram is a reasonable approximation to the invariant measure $\mu_{\mathcal{T}}$ follows from Eq. (3.8b). One feature that emerges from Fig. 1 is that the measure becomes progressively smoother as $\gamma\rightarrow 1$. A formal way of characterizing the smoothness, or lack thereof, of the measure $\mu_{\mathcal{T}}$ is to compare it with the Lebesgue measure m on the unit interval. (Integration with respect to Lebesgue is equivalent to standard Riemann integration.) Thus, $\mu_{\mathcal{T}}$ is said to be absolutely continuous with respect to m if $\mu_{\mathcal{T}}(B)=0$ for every Borel subset $B\in\mathcal{B}([0,1])$ for which $m(B)=0$. In this case it is possible to define a density for $\mu_{\mathcal{T}}$ so that $\mu_{\mathcal{T}}(B)=\int_B \rho\, dm$. On the other hand, $\mu_{\mathcal{T}}$ is said to be mutually singular with respect to m if there exist two disjoint Borel sets A and B such that (1) $\mu_{\mathcal{T}}(C)=0$ whenever $C\cap A=\phi$ and (2) $m(C)=0$ whenever $C\cap B=\phi$ [27]. In this case a density ρ does not exist. It can be shown [23] that $\mu_{\mathcal{T}}$ satisfies one of these two properties for all $\gamma\in[0,1]$. However, it is not known into which category $\mu_{\mathcal{T}}$ falls for particular values of γ except in special cases. For example, $\gamma=(\sqrt{5}-1)/2$ is singular [Fig. 1(b)], whereas $\gamma=\sqrt[n]{\frac{1}{2}}$ is absolutely continuous for $n\geq 2$ [Fig. 1(c)]. (Similar behavior occurs when $\lambda<1$. However, the histograms are no longer symmetric about the point $V=\frac{1}{2}$, but are weighted towards unity. This reflects the fact that $\Phi_0>\Phi_1$ when $\lambda<1$.)

In the presence of finite, nonzero temperatures T, the IFS of Eq. (4.4) has state-dependent probabilities and satisfies Eq. (3.15). Hence, the limiting behavior of the system is still characterized by a unique invariant measure $\mu_{\mathcal{T}}$. Moreover, the measure exhibits fractal-like structures similar to those described above. Differences do occur, however, at zero temperatures ($T=0$), i.e., in the absence of threshold noise. For example, there are changes in the interval over which the long-term dynamics is confined as well as in the response characteristics of the neuron. Moreover, absorbing states can occur.

Consider for the moment the deterministic case $\lambda=1$, $T=0$ (zero synaptic and threshold noise) in which Eq. (4.1) describes the Nagumo-Sato model of a neuron [22]. Also assume that $0<I<q\epsilon$. The membrane potential V then converges to the interval $[I-q\epsilon,I]\subset[V_1,V_0]$ and the dynamics is generated by a piecewise-linear map F, $F(V)=\gamma V-q\epsilon\Theta(V)+I$ (see Fig. 2). The typical limiting behavior of the system is periodic. (For other values of I the dynamics is trivial.) Moreover, the average firing rate defined by

$$\bar{a}=\lim_{M\to\infty}\frac{1}{M}\sum_{m=1}^{M}a(m)\ ,\quad a(m)=\Theta[V(m)] \tag{4.6}$$

is independent of the initial state $V(0)$ and, as a function

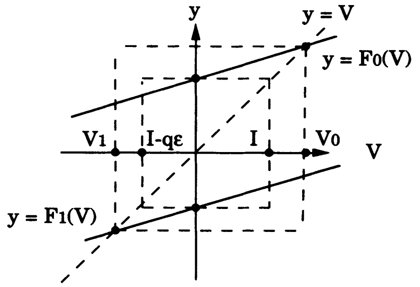

FIG. 2. Graph of the mappings F_0 and F_1 of Eq. (4.5). Dashed rectangles indicate regions of convergence.

of the external input I, forms a complete devil's staircase. That is, $\bar{a}$ is a continuous, monotonic function of I which assumes rational values on nonempty intervals of I and is irrational on a Cantor set of I [see Fig. 3(a)]. If $\bar{a}$ is rational, $\bar{a}=p/q$, then there is a periodic orbit of period q that is globally attracting. On the other hand, when $\bar{a}$ is irrational, there are no periodic points and the attractor is a Cantor set [35,3].

