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Amplitude death in nonlinear oscillators with nonlinear coupling

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Amplitude death is the cessation of oscillations that occurs in coupled nonlinear systems when fixed points are stabilized as a consequence of the interaction. We show here that this phenomenon is very general: it occurs in nonlinearly coupled systems in the absence of parameter mismatch or time delay although time-delayed interactions can enhance the effect. Application is made to synaptically coupled model neurons, nonlinearly coupled Rössler oscillators, as well as to networks of nonlinear oscillators with nonlinear coupling. By suitably designing the nonlinear coupling, arbitrary steady states can be stabilized.

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Coupled nonlinear dynamical systems have been extensively studied from both theoretical and experimental points of view in the past few decades. Natural systems are rarely isolated, and thus studies of coupled dynamical systems which arise in a variety of contexts in the physical, biological, and social sciences have broad relevance to many areas of research. Coupling gives rise to new phenomena such as synchronization, hysteresis, phase locking, phase shifting, phase-flip, or riddling [1,2].

Amplitude death (AD) is an important phenomenon that can also occur in strongly coupled nonlinear oscillators when their interaction causes a pair of fixed points to become stable and attracting [3–6]. First observed in chemical systems [3] it has become evident through a number of recent theoretical and experimental studies that this phenomenon is quite widespread. The work of Reddy *et al.* [5] showed that amplitude death could occur in identical systems if the interaction was delayed, belying earlier ideas that AD could occur only when the oscillators were mismatched [4]. Several theoretical and experimental studies of amplitude death in delay–coupled systems [6–9] have further contributed to the understanding of the effect. Very recently, a new setting for the occurrence of AD was proposed by Karnatak *et al.* [10]: when the coupling is through dissimilar or conjugate variables, oscillations can cease even when there is no time delay in the coupling and when the oscillators are identical.

Existing scenarios of amplitude death involve the stabilization of existing fixed points which are unstable in the uncoupled system. This includes the situation when the fixed points shift due to parameter mismatch. Since these are largely dependent on the nature of the uncoupled system, an important question in this context is thus one of control: is there a general strategy by which AD can be achieved?

In this Brief Report, we show that AD commonly occurs in nonlinearly coupled oscillators and can be achieved by *design* in both identical or mismatched coupled oscillators and via either instantaneous or delayed interactions. The regime of AD can be found by choosing a proper *nonlinear* coupling function, which can create new fixed points and stabilize them. This nonlinear coupling may be a given—in the sense that the systems under study are naturally coupled in this manner—or can be constrained by what is possible experimentally. Instances of both such nonlinear coupling strategies are discussed below in applications to a neuronal model with synaptic coupling, as well as to Rossler systems.

Consider a coupled system of N nonlinear oscillators. Each oscillator when isolated follows $\dot{\mathbf{X}}=\mathbf{F}(\mathbf{X})$, where $\mathbf{X}$ is an m-dimensional vector of dynamical variables and $\mathbf{F}(\mathbf{X})$ is the velocity field. The coupled system is described by the following equations:

$$\dot{\mathbf{X}}_i=\mathbf{F}_i(\mathbf{X}_i)+\frac{\epsilon}{K_i}\sum_{j=1}^{K_i}A_{ij}\mathbf{H}(\mathbf{X}_i,\mathbf{X}_j,\tau),\quad i=1,\ldots,N,\qquad(1)$$

where subscript i in $\mathbf{X}_i$ and $\mathbf{F}_i$ represents ith oscillator. Here K_i is the number of connections to the ith oscillator, namely, its degree and $1\leq K_i<N$, and ϵ is coupling strength. The connection topology is given as $A_{ij}=1$ if oscillators i and j are connected to each other and $A_{ij}=0$ otherwise. The coupling function $\mathbf{H}:R^m\rightarrow R^m$ specify the manner in which the oscillators i and j are coupled, with $\mathbf{H}(\mathbf{X}_i,\mathbf{X}_j,\tau)$ being a function of $\mathbf{X}_i(t)$ and $\mathbf{X}_j(t-\tau)$.

