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Controlling synchrony in oscillatory networks with a separate stimulation-registration setup

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Abstract – We present a demand-controlled method for desynchronization of globally coupled oscillatory networks utilizing a configuration with an observed and stimulated subsystem. The stimulated subsystem is subjected to a proportional-integro-differential (PID) feedback derived from the mean field of the observed subsystem. Our method enables to restore desynchronized states in both subsystems in a robust way. We develop an analytical theory for the Kuramoto model and analytically derive a threshold of the stimulation parameters for the desynchronization transition in ensembles of phase and van der Pol oscillators. We also numerically demonstrate the efficacy of the approach for ensembles of globally coupled Landau-Stuart and relaxation van der Pol oscillators. Our approach is particularly important for applications to physical and biological systems which do not allow for a simultaneous registration and stimulation of the whole network, as in the case of electrical brain stimulation.

Introduction. – Synchronization is an important phenomenon in physical, chemical, and biological systems [1–5]. In the brain, communication within and between neuronal populations is mediated by synchronization processes [6]. Conversely, extremely strong synchronization may severely impair brain function. For instance, Parkinsonian tremor is caused by the synchronization of oscillatory neurons located in the thalamus and basal ganglia [7], which under healthy conditions fire in an uncorrelated manner [8].

Electrical deep brain stimulation (DBS) is the standard therapy for medically refractory movements disorders, *e.g.*, Parkinson's disease and essential tremor [9,10]. In DBS, electrical high-frequency (>100 Hz) stimulation is permanently delivered via depth electrodes located in affected target areas. DBS has been developed empirically and appears to strongly manipulate the neuronal firing, *e.g.*, by blocking neuronal action [10]. DBS may cause side effects, and its therapeutic effect may be limited or may decrease over time [11]. Hence, there is a significant clinical need for less invasive stimulation techniques, which restore desynchronized —*i.e.*, normal [8]— dynamics in networks of oscillatory neurons [12].

Accordingly, stimulation techniques have been developed which effectively desynchronize oscillatory networks by utilizing phase resetting principles [12–14] or delayed feedback stimulation [15–20]. For applications to biological systems it is essential that desynchronization is robust against variations of system parameters. Significant time variations of the neurons' frequency are, *e.g.*, observed in DBS target areas [21]. Multisite coordinated reset stimulation [14], linear multisite delayed feedback [16,17], and nonlinear delayed feedback [18,19] effectively desynchronize and are robust against parameter variations. While the coordinated reset requires repetitive stimulus administration, the feedback techniques work at minimal stimulation intensities.

However, concerning the applicability of feedback methods, there is still a fundamental problem. Desynchronization can effectively be achieved, provided that (nearly) the whole network can be registered and stimulated, as shown previously [15,16,18,22]. But in certain experimental systems it is impossible to register and stimulate the whole network at the same time. In DBS, *e.g.*, the stimulation current exceeds the measured neuronal currents by several orders of magnitude, so that measurements are corrupted by strong artifacts [23].

To overcome this issue, we split the whole population of coupled oscillators into two separate subpopulations, one being exclusively measured and the other being exclusively stimulated (see also refs. [17,19], where, in contrast to the present study, a drive-response coupling scheme of populations has been considered). This leads to a considerably more difficult control problem as considered formerly. The feedback control algorithms discussed so far in the literature [15,16,18] fail if they are applied to oscillatory networks with a separate stimulation-registration setup. In contrast, we here use the proportional-integro-differential (PID) feedback control technique, which is widely accepted in the classical control theory, but has not yet been applied to such a desynchronization issue. By using the mean-field signal of the measured subpopulation as PID feedback and delivering it to the stimulated subpopulation, we achieve a robust desynchronization of the whole network, at minimal stimulation intensity. Oscillatory neuronal activity is characteristic for Parkinson's disease [7,8]. To capture this fundamental dynamical feature, we here model a periodically active neuron in a normal-form type approach by means of a Landau-Stuart or a phase oscillator and, hence, a neuronal population by a network of N globally coupled limit-cycle and phase oscillators [1,24–26]. We demonstrate the efficacy of our approach for ensembles of globally coupled Landau-Stuart and (as an extension) relaxation van der Pol oscillators and derive a theory for the Kuramoto model of phase oscillators. We analytically classify regions in the parameter space, where the stimulation results in a robust desynchronization of both subpopulations.

