Mean-field theory of globally coupled integrate-and-fire neural oscillators with dynamic synapses

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We analyze the effects of synaptic depression or facilitation on the existence and stability of the splay or asynchronous state in a population of all-to-all, pulse-coupled neural oscillators. We use mean-field techniques to derive conditions for the local stability of the splay state and determine how stability depends on the degree of synaptic depression or facilitation. We also consider the effects of noise. Extensions of the mean-field results to finite networks are developed in terms of the nonlinear firing time map. [S1063-651X(99)09108-4]

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I. INTRODUCTION

An important property of synaptic transmission between cortical cells is that the postsynaptic response depends on the temporal sequence of action potentials arriving at the presynaptic terminal [1]. This form of short-term synaptic plasticity can lead to either an effective reduction in the amplitude of response (*synaptic depression*) or an effective increase in response (*synaptic facilitation*). Recent studies of excitatory pathways in slices of cortical pyramidal cells found that, under repeated stimulation, the dominant form of short-term plasticity is synaptic depression, which develops after only a few spikes [2–4]. These studies also established how synaptic depression could provide a dynamical gain mechanism that increases sensitivity to small input rate changes, as well as an enhanced capability of detecting synchronous activity (see also [5]). Given the fact that synaptic depression (and facilitation) can significantly influence the response of single neurons to incoming spike trains, it is likely that such factors also affect behavior at the network level. Indeed, a recent theoretical investigation of a discrete-time oscillator network suggests that dynamic synapses could support a mechanism for central pattern generation [6]. Moreover, complex patterns of network activity have been found in a rate model describing a large population of excitatory neurons with dynamic synapses [7].

In this paper we analyze the effects of synaptic depression and facilitation on mode-locking in a globally coupled network of N integrate-and-fire (IF) neuronal oscillators. We first show how synaptic depression (facilitation) can increase (decrease) the collective period of oscillations of a phase-locked state (Sec. II). We then use mean-field theory (MFT) to derive an evolution equation for the mean activity of the population in the large-N limit (Sec. III). This extends previous work on activity-independent synapses [8–12] by introducing a second macroscopic variable that determines the total synaptic input. (In the absence of dynamic synapses the latter is directly related to the population activity). From a computational viewpoint, one of the interesting properties of the population activity is that it can respond almost instantaneously to sudden changes in input [13,14]. The network is usually assumed to be in a so-called asynchronous or splay state—all the neurons fire at the same mean rate but the firing times are maximally distributed over the common firing period. We use our mean-field equations to determine how the stability of the splay state is affected by dynamic synapses. We also show how mean-field theory can be extended to take into account the effects of noise (Sec. IV). Finally, we discuss an alternative to the mean-field approach in which the firing times are considered as the fundamental dynamical variables [15–22]. Such an approach is more generally applicable to finite, inhomogeneous networks with arbitrary connectivity, and has recently led to a number of insights concerning the dynamics of strongly coupled spiking neurons [20,21]. We use the firing time approach to determine how the results of mean-field theory can be extended to finite networks (Sec. V).

II. SYNAPTIC DEPRESSION AND FACILITATION IN AN IF NETWORK

Consider a homogeneous network of N globally coupled integrate-and-fire (IF) neurons. Let $U_j(t)$ denote the membrane potential of the jth neuron at time t with $j=1,\ldots,N$. Each neuron evolves according to the equation

$$\tau_m \frac{dU_j(t)}{dt} = I - U_j(t) + \frac{g}{N-1}\sum_{k\neq j} R_k(t), \qquad (2.1)$$

where τ_m is the membrane time constant, g is some global coupling constant, I is a constant external input, and $R_k(t)$ represents the post-synaptic response induced by the input spike train from the kth neuron. For convenience we fix the units of time by setting $\tau_m=1$; typically the membrane time constant is of the order 10 msec. The sign of g determines whether the network is excitatory ($g>0$) or inhibitory ($g<0$). Equation (2.1) is supplemented by the reset condition $U_j(t^+)=0$ whenever $U_j(t)=1$. Suppose that an isolated action potential evokes a post-synaptic potential (PSP) whose shape can be represented by an α function, $\alpha^2 t e^{-\alpha t}$. Let T_j^m, integer m, denote the mth firing time of the jth neuron, that is, $T_j^m = \inf\{t | U_j(t)\geq 1; t\geq T_j^{m-1}\}$. In the case of activity-independent synapses, the total response $R_k(t)$ at time t can be obtained by simply summing the responses arising from the individual spikes. Therefore, assuming that each spike takes a time τ_a to propagate along an axon connecting any two neurons, the total response is $R_k(t)=\Sigma_{m\in\mathbb{Z}} J(t-T_k^m)$, where

$$J(\tau) = \alpha^2(\tau-\tau_a)e^{-\alpha(\tau-\tau_a)}\Theta(\tau-\tau_a). \qquad (2.2)$$

Here $\Theta(\tau)=1$ if $\tau>0$ and is zero otherwise.

In order to incorporate the effects of dynamic synapses, we modify $R_k(t)$ along the lines of the phenomenological model considered in Refs. [2,4]. (See also the review of Abbott and Marder [23]). This essentially involves the introduction of an amplitude factor $C(T_k^m)$ that adjusts the magnitude of the single spike response at time T_k^m based on previous input history:

$$R_k(t)=\sum_{m\in\mathbb{Z}} C(T_k^m)J(t-T_k^m). \tag{2.3}$$

Following the arrival of a spike at a presynaptic terminal, C is increased in the case of facilitation and decreased in the case of depression. It is mathematically convenient to model the former as an additive process and the latter as a multiplicative process in order to avoid possible divergences (see below). That is, $C\to C+\gamma-1$ with $\gamma>1$ for facilitation, and $C\to\gamma C$ with $\gamma<1$ for depression. In between spikes, C is assumed to return to its equilibrium value of one according to the exponential process

$$\tau_c\frac{dC}{dt}=1-C, \tag{2.4}$$

where τ_c is an appropriately chosen time constant (τ_c can vary between around 100 msec and a few seconds [4]). For a given sequence of jumps at times $\{T_k^m, m\in\mathbb{Z}\}$, Eq. (2.4) can be solved iteratively for the amplitude $C(T_k^m)$. One finds that

$$C(T_k^m)=1+(\gamma-1)\sum_{m'<m}\hat{\gamma}^{m-m'-1}e^{-(T_k^m-T_k^{m'})/\tau_c}, \tag{2.5}$$

with

$$\hat{\gamma}=\gamma \ \text{(depression)}, \quad \hat{\gamma}=1 \ \text{(facilitation)}. \tag{2.6}$$

Suppose that we restrict our attention to phase-locked solutions of Eq. (2.1) in which every oscillator resets or fires with the same self-consistent period T [17,20,21]. The state of each oscillator is then characterized by a constant phase $\phi_k\in\mathbb{R}\backslash\mathbb{Z}$ such that the firing times are of the form

$$T_k^m=(m-\phi_k)T \tag{2.7}$$

for all $m\in\mathbb{Z}$ and $k=1,\ldots,N$. Under such an ansatz, the amplitude factor $C(T_k^m)$ in Eq. (2.3) reduces to its steady-state value $C_\infty(T)$ so that

$$R_k(t)=C_\infty(T)\sum_{m\in\mathbb{Z}}J[t-(m-\phi_k)T]. \tag{2.8}$$

The amplitude $C_\infty(T)$ is obtained by substituting Eq. (2.7) into Eqs. (2.5) and (2.6), and summing the resulting geometric series [23]:

$$C_\infty(T)=\frac{1+(\gamma-2)e^{-T/\tau_c}}{1-e^{-T/\tau_c}} \ \text{(facilitation)}, \tag{2.9}$$

$$C_\infty(T)=\frac{1-e^{-T/\tau_c}}{1-\gamma e^{-T/\tau_c}} \ \text{(depression)}. \tag{2.10}$$

Note that $C_\infty(T)\equiv 1$ in the case of activity-independent synapses ($\gamma=1$). It is clear from Eq. (2.9) that if $\gamma<1$ then $C_\infty(T)<0$ for a range of values of T, which reflects the possibility that the series (2.5) diverges. Hence, we do not use an additive model of synaptic depression. Similar comments concerning Eq. (2.10) precludes a multiplicative model of synaptic facilitation.