If synaptic noise is now introduced ($\lambda<1$, $T=0$), the long-term dynamics is confined to the interval $[I-q\epsilon, V_0]$ assuming, as before, that $0<I<q\epsilon$; N.B., for $V<0$ the dynamics is generated by the map F_0 along (Fig. 2). Although the dynamics is no longer periodic, the steplike nature of the neuron's response characteristics tend to be preserved even for high values of synaptic noise [Fig. 3(b)]. This should be contrasted with the effect of threshold noise ($T>0$, $\lambda=1$), which smooths the response characteristics of the neuron [Fig. 3(c)]. This feature may be understood by noting that Eqs. (3.21) and (3.24) imply that the average firing rate may be rewritten as

$$\bar{a}=\int \psi(V)\,d\mu_{\mathcal{T}}(V)\ , \tag{4.7}$$

where the right-hand side of (4.7) is a differentiable function of the external input I provided that ψ is a smooth function of V, i.e., $T>0$.

Finally, note that if $T=0$ and $I<0$, then the fixed point $V=V_0<0$ is a unique absorbing state. This follows from the fact that for all initial conditions, there exists an integer M such that $V(m)<0$ for $m>M$ and $\Phi_1(V)=0$ whenever $V<0$.

V. DISCUSSION

In this paper we have formulated the stochastic dynamics of a time-summating binary network with quantal synaptic noise in terms of a random IFS on the space of membrane potentials. We have then used results from the theory of IFS's to determine the limiting behavior of the network, which was illustrated by a simple example of a single neuron with inhibitory feedback. In this final section we discuss some of the implications of the above for learning in neural networks.

As briefly mentioned at the end of Sec. II, there is a significant difference between the dynamical role of

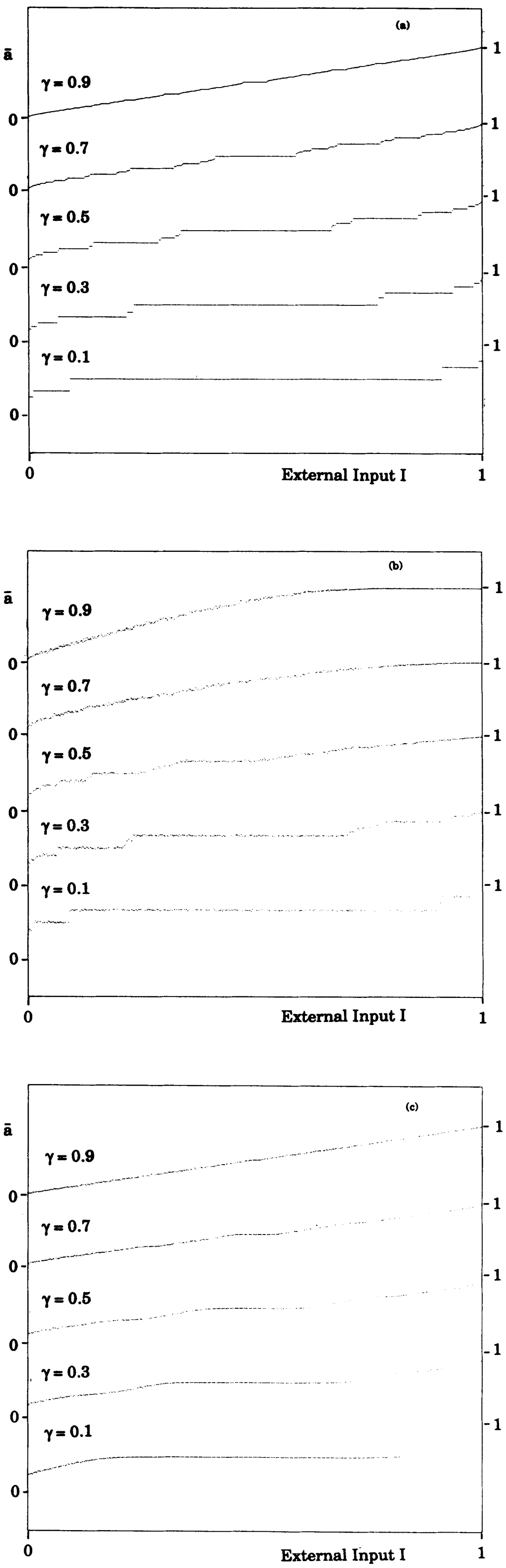

FIG. 3. Response characteristics of a single neuron with inhibitory feedback. The average firing rate $\bar{a}$ is plotted as a function of the external input I for various values of the decay rate γ and different sources of noise. (a) $\lambda=1.0$, $T=0$ (zero noise); (b) $\lambda=0.5$, $T=0.0$ (synaptic noise); (c) $\lambda=1.0$, $T=0.04$ (threshold noise).