PD control of globally coupled Landau-Stuart oscillators. – Consider an ensemble of N globally coupled and stimulated Landau-Stuart oscillators, representing a normal form of a supercritical Andronov-Hopf bifurcation

$$\begin{aligned}\dot{z}_j &= (\mathrm{i}\omega_j + 1 - |z_j|^2)z_j + KZ,\ \ j = 1,2,\dots,N_1,\\ \dot{z}_j &= (\mathrm{i}\omega_j + 1 - |z_j|^2)z_j + KZ + S(t),\ \ j = N_1+1,\dots,N.\end{aligned} \tag{1}$$

The individual oscillators $z_j = x_j + \mathrm{i}y_j$, $j = 1,\dots,N$ without coupling ($K = 0$) and without stimulation ($S(t) = 0$) perform a uniform rotation around the origin with the natural frequencies ω_j and amplitudes 1. The oscillators are globally coupled via the mean field $Z = N^{-1}\sum_{k=1}^{N} z_k$ with the coupling strength K. The whole population is split into two sub-populations of N_1 and $N_2 = N - N_1$ units. The oscillators $j = 1,\dots,N_1$ of the first subsystem are not exposed to the control perturbation and serve as an observable subsystem. The oscillators $j = N_1+1,\dots,N$ of the second subsystem are stimulated with a proportional-differential (PD) feedback signal

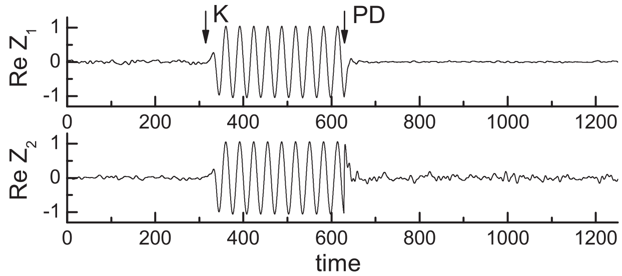

Fig. 1: Mean fields of the observed ($\mathrm{Re}\,Z_1$) and stimulated ($\mathrm{Re}\,Z_2$) subsystem of the ensemble of oscillators (1) for $N_1 = N_2 = 500$, $K = 0.5$, $P = 2$, and $D = 4$. The frequencies $\{\omega_j\}$ are Gaussian distributed with deviation $\sigma = 0.2$ around the mean frequency $\Omega = 0.2$. The arrows indicate the onset of coupling (K) and stimulation (PD).

$$S(t) = PZ_1(t) + D\dot{Z}_1(t), \tag{2}$$

where we suppose that an observable signal is the mean field of the first subsystem $Z_1 = N_1^{-1}\sum_{k=1}^{N_1} z_k$. The parameters P and D define the strength of the proportional and differential feedback, respectively. Note, as will be shown below, if the coupling in the ensemble is rather weak, the desynchronization can be achieved by applying the proportional feedback only. In contrast, in the case of strong coupling the stimulation signal must also contain the differential feedback for robust desynchronization.

Numerical results presented in fig. 1 show the dynamics of the mean fields Z_1 and $Z_2 = N_2^{-1}\sum_{k=N_1+1}^{N} z_k$ of the separated subsystems of ensemble (1) in three successive stages: i) without coupling and control ($K = P = D = 0$), ii) with coupling but without control ($K \neq 0,\ P = D = 0$), and iii) in the presence of both coupling and control ($K \neq 0,\ P \neq 0,\ D \neq 0$). In the first stage small fluctuations of the mean fields are related to the finite-size effect. Switching on the coupling leads to a spontaneous synchronization characterized by a large amplitude of the mean field. Then, after additionally switching on the PD control, the oscillators desynchronize, and the mean fields of both subsystems become again small, as they were without coupling. As soon as the desired desynchronized state is achieved, the observable signal Z_1 vanishes and thus the stimulation signal $S(t)$ vanishes as well, which demonstrates the noninvasive character of the suggested control technique. Another important issue of the control is that the stimulation does not destroy the natural oscillatory activity of the individual elements of the ensemble. In the stimulation regime, the current mean frequencies $\overline{\omega}_j$ of the individual oscillators differ from the natural frequencies ω_j by a value of the order 10^{-3}. Thus the stimulation practically restores the frequencies of the oscillators to their natural ones.

Analytical treatment for the Kuramoto model. – To explore the main properties of the PD control algorithm, we investigate the phase dynamics of the ensemble (1). Substituting in eq. (1) $z_j = \rho_j e^{i\theta_j}$ and neglecting the dynamics of the amplitudes ρ_j, one obtains the following equation for the phases θ_j:

$$\dot{\theta}_j = \omega_j + \frac{K}{N}\sum_{k=1}^{N}\sin(\theta_k - \theta_j) - H(j - N_1)F_j, \tag{3a}$$

$$F_j = \frac{P}{N_1}\sum_{k=1}^{N_1}\sin(\theta_k - \theta_j) + \frac{D}{N_1}\sum_{k=1}^{N_1}\dot{\theta}_k\cos(\theta_k - \theta_j). \tag{3b}$$

Here F_j is the corresponding phase representation of the above stimulation signal S and $H(\cdot)$ is the Heaviside function defined as $H(k) = 0$ if $k \leqslant 0$ and $H(k) = 1$ if $k > 0$.