For a given set of phases $\Phi=(\phi_1,\ldots,\phi_N)$, substitute Eq. (2.8) into Eq. (2.1) and integrate over the interval $t\in(-T\phi_j, T-T\phi_j)$ using the reset condition $U_j(-\phi_j T)=0$ and $U_j(T-\phi_j T)=1$. This leads to the phase equation

$$1=I[1-e^{-T}]+g_N C_\infty(T)\sum_{k\neq j}K(\phi_k-\phi_j,T), \quad j=1,\ldots,N, \tag{2.11}$$

where $g_N=g/(N-1)$ and

$$K(\phi,T)=\sum_{m\in\mathbb{Z}}\int_0^T e^{t-T}J[t+(m+\phi)T]dt. \tag{2.12}$$

After choosing some reference oscillator, Eq. (2.11) determines $N-1$ relative phases and the collective period T.

It is clear from Eq. (2.11) that the presence of dynamic synapses does not alter the basic structure of phase-locked solutions of Eq. (2.1). The phase interaction function $K(\phi,T)$ is simply scaled by the steady-state amplitude $C_\infty(T)$, the main effect of which is to modify the collective period T. Therefore, just as in the case of activity-independent synapses where $C_\infty(T)\equiv 1$, the different classes of solution can be determined using group theoretic methods [24]. Of particular interest are the so-called maximally symmetric solutions for which Eq. (2.11) reduces to a single equation for the collective period T. The underlying symmetry of the system guarantees the existence of these solutions, assuming that a self-consistent T can be found. (This is a realization of the equivariant branching lemma [25]). In this paper we shall focus on the *synchronous* or *in-phase* solution, $\phi_j=\phi$ for all $j=1,\ldots,N$, and the *splay* or *rotating wave* states $\phi_j=\phi\pm j/N$. For these maximally symmetric solutions, Eq. (2.11) takes the form

$$1=I[1-e^{-T}]+g_N C_\infty(T)\times\sum_{k=1}^{N-1}\sum_{m\in\mathbb{Z}}\int_0^T e^{t-T}J[t+(m+k\chi/N)T]dt, \tag{2.13}$$

with $\chi=\pm 1$ corresponding to the splay states and $\chi=0$ corresponding to the in-phase state.

To illustrate the effects of synaptic depression/facilitation on the collective period of oscillations T, consider the large-N limit of Eq. (2.13) in the case of the splay state ($\chi=1$). Using Fourier/Laplace transforms, it can be shown that (see the Appendix)

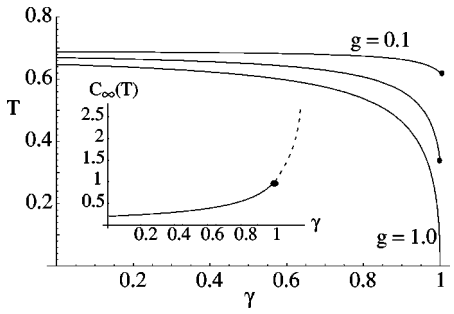

FIG. 1. Collective period T of a splay state in the large-N limit as a function of γ in the case of synaptic depression. Results are shown for $g=0.1,0.5,1.0$ and $I=2.0$. Inset: variation of $C_\infty(T)$ with γ for $g=0.1$ and $I=1.1$. Dashed portion of curve represents continuation into the facilitating regime ($\gamma>1$), which corresponds to the upper branch of Fig. 2 for $g=0.1$.

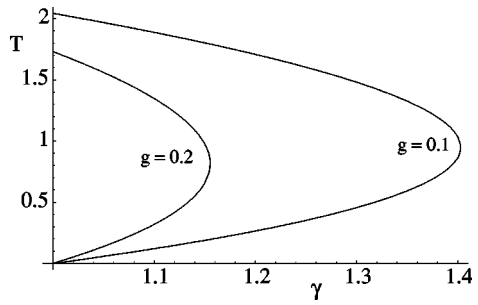

FIG. 2. Collective period T of a splay state in the large-N limit as a function of γ in the case of synaptic facilitation. Here $g=0.1,0.2$ and $I=1.1$. Beyond a critical value of γ there no longer exists a nonzero solution for T. For a given g, the upper branch is the continuation of the nontrivial activity-independent solution at $\gamma=1$.

$$\frac{1}{N-1}\sum_{k=1}^{N-1}\sum_{m\in\mathbb{Z}} J[t+(m+k/N)T] = \frac{1}{T}\left[\tilde{J}(0)-\frac{1}{N-1}\sum_{n\neq 0}\tilde{J}(2\pi i n/T)\right], \tag{2.14}$$

where $\tilde{J}(\lambda)$ is the Laplace transform of the delay kernel $J(t)$ of Eq. (2.2),

$$\tilde{J}(\lambda)=\frac{\alpha^2 e^{-\tau_a\lambda}}{(\alpha+\lambda)^2}. \tag{2.15}$$

Therefore, taking the large-N limit of Eq. (2.13) and noting that $\tilde{J}(0)=1$, we obtain the self-consistency equation

$$T=\ln\left[\frac{I+gC_\infty(T)/T}{I-1+gC_\infty(T)/T}\right]. \tag{2.16}$$

The dependence of the (unique) nontrivial solution of Eq. (2.16) as a function of the degree of synaptic depression is illustrated in Fig. 1 for $g>0$ [26]. (In all figures the variables are in dimensionless units obtained by taking $\tau_m=1$ and the firing threshold to be unity.) It can be seen that decreasing γ increases the collective period T, that is, depressive synapses reduce the mean firing rate in an excitatory network. On the other hand, facilitating synapses increase the firing rate as shown in Fig. 2. (The effects of synaptic depression and facilitation on T are reversed for inhibitory networks.) Interestingly, it can be seen from Fig. 2 that for fixed positive coupling g there exists a critical value $\gamma_c>1$ such that if $1<\gamma<\gamma_c$ then there exist two nontrivial solution branches for T, whereas there are no nontrivial solutions when $\gamma>\gamma_c$. The upper branch for a given g and $1<\gamma<\gamma_c$ is the continuation from the activity-independent case and, hence, we shall focus on the stability properties of this solution in subsequent sections rather than the lower branch. Finally, note that the collective period tends to depend only weakly on the size of the network N.