presynaptic and postsynaptic contributions to the synaptic weight $w(m)=q_0u(m)\epsilon$. The presynaptic part is associated with the stochastic release of $u(m)$ packets of chemical transmitters according to a binomial distribution of the form (4.2). The postsynaptic part corresponds to the efficiency with which transmitters bind to receptors in the postsynaptic membrane and is characterized by the efficacy ϵ. The mean and variance of the random weight $w(m)$ are given by $\bar{w}=q_0\epsilon\lambda L$ and $\Delta w=(q_0\epsilon)^2\lambda L(1-\lambda)$, respectively, where L is the (fixed) maximum number of packets that can be released. Thus, at each synapse there are (at least) two control parameters ϵ and λ, with the latter controlling the local level of noise. It is clear that if learning only involves presynaptic modifications in which λ gradually increases from 0 to 1, for fixed ϵ, then the noise generated by a single synapse is small at the initial phase of the learning period, increases to a maximum at $\lambda=\frac{1}{2}$, as the learning proceeds, and then decreases to zero at $\lambda=1$ [36]. Such a mechanism can enhance learning by preventing the system from becoming trapped in local minima, as suggested originally by Taylor [12]. (A more sophisticated way of controlling synaptic noise during learning has been proposed by Hanson [37].) On the other hand, if learning is purely postsynaptic, with ϵ increasing for fixed $\lambda<1$, then the variance increases monotonically as learning proceeds. The presence of residual noise after training can enhance the generalization ability of a network [38].

Turning from the local synaptic description to a more global IFS formulation of network dynamics, one can consider learning in terms of the following *inverse problem*. First, not that for fixed threshold noise, decay factors γ_i, and external inputs I_i, the set $\{(F_\alpha,\Phi_\alpha)|\alpha\in\Omega\}$, defined by Eqs. (2.6) and (2.7), determines a family of random IFS's acting on $\mathcal{M}$, which is parametrized by the set

$$\Gamma=\{(\epsilon_{ij},\lambda_{ij})|i,j=1,\ldots,N,i\neq j\} .$$

We shall also assume that the dynamics is ergodic in the sense of case (a) of Sec. III. [An alternative mode of network operation is to work in nonergodic regimes, either by allowing absorbing states or by taking the thermodynamic limit, so that there is then a dependence on the initial condition $\mathbf{V}(0)$.] The inverse problem is to find a set Γ such that the invariant measure $\mu_{\mathcal{F}}$ of the associated IFS $\mathcal{F}$ is "sufficiently close" to some desired measure labeled by the external input $\mathbf{I}$. (More generally, learning is concerned with matching invariant and desired measures for a whole set of external inputs.) A number of comments are in order.

(a) The inverse problem for IFS's has attracted a great deal of attention recently in the area of fast image generation, since the encoding of a pixel image in terms of a relatively small number of IFS parameters could improve greatly on standard methods of date compression [20]. One approach to the inverse problem, for state-independent probabilities Φ_α, is based on the collage theorem [16]: Let ν be a probability measure on $\mathcal{M}$ and suppose that the Markov operator K, defined in Eq. (3.5), satisfies

$$d_H(\nu,K[\nu])<\epsilon , \tag{5.1}$$

where d_H is the Hutchinson metric, Eq. (3.6). Then

$$d_H(\mu_{\mathcal{F}},\nu)<\epsilon/(1-\gamma) , \tag{5.2}$$

where $\mu_{\mathcal{F}}$ is the invariant measure and $\gamma=\max_i\{\gamma_i\}$. Thus, the collage theorem implies that one can forget about the invariant measure $\mu_{\mathcal{F}}$ and simply concentrate on finding an IFS such that $d_H(\nu,K[\nu])$ is minimized. Such a search usually exploits the self-similarity of the fractal structures inherent in typical IFS's. It would be of interest to see whether these techniques can be extended to the neural-network context.