Without stimulation ($F_j = 0$), eq. (3a) transforms to the classical Kuramoto model [1]. We assume that the frequencies ω_j are randomly chosen from a symmetric probability density $g(\omega)$, $g(\Omega + \omega) = g(\Omega - \omega)$, where Ω is the mean frequency. Then the critical coupling of spontaneous synchronization is given by [1,2]

$$K_0 = 2/\pi g(\Omega). \tag{4}$$

For $K < K_0$, the system relaxes to an incoherent state, where all oscillators are not synchronized, but for $K > K_0$, mutual synchronization occurs in a group of oscillators.

Our aim is to define the synchronization threshold in the presence of the PD control signal F_j. We characterize the synchronization by the complex order parameters

$$r_1 e^{i\psi_1} = \frac{1}{N_1}\sum_{k=1}^{N_1} e^{i\theta_k}, \quad r_2 e^{i\psi_2} = \frac{1}{N_2}\sum_{k=N_1+1}^{N} e^{i\theta_k}, \tag{5}$$

where $r_{1,2}$ measure the phase coherence of the population in each subsystem. These parameters vary in the interval $[0,1]$ such that small values of $r_{1,2}$ indicate the incoherent state while values close to 1 represent the strong mutual synchronization in each subsystem. The synchronization in the whole system can be characterized by the total order parameter $re^{i\psi} = N^{-1}\sum_{k=1}^{N} e^{i\theta_k}$. To solve the problem analytically we write the system (3) in the infinite-N limit. For convenience, we choose a coordinate system rotating with the mean frequency Ω. Then the density $g(\omega)$ has zero mean, and eq. (3b) transforms to

$$F_j = \frac{P}{N_1}\sum_{k=1}^{N_1}\sin(\theta_k - \theta_j) + \frac{D}{N_1}\sum_{k=1}^{N_1}(\dot{\theta}_k + \Omega)\cos(\theta_k - \theta_j). \tag{6}$$

We consider each oscillator as a particle moving around a cycle. For each frequency ω, let $\rho^{\omega}_{1,2}(\theta, t)$ denote the density of oscillators at angle θ at time t in the subsystem marked by the subscript, and let $v^{\omega}_{1,2}(\theta, t)$ denote the local phase velocity of an oscillator also considered at angle θ and at time t in the corresponding subsystem (see ref. [27] and below). Then the densities $\rho^{\omega}_{1,2}$ satisfy the continuity equations

$$\frac{\partial}{\partial t}\rho^{\omega}_{1,2}(\theta, t) = -\frac{\partial}{\partial \theta}\left[\rho^{\omega}_{1,2}(\theta, t) v^{\omega}_{1,2}(\theta, t)\right], \tag{7}$$

which express conservation of oscillators of frequency ω in each subsystem. The velocities $v^{\omega}_{1,2}(\theta, t)$ read

$$\begin{aligned} v^{\omega}_1(\theta, t) &= \omega + K\int_0^{2\pi}\int_{-\infty}^{\infty}\sin(\theta' - \theta)[n_1\rho^{\omega'}_1(\theta', t) \\ &\quad + n_2\rho^{\omega'}_2(\theta', t)]g(\omega')\,d\omega' d\theta', \\ v^{\omega}_2(\theta, t) &= v^{\omega}_1(\theta, t) - \int_0^{2\pi}\int_{-\infty}^{\infty}\rho^{\omega'}_1(\theta', t)[P\sin(\theta' - \theta) \\ &\quad + D(v^{\omega'}_1(\theta', t) + \Omega)\cos(\theta' - \theta))]g(\omega')\,d\omega' d\theta', \end{aligned}$$

where $n_{1,2} = N_{1,2}/N$ denotes the relative number of oscillators in the first and second subsystems such that $n_1 + n_2 = 1$. Here in the continuum limit $N \to \infty$ we have replaced the sums in eqs. (3) by integrals. Similarly, in the limit $N \to \infty$ the above order parameters (5) become

$$r_{1,2}e^{i\psi_{1,2}} = \int_0^{2\pi}\int_{-\infty}^{\infty} e^{i\theta}\rho^{\omega}_{1,2}(\theta, t)g(\omega)\,d\omega d\theta. \tag{8}$$

This yields a system of nonlinear integro-partial-differential equations for $\rho^{\omega}_{1,2}$. We also require $\rho^{\omega}_{1,2}$ to be positive, 2π periodic in θ with $\int_0^{2\pi}\rho^{\omega}_{1,2}(\theta, t)d\theta = 1$.