III. MEAN-FIELD THEORY

One method for studying the dynamics of a large globally coupled network is to reformulate the dynamics as a continuity equation describing a flow of phases [8,9]. An alternative approach [10–12], which we shall follow here, is to construct a mean-field equation for the population activity

$$A(t)=\lim_{N\to\infty}\frac{1}{N}\sum_{j=1}^{N}\sum_{m\in\mathbb{Z}}\delta(t-T_j^m). \tag{3.1}$$

Here $A(t)\Delta t$ determines the fraction of neurons firing in the small interval of time Δt. In the mean-field limit all oscillators have the same synaptic input $R(t)$,

$$R(t)=\int_0^\infty J(\tau)X(t-\tau)d\tau, \tag{3.2}$$

where $X(t)$ is an additional macroscopic variable [see Eqs. (2.1) and (2.3)],

$$X(t)=\lim_{N\to\infty}\frac{1}{N}\sum_{j=1}^{N}\sum_{m\in\mathbb{Z}}C(T_j^m)\delta(t-T_j^m). \tag{3.3}$$

In the case of activity-independent synapses, $X(t)$ reduces to $A(t)$.

Suppose that if an oscillator last fired at time $\hat{t}$, then it fires again with probability one at time $t=\hat{t}+T(\hat{t})$. It follows that in the mean-field limit, the activity $A(t)$ satisfies the integral equation [12]

$$A(t)=\int_{-\infty}^{t}\delta[t-\hat{t}-T(\hat{t})]A(\hat{t})d\hat{t}=\left[1+\frac{dT}{d\hat{t}}\right]^{-1}A(t-T). \tag{3.4}$$

In order to obtain a closed system of equations, it is first necessary to express the function $T(\hat{t})$ in terms of the mean field $R(t)$. Let us solve the IF equation (2.1) in the mean-field limit for successive firing times $\hat{t}$ and $\hat{t}+T$. This leads to the implicit equation

$$1=I[1-e^{-T}]+g\int_0^T e^{s-T}R(s+\hat{t})ds. \tag{3.5}$$

Differentiating both sides of Eq. (3.5) with respect to $\hat{t}$ then gives

$$\frac{dT}{d\hat{t}} = -\frac{g\int_0^T e^{s-T}R'(s+\hat{t})ds}{I-1+gR(\hat{t}+T)}. \tag{3.6}$$

In the case of activity-independent synapses, (3.4) and (3.6) form a closed system of equations, since $R(t)=\int_0^\infty J(\tau)A(t-\tau)d\tau$. Unfortunately, this is no longer true for dynamic synapses, since $R(t)$ then satisfies Eq. (3.2) with the macroscopic dynamics of $X(t)$ still undetermined. Constructing a dynamical mean-field equation for $X(t)$ does not appear possible unless additional approximations are made. Here we shall work within a linear approximation scheme, which is used to analyze the stability of the splay state.

In the mean-field limit the splay state is a state with time-independent activity for which $A(t)=A_0\equiv 1/T$ and $X(t)=X_0\equiv C_\infty(T)A_0$, where T is the solution to the self-consistency equation (2.16). Consider perturbations about the splay state of the form

$$a(t)\equiv A(t)-A_0=\tilde{a}(\lambda)e^{\lambda t},\quad x(t)\equiv X(t)-X_0=\tilde{x}(\lambda)e^{\lambda t}, \tag{3.7}$$

where $\lambda\in\mathbb{C}$. Substituting (3.7) into (3.2) implies that $R(t)=X_0+e^{\lambda t}\tilde{J}(\lambda)\tilde{x}(\lambda)$ and $R'(t)=e^{\lambda t}\lambda\tilde{J}(\lambda)\tilde{x}(\lambda)$, where $\tilde{J}(\lambda)$ is the Laplace transform (2.15). Substituting Eq. (3.6) into (3.4) and expanding to first order in $\tilde{a}(\lambda)$ and $\tilde{x}(\lambda)$ then gives

$$\tilde{a}(\lambda)[e^{\lambda T}-1]=gA_0\tilde{x}(\lambda)\frac{\lambda\tilde{J}(\lambda)}{[1+\lambda]}[e^{\lambda T}-e^{-T}][e^T-1]. \tag{3.8}$$

We have used the result that $I-1+gA_0C_\infty(T)=[e^T-1]^{-1}$ [see Eq. (2.16)].

It remains to derive an expression for $\tilde{x}(\lambda)$ in terms of $\tilde{a}(\lambda)$. This will be accomplished by linearizing Eqs. (3.1) and (3.3) about the splay state, and using this to construct a linear differential equation for $x(t)$ in terms of $a(t)$. In order to carry out this linearization procedure, it is necessary to consider perturbations of the individual firing times (see Sec. V). Let $\hat{T}_k^m=(m+k/N)T$ denote the firing times of the splay state and consider the perturbed state $T_k^m=\hat{T}_k^m+u_k^m$ with $u_k^m=a_ke^{m\lambda T}$. Expanding Eq. (3.3) to first order in a_k using Eq. (2.5) yields the linear equation

$$x(t)\approx C_\infty(T)\Bigg[a(t)-(\gamma-1)\Gamma(\lambda T)\lim_{N\to\infty}\frac{1}{N}\times\sum_{k=1}^{N}\sum_{m\in\mathbb{Z}}e^{m\lambda T}a_k\delta(t-\hat{T}_k^m)\Bigg], \tag{3.9}$$

where

$$\Gamma(\lambda)=\sum_{m'<m}\hat{\gamma}^{m-m'-1}e^{-(m-m')T/\tau_c}(1-e^{-(m-m')\lambda})=\left[\frac{e^{-T/\tau_c}}{1-\hat{\gamma}e^{-T/\tau_c}}-\frac{e^{-T/\tau_c-\lambda}}{1-\hat{\gamma}e^{-T/\tau_c-\lambda}}\right]. \tag{3.10}$$

Similarly, expanding Eq. (3.1) gives

$$a(t)\approx-\lim_{N\to\infty}\frac{1}{N}\sum_{k=1}^{N}\sum_{m\in\mathbb{Z}}e^{m\lambda T}a_k\delta'(t-\hat{T}_k^m). \tag{3.11}$$

Hence, comparison of Eqs. (3.9) and (3.11) leads to the linear differential equation [valid to first order in $a(t)$]

$$\frac{dx(t)}{dt}=C_\infty(T)\left[\frac{da}{dt}+(\gamma-1)\Gamma(\lambda T)a(t)\right]. \tag{3.12}$$

[More precisely, this relationship between the two distributions $a(t)$ and $x(t)$ should be formulated in terms of integrals $\int_{-\infty}^{\infty}f(t)a(t)dt$ and $\int_{-\infty}^{\infty}f(t)a(t)dt$ for an arbitrary smooth function $f(t)$ such that $\int_{-\infty}^{\infty}f(t)dt<\infty$.] Substituting Eq. (3.7) into Eq. (3.12) yields the result

$$\tilde{x}(\lambda)=C_\infty(T)\tilde{a}(\lambda)\left[1+\frac{(\gamma-1)}{\lambda}\Gamma(\lambda T)\right]. \tag{3.13}$$

Finally, combining Eqs. (3.8) and (3.13), we obtain the characteristic equation

$$(e^{\lambda T}-1)=g\Lambda(T)[\lambda+(\gamma-1)\Gamma(\lambda T)]\frac{\tilde{J}(\lambda)}{1+\lambda}(e^{\lambda T}-e^{-T}), \tag{3.14}$$

where $\Lambda(T)=\{[C_\infty(T)]/T\}(e^T-1)$.

Note that there are two major γ-dependent contributions to Eq. (3.14) for a given T. First, there is a static contribution associated with a simple rescaling of the coupling according to $g\to C_\infty(T)g$. Second, there is a dynamic contribution represented by the term $(\gamma-1)\Gamma(\lambda T)$ in Eq. (3.14). Although the static contribution accounts for the qualitative nature of the effect of synaptic depression/facilitation on stability as described below, it underestimates the size of this effect.