(b) In practice, one is interested in specifying a distribution P on the space of binary outputs $\{0,1\}^N$, rather than a probability measure μ on $\mathcal{M}$, with the two related by the projection (3.20). Obviously, such a specification does not determine the underlying measure uniquely. This has certain similarities with the moment method for the inverse problem [39]: to match a prescribed number of moments $x_{i,n}$ of the invariant measure $\mu_{\mathcal{F}}$ to those of a target measure, where

$$x_{i,n}=\int(V_i)^n d\mu_{\mathcal{F}}(\mathbf{V}) .$$

(c) One of the features of the IFS analysis is the introduction of metric structures, e.g., the Euclidean metric on the space of membrane potentials $\mathcal{M}$ and the corresponding Hutchinson metric on the space of probability measures $\mathcal{P}(\mathcal{M})$. An interesting issue is whether or not this metric structure can be used to develop an information theoretic approach to neural networks along lines analogous to the geometric method of Amari [40]. Note, however, that a major difference between the IFS formalism and Amari's formalism is that the presence of fractal-like structures in the former prohibits the use of standard differential geometry.

In conclusion, there are two major aspects of our work that warrant further study. The first aspect is that the discrete-time, leaky-integrator model considered in this paper incorporates a number of biological features missing from standard models such as Hopfield [32]. These include leaky-integrator effects reflecting the temporal aspects of the process by which neurons integrate their inputs, nonlinearities such as shunting effects, and details of the stochastic and quantal nature of synaptic processing. Furthermore, the various parameters of the model may be identified directly with their biological counterparts, e.g., postsynaptic efficacy and the mean and the variance of vesicular release. On the other hand, the model is sufficiently simple for mathematical analysis to be possible. Hence, it provides a useful framework for investigating the roles played in information processing by a wide range of biological features. In the case of noise processes, we have shown that presynaptic and postsynaptic contributions to the weights have different dynamical roles and lead to differences in the behavior of the variance during learning. Moreover, the response characteristics of individual neurons appear to depend on the particular source of noise present, i.e., synaptic or threshold. Other features of the model, such as the role of leaky-integrator effects in temporal signal processing, are considered elsewhere [2,8,9].

The second aspect of our work concerns the dynamics of networks of coupled random maps. Such systems are of interest for a number of reasons. First, as discussed above, some of the techniques developed to solve the inverse problem for random IFS's may be applicable to the problem of learning in neural networks. Second, in the deterministic limit the system reduces to a network of coupled circle maps [3], which has recently been shown to provide a useful tool to simulate the behavior of the temporal correlation and decorrelation of the spiking of groups of neurons [41]; such behavior has been suggested as a possible mechanism for linking together features to form a single coherent object—the binding problem [42]. Furthermore, if some spatial structure is imposed on the network, then we obtain an example of a coupled map lattice that can display a wide range of cooperative phenomena [43]. Therefore, the results of this paper may be useful in understanding the effects of noise on such systems.

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[1] P. C. Bressloff, Phys. Rev. A **44**, (1991).

[2] P. C. Bressloff and J. G. Taylor, in *Proceedings of the International Neural Network Conference, Paris, 1990* (Kluwer Academic, 1990), p. 1009; Neural Networks **4**, 789 (1991).

[3] P. C. Bressloff and J. Stark, Phys. Lett. A **150**, 187 (1990).

[4] K. Aihara, T. Takabe, and M. Toyoda, Phys. Lett. A **144**, 333 (1990).

[5] M. I. Jordan (unpublished).

[6] W. S. Stornetta, T. Hogg, and B. A. Huberman, in *Neural Information Processing Systems,* edited by D. Anderson (MIT Press, Boston, 1987).

[7] M. C. Mozer, Complex Systems **3**, 349 (1987).

[8] J. G. Taylor and M. Reiss, Neural Networks **4**, 773 (1991); J. G. Taylor, Int. J. Neural Syst. **2**, 47 (1991).

[9] P. C. Bressloff and J. G. Taylor, J. Phys. A **25**, 833 (1992).

[10] A. Lasota and M. C. Mackey, Physica D **28**, 143 (1987).

[11] B. Katz, *The Release of Neural Transmitter Substance* (Liverpool University, Liverpool, England, 1969).

[12] J. G. Taylor, J. Theor. Biol. **36**, 513 (1972); G. L. Shaw and R. Vasudevan, Math. Biosci. **21**, 207 (1974).