Our main result here is that it is possible to construct coupling functions $\mathbf{H}$ such that any desired new fixed points can be created and upon variation in coupling parameters ϵ and the time delay τ these fixed points can often be stabilized. The control and design of specific stable states can offer considerable flexibility in a number of situations of practical interest.

We first demonstrate these results in a synaptically coupled network of identical Hindmarsh-Rose (HR) neurons [11], with $\mathbf{X}_i=[x_i,y_i,z_i]^T$ and $\mathbf{H}=[h(x_i(t),x_j(t-\tau)),0,0]^T$, where h is a nonlinear function and the superscript T denotes the transpose. The equations describing the dynamics of a network of N nodes are therefore

$$\dot{x}_i=ax_i^2-x_i^3-y_i-z_i-\frac{\epsilon}{K_i}\sum_{j=1}^{K_i}A_{ij}h(x_i,x_j,\tau),$$

$$\dot{y}_i=(a+b)x_i^2-y_i,$$

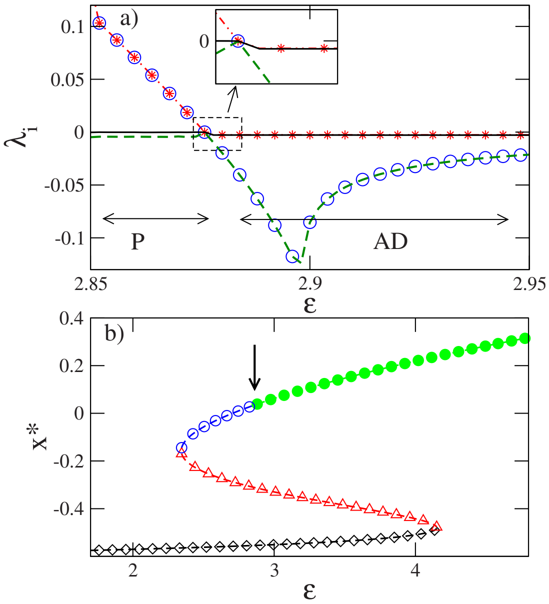

FIG. 1. (Color online) Variation in (a) the two largest Lyapunov exponents, λ_i, and (b) the fixed points, x^* of Eq. (2), as a function of coupling parameter, ϵ. In (a), the real part of the two largest eigenvalues at the lower fixed point shown in (b). The inset gives an expanded view of the curves near the bifurcation; see the text for explanations.

$$\dot{z}_i = c(dx_i + e - z_i), \tag{2}$$

where

$$h(x_i,x_j,\tau) = \frac{(x_i - V_s)}{[1 + \exp\{-\beta[x_j(t-\tau) - \Theta_s]\}]}. \tag{3}$$

At each neuron i the notation is as follows: x_i is the membrane potential and y_i and z_i are the fast and slow currents respectively. The external parameters are the synaptic coupling strength ϵ and delay τ. In our simulations [12], the reversal potential V_s is fixed at $V_s=2$ at which the synapse is excitatory, the spiking threshold is fixed at $\Theta_s=-0.25$ and the synaptic coupling function is taken to be sigmoidal, and we take $a=2.8$, $b=1.6$, $c=0.001$, $d=9$, and $e=5$ [13].

First consider $N=2$ and $\tau=0$, namely, two neurons coupled without delay. Shown in Fig. 1(a) is the variation in largest two Lyapunov exponents [solid (black) and dashed (green)] as a function of coupling strength ϵ. The first zero Lyapunov exponent becomes negative at $\epsilon_c \approx 2.83\ldots$, after which all Lyapunov exponents are negative: this corresponds to the amplitude death regime. Trajectories before and after this transition are shown in Figs. 2(a)–2(c), respectively, clearly showing that the system goes from an oscillatory regime to amplitude death.