In the weak-coupling regime, solutions of Eq. (3.14) are of the form $\lambda T=2\pi in+\Lambda_n$ for integer n and $\Lambda_n=O(g)$. The term Λ_n can be calculated by performing a perturbation expansion in the coupling g. The lowest order contribution is simply determined by setting $\lambda T=2\pi in$ on the right-hand side of Eq. (3.14):

$$\Lambda_n=g\Lambda(T)(1-e^{-T})\left(\frac{2\pi in}{T+2\pi in}\right)\tilde{J}(2\pi in/T)+O(g^2). \tag{3.15}$$

It follows from Eq. (3.15) that dynamic synapses do not alter the weak-coupling stability of a splay state other than indirectly through a modification of its collective period T (see Figs. 1 and 2). Therefore, we can apply the stability results previously obtained for activity-independent synapses [9,11,12].

(1) For zero axonal delays ($\tau_a=0$) and excitatory coupling ($g>0$), the splay state is stable with respect to excitation of the nth mode if and only if $\alpha<\alpha_n$, where

$$\alpha_n=-1+\sqrt{1+4n^2\pi^2/T^2}. \tag{3.16}$$

Hence, it is stable for sufficiently slow synapses, that is, $\alpha<\alpha_1$. The splay state is always unstable in the case of in-

hibitory coupling, since the condition for stability with respect to the nth harmonic is now $\alpha > \alpha_n$, which cannot be satisfied for all n.

(2) The splay state is almost always unstable for nonzero delays (in the noise-free case).

(3) For large n, $|\Lambda_n| \sim 1/n^2$ so that higher harmonics grow or decay slowly.

Note that although the zero delay case is a singular limit in the absence of noise, it becomes nonsingular for arbitrarily small amounts of noise, where instabilities with respect to higher harmonics are suppressed (see Refs. [9,11,12] and Sec. IV). Finite-size effects play a similar role, for, as will be shown in Sec. V, Eq. (3.14) still holds for finite N except that n is now restricted to values in the range $0 \leqslant n \leqslant N-1$ [and g is scaled by a factor $N/(N-1)$].

A numerical investigation of the zero delay case with activity-independent synapses and excitatory coupling shows that increasing g can stabilize the splay state for values of $\alpha > \alpha_1$ [9]. This occurs due to eigenvalues associated with low order harmonics crossing over into the left-half complex plane. We shall investigate how this result depends on γ. Set $\lambda T = i\beta, \beta \in \mathbb{R}$ in Eq. (3.14) and equate real and imaginary parts to obtain the pair of equations

$$\cos(\beta) - 1 = g\Lambda(T)\Big((\gamma-1) q_0(\beta) P_0(\beta) - \Big[\frac{\beta}{T} + (\gamma-1) q_1(\beta)\Big] P_1(\beta)\Big), \quad (3.17)$$

$$\sin(\beta) = g\Lambda(T)\Big(\Big[\frac{\beta}{T} + (\gamma-1) q_1(\beta)\Big] P_0(\beta) + (\gamma-1) q_0(\beta) P_1(\beta)\Big), \quad (3.18)$$

where $q_0(\beta) = \mathrm{Re}\,\Gamma(i\beta), q_1(\beta) = \mathrm{Im}\,\Gamma(i\beta)$,

$$P_0(\beta) = [\cos(\beta) - e^{-T}] p_0(\beta) - \sin(\beta) p_1(\beta),$$

$$P_1(\beta) = \sin(\beta) p_0(\beta) + [\cos(\beta) - e^{-T}] p_1(\beta),$$

and $p_0(\beta) = \mathrm{Re}[\tilde{J}(i\beta/T)/(1+i\beta/T)]$, $p_1(\beta) = \mathrm{Im}[\tilde{J}(i\beta/T)/(1+i\beta/T)]$. For a given coupling g, we search for the smallest α for which a nonzero solution β of Eqs. (3.17) and (3.18) exists. The results are shown in Fig. 3 for synaptic depression. It can be seen that increasing the degree of synaptic depression (by reducing γ) leads to a reduction in the critical inverse rise time for destabilization of the splay state. In other words, synaptic depression decreases the region in the (g, α^{-1}) plane over which the splay state is stable. The γ-dependent shift in the stability curves can be understood qualitatively in terms of the static rescaling of the coupling $g \to gC_\infty(T)$. Since $C_\infty(T) < 1$ for synaptic depression (see inset of Fig. 1), there is an effective reduction in the coupling that results in destabilization. This effect is further enhanced by dynamic contributions [associated with the term $(\gamma - 1)\Gamma(\lambda T)$ in Eq. (3.14)]. On the other hand, synaptic facilitation has a stabilizing effect in the sense that it enlarges the region of stability as shown in Fig. 4. This is qualitatively consistent with an effective increase in the coupling $g \to gC_\infty(T)$ with $C_\infty(T) > 1$ for synaptic facilitation.

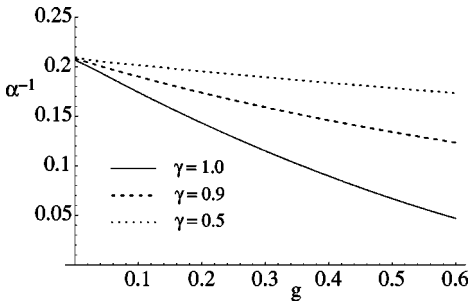

FIG. 3. Destabilizing effect of synaptic depression in an excitatory network with zero axonal delays and finite rise time α^{-1}. The boundary curve separating stable and unstable regions of the splay state is shown for various values of γ and fixed external input $I = 1.5$. Stability holds above each boundary curve.

IV. NOISE

One of the powerful features of the MFT approach to population dynamics is that it provides an analytically tractable framework for incorporating the effects of noise, which can be achieved through a generalization of the activity integral equation (3.4) [12,27]. Suppose for simplicity that the dynamics is described by a renewal process. That is, there exists a conditional probability density $P_X(t|\hat{t})$ such that $P_X(t|\hat{t})\delta t$ is the probability of firing in the interval $[t, t+\delta t]$ given that the last spike occurred at $\hat{t}$. The subscript X indicates that the probability density depends on the time course of the mean field $X(t')$ [Eq. (3.3)] for $t' < t$. The integral equation (3.4) for the population activity $A(t)$ now becomes

$$A(t) = \int_{-\infty}^{t} P_X(t|\hat{t}) A(\hat{t}) d\hat{t}, \quad (4.1)$$

with A appropriately normalized [27].