[13] P. C. Bressloff, in *New Developments in Neural Computing,* edited by J. G. Taylor and C. L. T. Mannion (Adam Hilger, Bristol, England, 1989); P. C. Bressloff and J. G. Taylor, Phys. Rev. A **41**, 1126 (1990).

[14] H. Korn and D. S. Faber, in *Synaptic Functions,* edited by W. M. Edelman, W. Gall, and W. M. Cowan (Wiley, New York, 1987), p. 57.

[15] P. J. Harrison, J. J. B. Jack, and D. M. Kullman, J. Physiol. (London) **412**, 43 (1989).

[16] M. F. Barnsely, *Fractals Everywhere* (Academic, New York, 1988); M. F. Barnsley and S. Demko, Proc. R. Soc. London, Ser. A **399**, 243 (1985).

[17] M. F. Barnsley, S. Demko, J. Elton, and J. S. Geronimo, Ann. Inst. Henri Poincaré **24**, 3 (1988).

[18] J. H. Elton, Ergod. Th. Dynam. Syst. **7**, 481 (1987).

[19] M. F. Norman, J. Math. Psychology **5**, 61 (1968).

[20] M. F. Barnsley and A. D. Sloan, Byte **13**, 215 (1988).

[21] J. Stark, IEEE Trans. Neural Networks **2**, 156 (1991); Neural Networks **4**, 679 (1991).

[22] J. Nagumo and S. Sato, Kybernetik **10**, 155 (1972).

[23] P. Erdos, Am. J. Math. **61**, 974 (1939); **62**, 180 (1940).

[24] P. C. Bressloff and J. Stark, in *Fractals and Chaos,* edited by A. J. Crilly, H. Jones, and R. A. Earnshaw (Springer-Verlag, New York, 1991), p. 145.

[25] A. J. Irwin, S. J. Fraser, and R. Kapral, Phys. Rev. Lett. **64**, 2343 (1990); J. Stark, *ibid.* **65**, 3357 (1990).

[26] J. Stark and P. C. Bressloff, in *Proceedings of the IMA conference on Wavelets, Fractals and Fourier Transforms* (Springer-Verlag, Berlin, 1992).

[27] W. Rudin, *Real and Complex Analysis* (McGraw-Hill, New York, 1987).

[28] A. Lasota and M. C. Mackey, *Probabilistic and Deterministic Systems* (Cambridge University Press, Cambridge, 1985).

[29] J. Hutchinson, Indiana U. J. Math. **30**, 713 (1981).

[30] K. J. Falconer, *The Geometry of Fractal Sets* (Cambridge University Press, Cambridge, 1985).

[31] W. D. Withers, Physica D **28**, 206 (1987); Ergod. Th. Dynam. Syst. **10**, 599 (1990).

[32] D. J. Amit, *Modeling Brain Function* (Cambridge University Press, Cambridge, 1989).

[33] W. A. Little, Math. Biosci. **19**, 101 (1974).

[34] P. Peretto, Biol. Cybern. **50**, 51 (1984); J. W. Clark, Phys. Rep. **158**, 91 (1988).

[35] J. P. Keener, Trans. Am. Math. Soc. **261**, 589 (1980).

[36] P. C. Bressloff and J. G. Taylor (unpublished).

[37] S. J. Hanson, Physica D **42**, 265 (1990).

[38] D. Hansel and H. Sompolinsky, Europhys. Lett. **11**, 687 (1990); G. Gyorgi, Phys. Rev. Lett. **64**, 2957 (1990).

[39] E. R. Vrscay and C. J. Roehring, in *Computers and Mathematics,* edited by E. Kaltofen and S. M. Watt (Springer-Verlag, Berlin, 1989), p. 250; C. R. Handy and G. Mantica, Physica D **43**, 17 (1990).

[40] S-I. Amari, Neural Networks **4**, 443 (1991).

[41] M. Bauer and W. Martienssen, Network **2**, 345 (1991).

[42] C. M. Gray, P. Koenig, A. K. Engel, and W. Singer, Nature **338**, 334 (1989); R. Eckhorn, R. Bauer, W. Jordan, M. Borsch, W. Kruse, M. Munk, and H. J. Reitbock, Biol. Cybern. **60**, 121 (1988).

[43] K. Kaneko, Physica D **41**, 137 (1990).