A further calculation shows that for two identical systems, the fixed points should be symmetric, and these are (x^*,y^*,z^*), where x^* is a real root of

$$x^{*3} + bx^{*2} + dx^* + e + \epsilon h(x^*,x^*,0) = 0 \tag{4}$$

and

$$y^* = (a+b)x^{*2}, \tag{5}$$

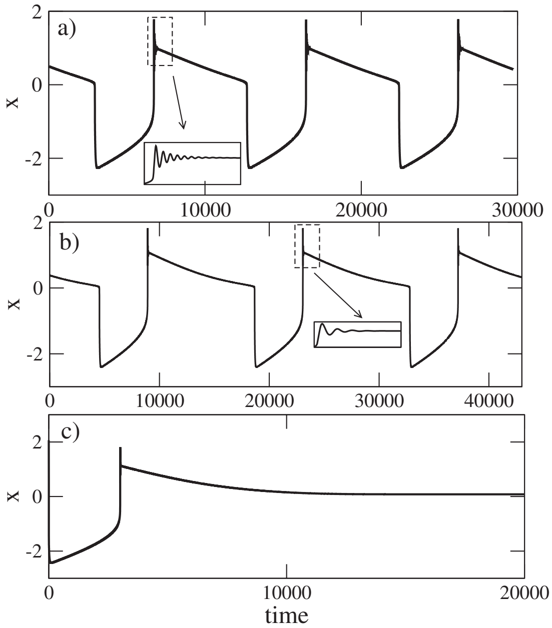

FIG. 2. Membrane potential x as a function of time for coupling strength (a) $\epsilon=2$, (b) $\epsilon=2.7$, and (c) $\epsilon=3.10$. The insets give an enlarged view of the spiking.

$$z^* = dx^* + e. \tag{6}$$

These fixed points are different from those of the uncoupled system since $h \neq 0$; their variation in ϵ is shown in Fig. 1(b). At low ϵ there is a single fixed point (open diamonds) but as ϵ is increased (around $\epsilon=2.345$) two new fixed points (shown by the open triangles and open circles) are created via a saddle-saddle bifurcation. On further increase in coupling strength (at $\epsilon=4.15$) two of these collide and a single fixed point remains.

The stability matrix of the system at these fixed points turns out to be

$$\begin{pmatrix} P(x^*) & -1 & -1 & -\epsilon R(x^*) & 0 & 0 \\ 2(a+b)x^* & -1 & -0 & 0 & 0 & 0 \\ cd & 0 & -c & 0 & 0 & \\ -\epsilon R(x^*) & 0 & 0 & P(x^*) & -1 & -1 \\ 0 & 0 & 0 & 2(a+b)x^* & -1 & 0 \\ 0 & 0 & 0 & cb & 0 & -c \end{pmatrix},$$

where

$$P(x^*) = 2ax^* - 3x^{*^2} - \epsilon Q(x^*), \tag{7}$$

$$Q(x^*) = 1/[1 + \exp\{-\beta(x^* - \Theta_s)\}], \tag{8}$$

$$R(x^*) = (x^* - V_s)\beta\frac{\exp\{-\beta(x^* - \Theta_s)\}}{[1 + \exp\{-\beta(x^* - \Theta_s)\}]^2}. \tag{9}$$

The eigenvalues [14] of the stability matrix at the fixed points can be computed, and for low values of the coupling strength when only one fixed point exists, this is unstable (open diamond) and the motion is oscillatory [Fig. 2(a)]. The fixed points created after $\epsilon=2.345$ are also unstable [open