The incoherent state with the vanishing order parameters $r_{1,2} = 0$ is characterized by uniform distributions $\rho^{\omega}_{1,2}(\theta) \equiv 1/2\pi$. We use the substitution

$$\rho^{\omega}_{1,2}(\theta, t) = 1/2\pi + c_{1,2}(\omega, t)e^{i\theta} + \text{c.c.} + \text{h.h.}, \tag{9}$$

to analyze the linear stability of this fixed point. Here $c_{1,2}$ are coefficients of the Fourier expansion of the densities $\rho_{1,2}$, and the abbreviations "c.c." and "h.h." stand for "complex conjugate" and "higher harmonics," respectively. The perturbation is written in this way because the order parameters $r_{1,2}$ depend only on $c_{1,2}$, but not on higher harmonics, and the linearized amplitude equations for $c_{1,2}$ decouple from the other harmonics (cf. ref. [2]):

$$\partial c_1/\partial t = -i\omega c_1 + K[n_1R_1(t) + n_2R_2(t)]/2, \tag{10}$$

$$\begin{aligned} \partial c_2/\partial t = -i\omega c_2 + K[(n_1 - D/2 + iD\Omega)R_1(t) \\ + (n_2 - D/2)R_2(t)]/2 + iDR_3(t)/2. \end{aligned} \tag{11}$$

Here we have introduced the functions

$$R_k(t) = \int_{-\infty}^{\infty} c_k(\omega, t)g(\omega)d\omega, \qquad k = 1, 2, 3, \tag{12}$$

with the auxiliary coefficient $c_3(\omega, t) \equiv \omega c_1(\omega, t)$. The dynamics of the order parameters are given by

$$r_{1,2}(t) = 2\pi|R_{1,2}(t)|. \tag{13}$$

For a slim notation we use the vectors: $\boldsymbol{c} \equiv (c_1\, c_2\, c_3)^T$, $\boldsymbol{R} \equiv (R_1\, R_2\, R_3)^T$. For any initial conditions $\boldsymbol{c}(\omega, 0) \equiv \boldsymbol{c}_0(\omega)$, via the Laplace transform we obtain

$$\boldsymbol{R}(t) = \frac{1}{2\pi i}\int_{\Gamma}[I_3 - A(s)g^*(s)]^{-1}(\boldsymbol{c}_0 g)^*(s)e^{st}ds \tag{14}$$

as solution of eqs. (10)–(12). Here the asterisk is related to the Hilbert transform operation, $f^*(s) \equiv \int_{-\infty}^{\infty} d\omega f(\omega)/(s+i\omega)$, I_3 is the 3×3 identity matrix, and

$$A(s) = \frac{1}{2}\begin{pmatrix} Kn_1 & Kn_2 & 0 \\ Kn_1(1-\frac{D}{2}) - P + iD\Omega & Kn_2(1-\frac{D}{2}) & iD \\ \alpha(s)Kn_1 & \alpha(s)Kn_2 & 0 \end{pmatrix},$$

where $\alpha(s) = (\omega g)^*(s)/g^*(s)$. The contour Γ is the vertical line in the region $\mathrm{Re}(s) > 0$ to the right of any singularities of the integrand. The singularities may appear as zeros of the determinant when calculating the inverse matrix in eq. (14):

$$\det[I_3 - A(s)g^*(s)] = 0, \quad \mathrm{Re}(s) > 0. \tag{15}$$

This characteristic equation can be solved analytically if $g(\omega)$ is a sufficiently simple function. In the following we consider the Lorentzian density

$$g(\omega) = \gamma/\pi(\gamma^2 + \omega^2). \tag{16}$$

We get $g^*(s) = 1/(s+\gamma)$, $(\omega g)^*(s) = -i\gamma/(s+\gamma)$ for $\mathrm{Re}(s) > 0$, and thus $\alpha = -i\gamma$. It follows that the matrix A is independent of s and the characteristic equation transforms to the simple quadratic equation