There are various ways of introducing noise into an IF network, including threshold noise, reset noise, and input noise [27]. Here we shall consider a phenomenological approach in which additive noise is introduced directly into the

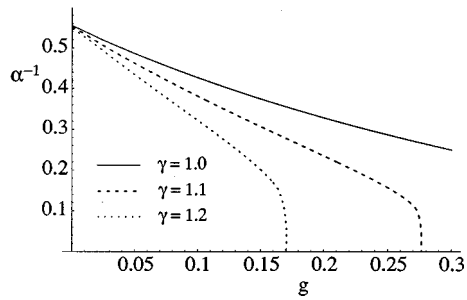

FIG. 4. Stabilizing effect of synaptic facilitation in an excitatory network with zero axonal delays and finite rise-time α^{-1}. The splay state with the largest collective period is selected (see Fig. 2). The boundary curve separating stable and unstable regions of the splay state is shown for various values of γ and fixed external input $I = 1.1$. Stability holds above each boundary curve.

firing times. First, solve Eq. (2.1) in the mean-field limit for a sequence of firing times $\{T_j^n, n \in \mathbb{Z}\}$. The resulting iterative equation for the firing times can be written in the form

$$e^{T_j^{n+1}}[I-1+gY(T_j^{n+1})]=e^{T_j^n}[I+gY(T_j^n)], \quad (4.2)$$

where

$$Y(t)=\int_0^\infty \hat{J}(\tau)X(t-\tau)d\tau, \quad \hat{J}(\tau)=\int_0^\tau e^{s-\tau}J(s)ds. \quad (4.3)$$

This leads to the following implicit equation for T_j^{n+1} as a function of T_j^n:

$$T_j^{n+1}=T_j^n+H(T_j^n,T_j^{n+1}), \quad (4.4)$$

where

$$H(t,t')=\ln\left[\frac{gY(t)+I}{gY(t')+I-1}\right]. \quad (4.5)$$

A stochastic IF model is now introduced by assuming that the firing times evolve according to the additive process

$$T_j^{n+1}=T_j^n+H(T_j^n,T_j^{n+1})+\xi_j^n, \quad (4.6)$$

where ξ_j^n, for integer n and $j=1,\ldots,N$, are independent random variables generated from a given probability density ρ. We shall assume that the width of the probability distribution is sufficiently narrow that the domain of ρ can be taken to be the whole real line. A further simplification can be obtained by taking $Y(t)$ to be a sufficiently slow function of time so that $H(T_j^n,T_j^{n+1})\approx H(T_j^n,T_j^n+\Delta T_j^n)$ with $\Delta T_j^n = H(T_j^n,T_j^n)$, which is uncorrelated with ξ_j^n. Under this approximation, Eq. (4.6) describes a renewal process with conditional probability density

$$P_X(t|\hat{t})=\rho(t-H(\hat{t},t^*)-\hat{t}), \quad (4.7)$$

where $t^*=\hat{t}+H(\hat{t},\hat{t})$. We shall use Eq. (4.1) and the conditional probability density (4.7) to investigate how noise can affect the stability of the splay state.

As in the noise-free case, define the splay state as a time-independent state $A(t)=A_0$ and $X(t)=X_0$. It follows from Eq. (4.6) that the firing times of the splay state (denoted by $\hat{T}_j^n$) evolve according to the simplified equation

$$\hat{T}_j^{n+1}=\hat{T}_j^n+H(X_0)+\xi_j^n, \quad (4.8)$$

with

$$H(X)=\ln\left[\frac{gX+I}{gX+I-1}\right]. \quad (4.9)$$

The activity A_0 is equal to the inverse of the mean interspike interval, that is,

$$\frac{1}{A_0}\equiv T=\int \xi\rho(\xi-H(X_0))d\xi=H(X_0)+\overline{\xi}. \quad (4.10)$$

For convenience we shall take $\overline{\xi}=0$. The constant field X_0 is obtained from Eq. (3.3) as

$$X_0=\sum_{m\in\mathbb{Z}}\langle C(\hat{T}^m)\delta(t-\hat{T}^m)\rangle, \quad (4.11)$$

where $\langle C(\hat{T}^m)\rangle=\lim_{N\to\infty}\Sigma_{i=1}^N C(\hat{T}_i^m)/N$ etc. For self-consistency, we require that the right-hand side of Eq. (4.11) is t independent. One way to ensure this is to assume that in the large-N limit the following approximation holds:

$$X_0\approx\sum_{m\in\mathbb{Z}}\langle C(\hat{T}^m)\rangle\langle\delta(t-\hat{T}^m)\rangle=\overline{C}(T)A_0, \quad (4.12)$$

where (for synaptic depression)

$$\overline{C}(T)=1+(\gamma-1)\sum_{m'<m}\gamma^{m-m'-1}\langle e^{-(\hat{T}^m-\hat{T}^{m'})/\tau_c}\rangle$$
$$=1+(\gamma-1)\sum_{m'<m}\gamma^{m-m'-1}e^{-(m-m')T/\tau_c}\langle e^{-(\xi^m+\xi^{m-1}+\ldots+\xi^{m'})/\tau_c}\rangle=\frac{1-\kappa e^{-T/\tau_c}}{1-\gamma\kappa e^{-T/\tau_c}}. \quad (4.13)$$

We have used the fact that the ξ_i^n are uncorrelated, so that $\langle e^{-(\xi^m+\xi^{m-1}+\ldots+\xi^{m'})/\tau_c}\rangle=\kappa^{m-m'}$ with $\kappa=\overline{e^{-\xi/\tau_c}}$. A result similar to Eq. (4.13) holds for synaptic facilitation:

$$\overline{C}(T)=\frac{1+(\gamma-2)\kappa e^{-T/\tau_c}}{1-\kappa e^{-T/\tau_c}}. \quad (4.14)$$

It follows from Eqs. (4.10) and (4.12) that the collective period of oscillations satisfies Eq. (2.16) with $C_\infty(T)$ replaced by $\overline{C}(T)$.

In order to determine the stability of the splay state in the presence of noise, consider perturbations of the form (3.7). Linearization of the integral equation (4.1) about the splay state gives

$$\tilde{a}(\lambda)[1-\tilde{\rho}_0(\lambda)]=gA_0\frac{\lambda\tilde{x}(\lambda)\tilde{J}(\lambda)}{1+\lambda}[e^T-1]$$
$$\times[e^{\lambda T}-e^{-T}]\tilde{\rho}_0(\lambda), \quad (4.15)$$

where

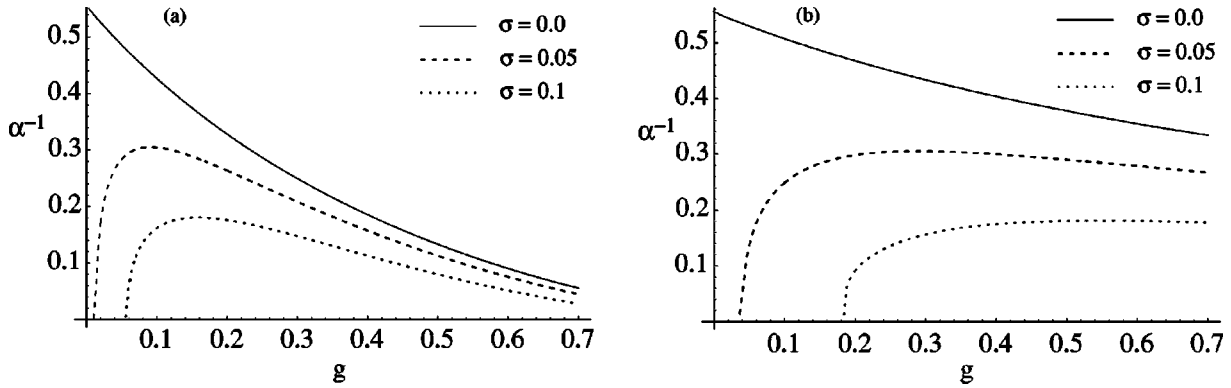

FIG. 5. Stability of the splay state as a function of excitatory coupling g and rise time α^{-1} in the presence of synaptic depression and noise. The boundary curve above which the splay state becomes stable is shown for $I=1.1$, $\tau_a=0$, and various values of the standard deviation σ. (a) $\gamma=1$ (activity-independent synapses). (b) $\gamma=0.5$ (synaptic depression).