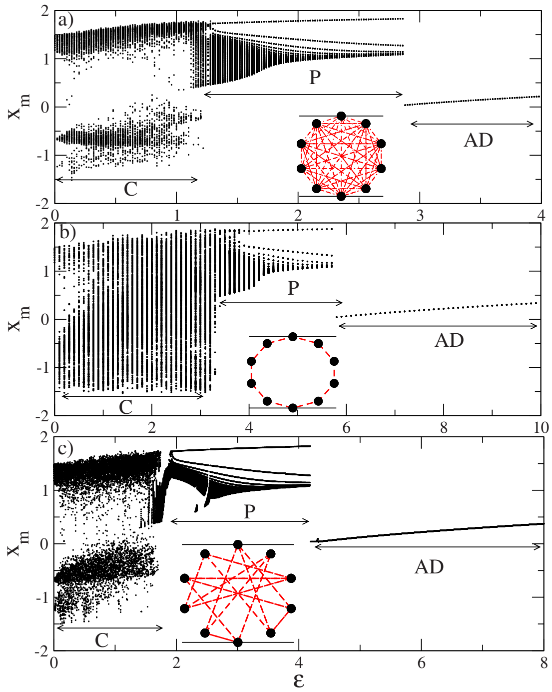

FIG. 3. (Color online) Bifurcation diagrams for the system of $N=10$ coupled oscillators with (a) global coupling, (b) nearest-neighbor coupling with periodic boundary conditions, and (c) a small-world network where each neuron is randomly connected to two others (shown in the insets). X_m corresponds to the maxima of x.

circles and triangles—see Fig. 1(b)], but as the coupling strength is further increased, around $\epsilon_c=2.83$ the real parts of all eigenvalues of one of the fixed points (upper fixed point, circles) become negative: this is the regime of amplitude death [although the other two fixed points (open diamond and triangle) remain unstable].

We plot the real part of the two largest eigenvalues corresponding to the fixed point (which gets stabilized) in Fig. 1(a) (open circles ○ and stars ⋆). Before the bifurcation both are equal and positive: the fixed point is unstable. After the bifurcation both the largest eigenvalues are distinct and negative; there is therefore a fixed point solution, namely, amplitude death. These eigenvalues coincide with the Lyapunov exponents [see Fig. 1(a)]; a typical trajectory before and after the bifurcation is shown in Figs. 2(b) and 2(c), respectively.

The nature of the dynamics prior to the saddle-saddle-type bifurcation is shown in Fig. 2(a) while Fig. 2(b) is just prior to amplitude death. The main difference is a change in the frequency of bursting which is also accompanied by a change in the spiking behavior [15] as can be seen in the inset to Fig. 2.

Networks of such oscillators coupled without time delay continue to show global amplitude death. Shown in Fig. 3 are bifurcation diagrams for networks of $N=10$ oscillators coupled in different topologies. Three cases considered here include (a) complete connection, namely, $K_i=N-1$ in Eq. (2), (b) a linear chain with periodic boundary conditions, $K_i=2$ as well as (c) random connections with $K_i=3$. The motion goes from being oscillatory to amplitude death as the

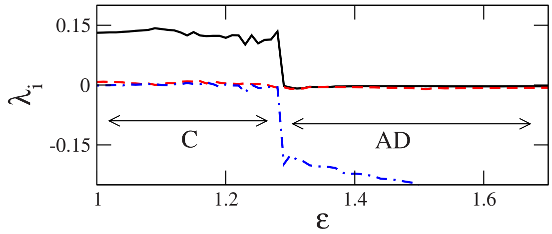

FIG. 4. (Color online) The largest three Lyapunov exponents as a function of the coupling ϵ for two HR neurons, Eq. (2) with $\tau=5$. Time-delayed interactions appear to lower the coupling threshold at which AD occurs in a given topology.

coupling strength is increased, though the threshold depends on the coupling topology. In general, though, the approach to the amplitude death regime is gradual with the range of oscillation becoming narrower with increasing coupling. The random coupling case [Fig. 3(c)] also suggests that AD can occur in the small world topology [16].

For nonzero τ the effect persists although it is more difficult to analyze since the system becomes effectively infinite dimensional. However, delay coupling is appropriate in many situations—for instance, in the study of spatially distributed biological systems—since signals are transmitted with finite velocity. As a representative case numerical results are presented for the above coupled Hindmarsh-Rose model, namely, Eq. (2) with $\tau=5$. The Lyapunov exponents as a function of the coupling strength are shown in Fig. 4 [12,17], and in comparison to the zero delay case, it can be seen that the transition to AD occurs for a lower coupling threshold. The effect is quite general and has been verified in delay-coupled Rössler oscillators as well.