$$\tilde{\rho}_0(\lambda)=\int_0^\infty \rho[s-H(A_0)]e^{-s\lambda}ds. \tag{4.16}$$

Following arguments similar to the deterministic case (Sec. III), it can be shown that $a(t)$ and $x(t)$ are related according to the linear equation (3.12) under the replacements $C_\infty(T)\to\overline{C}(T)$ and $\Gamma(\lambda)\to\overline{\Gamma}(\lambda)$, with $\overline{C}(T)$ satisfying Eq. (4.13) or (4.14) and

$$\overline{\Gamma}(\lambda)=\frac{\kappa e^{-T/\tau_c}}{1-\hat{\gamma}\kappa e^{-T/\tau_c}}-\frac{\kappa e^{-T/\tau_c-\lambda}}{1-\hat{\gamma}\kappa e^{-T/\tau_c-\lambda}}. \tag{4.17}$$

[We are again assuming that the approximation (4.12) is valid.] We conclude that in the presence of noise, the characteristic equation for the splay state takes the form

$$1-\tilde{\rho}_0(\lambda)=g\overline{\Lambda}(T)[\lambda+(\gamma-1)\overline{\Gamma}(\lambda T)] \times\frac{\tilde{J}(\lambda)}{1+\lambda}(e^{\lambda T}-e^{-T})\tilde{\rho}_0(\lambda), \tag{4.18}$$

where $\overline{\Gamma}(\lambda)=[\overline{C}(T)/T](e^T-1)$. In the deterministic limit $\tilde{\rho}_0(\lambda)\to e^{-\lambda T}$ with $A_0=1/T$ and T satisfying Eq. (2.16), Eq. (4.18) reduces to Eq. (3.14).

It is clear from Eq. (4.18) that in the weak-coupling regime, solutions λ must have negative real part in order for the left-hand side of (4.18) to be $O(g)$. Therefore, we expect the stability of the splay state to persist to arbitrarily large values of α when g is sufficiently weak. Moreover, since the modulus of the right-hand side vanishes when $|\lambda|\to\infty$ it follows that high order harmonics are suppressed. Consequently, the critical value of α for destabilization of an excitatory network with zero axonal delays and intermediate or strong coupling g should increase with the level of noise. This is indeed found to be the case, both for activity-independent synapses [see Fig. 5(a) and Refs. [9,11]] and dynamic synapses [see Fig. 5(b)]. In the construction of Fig. 5 (and subsequent figures) we have taken $\rho(\xi)=e^{-\xi^2/2\sigma^2}$ with standard deviation $\sigma\ll T$ so that $\tilde{\rho}(\lambda)\approx e^{-\lambda T+\lambda^2\sigma^2/2}$ and $\kappa=e^{\sigma^2/2\tau_c^2}$. Another important consequence of noise is that it can stabilize the splay state in an inhibitory network by suppressing higher harmonics [9]. This is illustrated in Figs. 6 and 7 where we plot the stability boundary curves for the first two harmonics as a function of α and $|g|$ with $\tau_a=0$. It can be seen that noise reduces the region of instability of these modes. Such an effect increases with the order n so that the splay state is stable in the region outside the boundary curves of the low harmonics. In particular, the splay state is stable for all α when the coupling is sufficiently weak. Interestingly, in the presence of noise, synaptic depression can actually have a stabilizing effect provided that the coupling

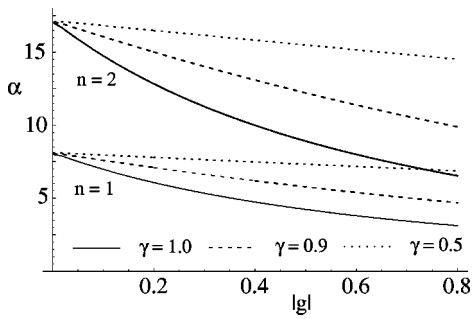

FIG. 6. Stability of the splay state as a function of inhibitory coupling $|g|$ and inverse rise time α for synaptic depression without noise. The stability boundary curves for the first two harmonics $n=1,2$ are shown for $I=2.0$, $\tau_a=0$ and various values of γ. A mode is stable above its boundary curve.

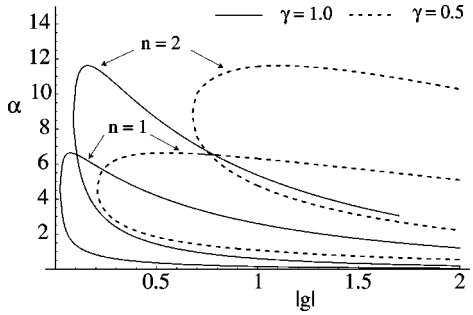

FIG. 7. Stability of the splay state as a function of inhibitory coupling $|g|$ and inverse rise time α in the presence of synaptic depression and noise ($\sigma=0.01$). The stability boundary curves for the first two harmonics $n=1,2$ are shown for activity-independent synapses (solid lines) and depressive synapses with $\gamma=0.5$ (dashed lines). Here $I=2.0$ and $\tau_a=0$. A mode is stable outside its boundary curve.

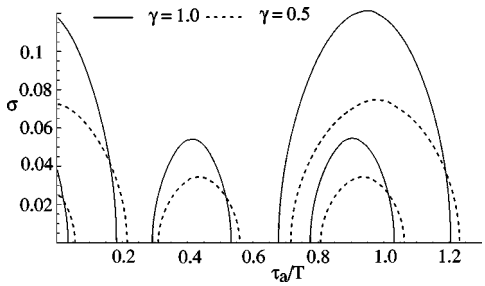

FIG. 8. Stability of the splay state as a function of axonal delay τ_a and noise σ for an excitatory network. The stability boundary curves for the first two harmonics $n=1,2$ are shown for activity-independent synapses (solid lines) and depressive synapses with $\gamma=0.5$ (dashed lines). For each γ the single high peak corresponds to $n=1$ and the pair of lower peaks corresponds to $n=2$. The delay τ_a has been scaled by the collective period T (which is approximately independent of σ and τ for weak coupling); the stability diagram is periodic with respect to T. We have taken $I=1.1$, $\alpha=10$, and $g=0.1$. A mode is stable outside its boundary curve.

is not too large. Indeed, Figs. 5(b) and 7 show that the stability boundary curves are shifted over to larger values of $|g|$ when γ is reduced from unity. An analogous result is found in excitatory networks with nonzero axonal delays as illustrated in Fig. 8. We plot the boundary curves of the first two harmonics as a function of σ and τ_a for fixed α and g. The region of stability outside the boundary curves of the lower harmonics is enlarged by depressive synapses. As in the noise-free case, these results can be understood qualitatively in terms of rescaling of the coupling according to $g \to gC_\infty(T)$.

V. FINITE NETWORKS

In this section we analyze the stability of the splay and in-phase states of a globally coupled IF network directly in terms of the firing times. This will be used to determine how the results of mean-field theory are modified for finite networks (in the absence of noise). Following along lines similar to Refs. [20,21], integrate Eq. (2.1) from T_j^n to T_j^{n+1} to generate the nonlinear firing time map

$$e^{T_j^{n+1}} = I[e^{T_j^{n+1}} - e^{T_j^n}] + g_N \sum_{k\neq j} \sum_{m\in\mathbb{Z}} C(T_k^m) \times \left[\int_{T_j^n}^{T_j^{n+1}} e^t J(t - T_k^m)\, dt \right]. \tag{5.1}$$

Set $T_j^n = (n + j\chi/N)T + u_j^n$, where u_j^n represents a perturbation of the splay ($\chi=\pm 1$) or in-phase ($\chi=0$) states, and expand Eq. (5.1) as a power series in the perturbations u_j^n. To $O(1)$ we recover Eq. (2.11) for the collective period T, whereas the $O(u)$ terms lead to an infinite-order linear difference equation given by

$$A_N[u_j^{n+1} - u_j^n] = g_N \sum_{k\neq j} \sum_{m\in\mathbb{Z}} B_1[n-m+(j-k)\chi/N] \times [u_k^m - u_j^n] + (\gamma-1) g_N \times \sum_{k\neq j} \sum_{m\in\mathbb{Z}} B_0[n-m+(j-k)\chi/N]\, \delta_k^m[u], \tag{5.2}$$

where

$$A_N = I - 1 + g_N C_\infty(T) \sum_{k=1}^{N-1} \sum_{m\in\mathbb{Z}} J([m+k\chi/N]T), \tag{5.3}$$

$$B_0(\phi) = C_\infty(T) \int_0^T e^{t-T} J(t+\phi T)\, dt, \quad B_1(\phi) = \frac{1}{T} \frac{dB_0(\phi)}{d\phi}, \tag{5.4}$$

and

$$\delta_k^m[u] = \sum_{m'<m} \hat{\gamma}^{m-m'-1} e^{-(m-m')T/\tau_c} [u_k^m - u_k^{m'}], \tag{5.5}$$

with $\hat{\gamma}$ defined by Eq. (2.6). Note that $B_r(\phi)=0$ for $r=0,1$ and $\phi<-1$ so that Eq. (5.2) does not violate causality.

The linear map (5.2) has a discrete spectrum that can be found by taking

$$u_k^m = e^{m\lambda} a_k, \quad a_k = e^{k(\lambda\chi + 2\pi i p)/N} \tag{5.6}$$

with $\lambda \in \mathbb{C}$, $0 \leq \mathrm{Im}\,\lambda < 2\pi$, and $p=0,\ldots,N-1$. This generates the characteristic equation

$$A_N[e^\lambda - 1] = g[\tilde{B}_{1N}(\lambda,p) - \tilde{B}_{1N}(0,0) + (\gamma-1)\tilde{B}_{0N}(\lambda,p)\Gamma(\lambda)], \tag{5.7}$$

where

$$\tilde{B}_{rN}(\lambda,p) = \frac{1}{N-1} \sum_{k=1}^{N-1} \sum_{m\in\mathbb{Z}} B_r(m+k\chi/N) \times e^{-(m+k\chi/N)\lambda} e^{-2\pi i p k/N} \tag{5.8}$$

for $r=0,1$ and $\Gamma(\lambda)$ is defined according to Eq. (3.10). Note that $B_{rN}(\lambda,p)$ and $\Gamma(\lambda)$ are analytic functions of λ in the right-half complex λ plane, but have a countable number of poles in the left-half plane. This can be seen explicitly in the case of $\Gamma(\lambda)$, Eq. (3.10), which has poles at $\lambda = -[T + |\ln(\hat{\gamma})|] + 2\pi i n, n\in\mathbb{Z}$, arising from the analytic continuation of the geometric series. The semianalyticity of $\tilde{B}_{rN}$ reflects causality. One solution of Eq. (5.7) is $\lambda=0, p=0$, which reflects invariance of the dynamics with respect to uniform phase shifts of the firing times, $T_j^m \to T_j^m + u$ for all j,m. Therefore, the condition for linear stability of a splay or in-phase state is that all remaining solutions of Eq. (5.7) satisfy $\mathrm{Re}\ \lambda < 0$.

Let us now consider the splay state by setting $\chi=1$. Using the Appendix, we can rewrite Eqs. (5.3) and (5.8) as

$$A_N = I - 1 + g \frac{C_\infty(T)}{T} \left[\tilde{J}(0) - \frac{1}{N-1} \sum_{n\neq 0} \tilde{J}(2\pi i n/T) \right], \tag{5.9}$$

where $\tilde{J}(\lambda)$ is the Laplace transform (2.15), and

$$\tilde{B}_{rN}(\lambda,p) = \tilde{B}_r(\lambda + 2\pi i p) - \frac{1}{N-1} \sum_{n\neq 0} \tilde{B}_r(\lambda + 2\pi i [p+n]), \tag{5.10}$$

with

$$\tilde{B}_r(\lambda) \equiv \int_{-\infty}^{\infty} e^{-\lambda\phi} B_r(\phi)\, d\phi = \frac{C_\infty(T)}{T}(e^{\lambda} - e^{-T}) \frac{[\lambda/T]^r}{1+\lambda/T}\, \tilde{J}(\lambda/T) \quad (5.11)$$

and $B_r(\phi)$ defined by Eq. (5.4). Substitute Eqs. (5.9) and (5.10) into the characteristic equation (5.7) and take the large-N limit. This generates the characteristic equation

$$(e^{\lambda}-1)\left(I - 1 + g\frac{C_\infty(T)}{T}\right) = g[\tilde{B}_1(\lambda + 2\pi i p) - \tilde{B}_1(0) + (\gamma - 1)\tilde{B}_0(\lambda + 2\pi i p)\Gamma(\lambda)], \quad (5.12)$$

where $p \in \mathbb{Z}$. Recall that $0 \leqslant \mathrm{Im}\,\lambda < 2\pi$. Therefore, in Eq. (5.12) we can absorb $2\pi i p$ into the definition of λ by extending the domain of λ to the whole complex plane. After substituting for $\tilde{B}_r$ using Eq. (5.11) and performing a rescaling $\lambda \to \lambda T$, we recover the mean-field characteristic equation (3.14).

For finite N, the modifications to the characteristic equation (5.12) can be deduced from Eq. (5.9) and (5.10). We shall illustrate this in the case of weak coupling. For sufficiently small $|g|$, all solutions of Eq. (5.7) in the complex λ plane will be either in a neighborhood of the real solution $\lambda = 0$ or in a neighborhood of one of the poles of $\tilde{B}_{rN}(\lambda, p), \Gamma(\lambda)$. Since the latter all have negative real parts, the stability of phase-locked solutions will be determined by the eigenvalues around the origin. Therefore, expanding Eq. (5.7) in powers of λ and using Eq. (5.3) shows that

$$\lambda[I-1] = g[\tilde{B}_{1N}(0,p) - \tilde{B}_{1N}(0,0)] + O(g^2). \quad (5.13)$$

Using the fact that $\tilde{B}_{1N}(0,p) - \tilde{B}_{1N}(0,0) = N\tilde{B}_1(2\pi i p)/(N-1)$ when $\chi = 1$ [see Eq. (5.10)], it follows that Eq. (5.13) reduces to Eq. (3.15) with $0 \leqslant n \leqslant N-1$ and $g \to Ng/(N-1)$. This also implies that higher harmonics are suppressed in finite networks. Consequently, for inhibition this confirms the result of Chow [22] that the splay state can be stable for finite N, even though in the mean-field limit the asynchronous state is unstable.