It is also possible to design the coupling function in order to create new fixed points that may be required. As an example, consider *two* identical coupled Rössler systems [18] without time delay,

$$\dot{x}_i = -y_i - z_i - \epsilon h(x_i,x_j,0),$$

$$\dot{y}_i = x_i + 0.1y_i,$$

$$\dot{z}_i = 0.1 + z_i(x_i - 14), \tag{10}$$

with $h(x_i,x_j,0)$ chosen so as to vanish for specific values of x_i and x_j. For $h(x_i,x_j,0)=(x_i-\alpha)(\beta-x_j)$, with $\alpha=1$ and $\beta=2$, shown in Fig. 5 are the three largest Lyapunov exponents, with the transition to AD occurring at $\epsilon=0.096$. A

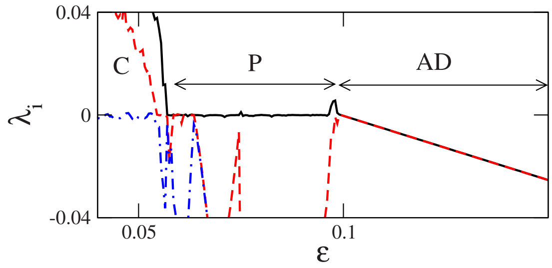

FIG. 5. (Color online) The largest three Lyapunov exponents as a function of the coupling ϵ for Rössler oscillators [Eq. (10)].

variety of different coupling forms can stabilize fixed points in this system; for instance $h(x_i,x_j,0)=(0.01x_i^2+x_i-2)(3.1-x_j-0.0001x_j^2)$ is also equally effective in achieving AD with other fixed points [15]. Since these fixed points do not exist in the uncoupled system, the nature of amplitude death in such instances differs from the case when AD is created with linear coupling [3,5,6,8].

In general, the nonlinear coupling functions, when not constrained by the natural manner in which systems interact, as in Eq. (2), can be designed in order to create new fixed points. For example, in the coupling function $\mathbf{H}(\mathbf{X}_i,\mathbf{X}_j,\tau)=(\mathbf{X}_i-\mathbf{\Delta})g(\mathbf{X}_i,\mathbf{X}_j,\tau)-\mathbf{F}_i(\mathbf{X}_i^*)$ for nonzero $g(\mathbf{X}_i,\mathbf{X}_j,\tau)$ the only fixed point solution that is possible is $\mathbf{X}_i^*=\mathbf{\Delta}$. Thus by choosing appropriate point $\mathbf{\Delta}$ one can control the amplitude death point as required (details will be presented elsewhere [15]).

In summary, we have examined the phenomenon of amplitude death in model nonlinear dynamical systems with nonlinear coupling. We have studied the effect in instantaneous as well as delay coupled systems and observe that AD is a general phenomenon that can occur in either situation. Typically, new fixed points are created and these get stabilized when parameters are varied. The same strategies are successful for networks of neuronal oscillators coupled in different topologies. We also make application to coupled Rössler oscillators wherein we design the coupling so as to achieve specific and arbitrary fixed points. Our results apply quite generally and have been verified both analytically as well as numerically for the coupled HR neuronal model. We have verified the persistence of AD in mismatched neuronal model [19] oscillators, as well as in other chaotic systems [15].

AD can be of considerable importance in controlling oscillatory dynamics, and thus the present methods are of potential utility in devising appropriate design strategies when irregular chaotic fluctuations need to be suppressed. For instance in laser modulation, different forms of coupling can be used to stabilize undesirable low-frequency chaotic fluctuations [9,20]. This transition also has a broad relevance in neuroscience applications: networks of neurons are known to have either instantaneous or time-delayed nonlinear coupling. The knowledge of how AD arises in such systems can therefore be useful in designing hybrid systems to control neuronal outputs [21].

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[1] A. Pikovsky, M. Rosenblum, and J. Kurths, *Synchronization: A Universal Concept in Nonlinear Science* (Cambridge University Press, Cambridge, 2001).
[2] K. Kaneko, *Theory and Applications of Coupled Map Lattices* (John Wiley and Sons, New York, 1993); E. Ott, *Chaos in Dynamical Systems* (Cambridge University Press, Cambridge, 1993); L. M. Pecora and T. L. Carroll, Phys. Rev. Lett. **64**, 821 (1990); G. B. Ermentrout, and N. Koppel, SIAM J. Appl. Math. **50**, 125 (1990); A. Prasad *et al.*, Pramana, J. Phys. **64**, 513 (2005).
[3] K. Bar-Eli, Physica (Amsterdam) **14D**, 242 (1985).
[4] R. E. Mirollo and S. H. Strogatz, J. Stat. Phys. **60**, 245 (1990); G. B. Ermentrout, Physica (Amsterdam) **41D**, 219 (1990); D. G. Aronson *et al.*, *ibid.* **41D**, 403 (1990).
[5] D. V. Ramana Reddy, A. Sen, and G. L. Johnston, Phys. Rev. Lett. **80**, 5109 (1998).
[6] F. M. Atay, Phys. Rev. Lett. **91**, 094101 (2003); R. Dodla, A. Sen, and G. L. Johnston, Phys. Rev. E **69**, 056217 (2004); D. V. Ramana Reddy, A. Sen, and G. L. Johnston, Phys. Rev. Lett. **85**, 3381 (2000); R. Herrero, M. Figueras, J. Rius, F. Pi, and G. Orriols, *ibid.* **84**, 5312 (2000); J.-W. Ryu *et al.*, Phys. Rev. E **70**, 036220 (2004); J. Zhou and Z. Liu, *ibid.* **77**, 056213 (2008); S. H. Strogatz, Nature (London) **394**, 316 (1998).
[7] Fixed points having odd number of real positive eigenvalues cannot be stabilized in time-delay systems; see K. Konishi, Phys. Lett. A **341**, 401 (2005).
[8] A. Prasad, Phys. Rev. E **72**, 056204 (2005); A. Prasad, J. Kurths, S. K. Dana, and R. Ramaswamy, *ibid.* **74**, 035204(R) (2006).
[9] P. Kumar, A. Prasad and R. Ghosh, J. Phys. B **41**, 135402 (2008); **42**, 145401 (2009).
[10] R. Karnatak, R. Ramaswamy, and A. Prasad, Phys. Rev. E **76**, 035201(R) (2007); Chaos **19**, 033143 (2009).
[11] J. L. Hindmarsh and R. M. Rose, Proc. R. Soc. London, Ser. B **221**, 87 (1984).
[12] The flows [Eqs. (2) and (10)] are integrated using Runge-Kutta fourth order scheme with integration step $\Delta t=0.005$ for $\tau=0$ and with $\Delta t=\tau/N$ where $N=300$ is fixed for delay coupling.
[13] I. Belykh, E. de Lange, and M. Hasler, Phys. Rev. Lett. **94**, 188101 (2005).
[14] This matrix turns out to be ill-conditioned. Therefore we compute the eigenvalues numerically using balancing, Hessenberg reduction, followed by QR algorithm techniques. Details can be found in W. H. Press *et al.*, *Numerical Recipes* (Cambridge University Press, Cambridge, 2007).
[15] A. Prasad *et al.* (unpublished).
[16] Z. Hou and H. Xin, Phys. Rev. E **68**, 055103(R) (2003).
[17] J. D. Farmer, Physica (Amsterdam) **4D**, 366 (1982).
[18] O. Rössler, Phys. Lett. **57A**, 397 (1979).
[19] P. Channell *et al.*, Phys. Rev. Lett. **98**, 134101 (2007).
[20] A. Prasad *et al.*, Phys. Lett. **314A**, 44 (2003).
[21] M. Sorensen *et al.*, J. Neurosci. **24**, 5427 (2004).