VI. IN-PHASE STATE

So far we have focused on how dynamic synapses affect the existence and stability of the splay state. In this final section we briefly discuss some results concerning the synchronous or in-phase state. The linearized map of the firing times for this state is given by Eq. (5.2) with $\chi = 0$. For large N, it can be rewritten in the form

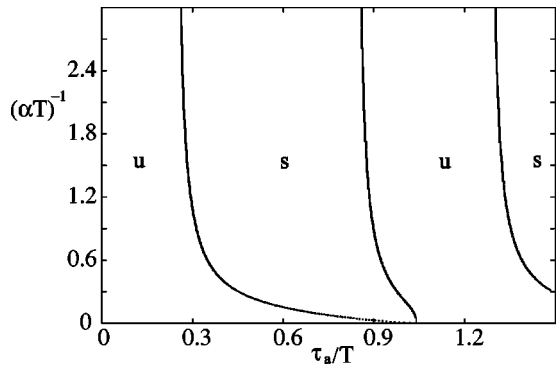

FIG. 9. Stability of the in-phase state $\phi = 0$ as a function of the dimensionless variables $[\alpha T]^{-1}$ and τ_a/T for weak excitatory coupling. Stable and unstable regions are denoted by s and u, respectively. The stability diagrams are periodic in τ_a with period T.

$$A[u_j^{n+1} - u_j^n] = g\sum_{m\in\mathbb{Z}} B_1(n-m)[\langle u^m\rangle - u_j^n] + g(\gamma-1) \times \sum_{m\in\mathbb{Z}} B_0(n-m) \sum_{m'<m} \Gamma_{mm'}[\langle u^m\rangle - \langle u^{m'}\rangle], \quad (6.1)$$

with $\Gamma_{mm'} = \hat{\gamma}^{m-m'-1} e^{-(m-m')T}$, $A = I - 1 + g\Sigma_{m\in\mathbb{Z}} J(mT)$, and

$$\langle u^m\rangle = \lim_{N\to\infty} \frac{1}{N}\sum_{j=1}^{N} u_j^m. \quad (6.2)$$

Following Ref. [18], we appeal to the law of large numbers and assume that for large N the mean perturbation $\langle u^m\rangle \approx 0$ for all m. Equation (6.1) then simplifies to the one-dimensional, first-order mapping

$$u_j^{n+1} = \left[1 - \frac{gC_\infty(T)K'(0,T)}{A}\right] u_j^n \equiv \beta_T u_j^n. \quad (6.3)$$

Since $C_\infty(T) > 0$ and $A > 0$, equation (6.3) implies that the in-phase state will be stable in the large-N limit if $|\beta_T| < 1$, that is, if $gK'(0,T) > 0$. This is a version of the mode-locking theorem of Gerstner, van Hemmen, and Cowan, [18], which we have shown extends to the case of a globally coupled IF network with dynamic synapses. One finds from Eqs. (2.2) and (2.12) that for $\tau_a = 0$ and inhibitory coupling ($g < 0$) the synchronous state is stable for all $0 < \alpha < \infty$. If the discrete delay τ_a is increased from zero, then alternating bands of stability and instability are created that are periodic in τ_a with period T (see Fig. 9). This periodicity can be deduced from the following Fourier series representation of $K(\phi, T)$:

$$K(\phi,T) = \alpha^2 \frac{1-e^{-T}}{T} \sum_{m\in\mathbb{Z}} e^{2\pi i m\phi} \times \frac{e^{-2\pi i m\tau_a/T}}{[\alpha + 2\pi i m/T]^2[1 + 2\pi i m/T]}. \quad (6.4)$$

It is clear from Eq. (6.4) that changes in T due to variation of the parameter γ (characterizing the degree of depression or facilitation) will alter stability through the dependence of $\mathrm{sign}[K'(\phi,T)]$ on the dimensionless parameters αT and τ_a/T.

Elsewhere we have shown that reducing the size of the network can induce new instabilities. For example, an inhibitory network of N IF oscillators and α-function synaptic interactions can desynchronize in the strong-coupling regime, leading to oscillator death (a state in which some neurons suppress the activity of others). More precisely, there exists a critical inverse rise time $\alpha_c(N)$ such that the in-phase state is stable for arbitrary coupling g when $\alpha>\alpha_c(N)$ but becomes unstable at some critical coupling $g_c(N)$ when $\alpha<\alpha_c(N)$. Moreover, $\lim_{N\to\infty}\alpha_c(N)=0$ so that the mean-field result is recovered in the large-N limit [28].

VII. CONCLUSION

In this paper we used mean-field techniques to explore the effects of dynamic synapses on mode locking in a homogeneous IF oscillator network. A number of results were obtained.

(1) Synaptic depression increases (decreases) the collective period of oscillations of the splay state in an excitatory (inhibitory) network. The opposite holds for synaptic facilitation.

(2) In the noise-free case, depressive synapses tend to have a destabilizing effect in the sense that they reduce the parameter domain over which the splay state is stable. On the other hand, synaptic facilitation tends to have a stabilizing effect. These modifications in stability involve a static contribution arising from a rescaling of the coupling strength according to $g\to C_\infty(T)g$, which is further enhanced by dynamic contributions associated with adaptation of the synapses.

(3) Synaptic depression can enhance the stabilizing effects of noise on the splay state for sufficiently weak coupling. As in the noise-free case, this effect has both a static contribution arising from a rescaling of the coupling g and a dynamic contribution.

(4) In the large-N limit, the stability criterion for the in-phase state is $gK'(0,T)>0$, irrespective of the degree of synaptic depression or facilitation, with $K(\phi,T)$ given by Eq. (2.12). However, dynamic synapses do influence stability indirectly through changes in the collective period T.

In future work we shall investigate the more general problem of phase-locking instabilities in networks of pulse-coupled IF neurons with dynamic synapses. It has recently been shown that, in the case of activity-independent synapses and strong coupling, phase-locked states can bifurcate to states exhibiting more complex forms of behavior including oscillator death, periodic bursting, and spatially periodic activity patterns [20,21]. It will be of interest to determine how these bifurcations are modified by synaptic depression and facilitation.

ACKNOWLEDGMENTS

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APPENDIX

Let $F(t)$ be an arbitrary function of t such that $\int_{-\infty}^{\infty}F(t)dt<\infty$. Define the average $\langle\langle F\rangle\rangle_N$ according to

$$\langle\langle F\rangle\rangle_N=\frac{1}{N-1}\sum_{j=1}^{N-1}\sum_{m\in\mathbb{Z}}F([m+j/N]T). \tag{A1}$$

In terms of the Fourier transform of $F(t)$,

$$\begin{aligned}\langle\langle F\rangle\rangle_N&=\frac{1}{N-1}\sum_{j=1}^{N-1}\sum_{m\in\mathbb{Z}}\int_{-\infty}^{\infty}e^{i\omega(m+j/N)T}\tilde{F}(\omega)\frac{d\omega}{2\pi}\\&=\frac{1}{N-1}\frac{1}{T}\sum_{j=1}^{N-1}\sum_{n\in\mathbb{Z}}\tilde{F}(2\pi n/T)e^{i[2\pi nj/N]}\\&=\frac{1}{T}\left[\tilde{F}(0)-\frac{1}{N-1}\sum_{n\neq 0}\tilde{F}(2\pi n/T)\right],\end{aligned} \tag{A2}$$

where

$$\tilde{F}(\omega)=\int_{-\infty}^{\infty}e^{-i\omega t}F(t)dt. \tag{A3}$$

In the large-N limit, we obtain the result

$$\langle\langle F\rangle\rangle_\infty\equiv\lim_{N\to\infty}\langle\langle F\rangle\rangle_N=\frac{1}{T}\int_{-\infty}^{\infty}F(t)dt. \tag{A4}$$

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