$$s^2 - \sigma s + \Delta + i\delta = 0 \tag{17}$$

with $\sigma = (1 - n_2 D/2)K/2 - 2\gamma$, $\Delta = \gamma^2 - \gamma K/2 + n_2 PK/4$, and $\delta = -n_2 K D\Omega/4$. Although this equation is derived for $\mathrm{Re}(s) > 0$ its solutions correctly define the characteristic values of the exponential decay of $\boldsymbol{R}(t)$ for $\mathrm{Re}(s) < 0$ as well. This is because an estimation of the integral in eq. (14) for $t > 0$ requires the shift of the contour Γ to the left and thus an analytical continuation of the integrand to the region $\mathrm{Re}(s) < 0$. Therefore, the incoherent state is stable if both solutions of the quadratic characteristic equation are in the left half plane, *i.e.*, when the following conditions are met $\sigma < 0$, $\Delta > \delta^2/\sigma^2$ or, equivalently,

$$D > \tilde{D} \equiv \frac{2}{n_2}\left(1 - \frac{2K_0}{K}\right), \tag{18a}$$

$$P > \tilde{P} \equiv \frac{K_0}{n_2}\left(1 - \frac{K_0}{K}\right) + \frac{4\Omega^2 D^2}{n_2 K (D - \tilde{D})^2}. \tag{18b}$$

Here $K_0 = 2\gamma$ is the critical coupling of the stimulation-free ($P = D = 0$) system (3) with the Lorentzian distributed frequencies ω_j. Note, these conditions can be satisfied by utilizing only the proportional feedback control, *i.e.*, for $D = 0$. In this case the conditions (18) can be simplified to

$$K < \tilde{K} \equiv K_0/(1 - n_2 P/K_0), \quad \text{when } P < K_0/2n_2,$$
$$2K_0, \quad \text{when } P \geqslant K_0/2n_2. \tag{19}$$

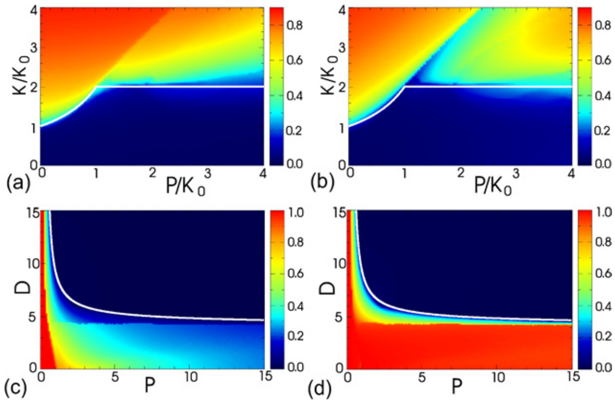

Fig. 2: The values of the time-averaged order parameters $\langle r_{1,2}(t)\rangle$ of (a), (c) observed and (b), (d) stimulated subsystems of the Kuramoto model (3) are encoded in color ranging from blue (incoherent state) to red (coherent state) for (a), (b) the proportional feedback control ($D = 0$) and (c), (d) proportional-differential feedback control. The number of oscillators $N_1 = N_2 = 1000$ and the other parameters are (a), (b) $\gamma = 0.2$ and $D = 0$ and (c), (d) $\gamma = 0.005$, $\Omega = 0.2$, and $K = 100K_0 = 1$. The analytically predicted critical coupling $\tilde{K} = \tilde{K}(P)$ (eq. (19)) in (a), (b) and threshold $\tilde{P} = \tilde{P}(D)$ (eq. (18b)) in (c), (d) are shown by white curves.

Here $\tilde{K}$ denotes the critical coupling under proportional ($D = 0$) feedback control with the strength P.

Figure 2(a), (b) shows that the analytical prediction (19) of the critical coupling $\tilde{K}$ depicted by white curves is in a good agreement with numerical simulations of eq. (3) for a finite number of oscillators. The incoherent state corresponds to the dark blue regions in fig. 2(a), (b). We see that the proportional feedback control is not very effective in the separate stimulation-registration control setup. The proportional feedback control can be successful only for small values of the coupling strength, which do not exceed the double critical coupling of the control-free system, $K < 2K_0$.

The PD control algorithm involving both the proportional and differential feedback components is more efficient. According to eq. (18) the incoherent state can be stabilized by the PD control for arbitrarily large values of the coupling strength K. Figure 2(c), (d) shows the stability region of the incoherent state in the plane of the parameters (P, D) (dark blue regions) for $K = 100K_0$. Again the numerical simulations of eq. (3) for a finite number of oscillators agree well with the analytical threshold values $\tilde{P} = \tilde{P}(D)$ defined by eq. (18) (white curves in fig. 2(c), (d)).

According to eq. (18), the desynchronization threshold values $\tilde{P}$ and $\tilde{D}$ of the parameters P and D increase if the relative number of oscillators n_2 in the stimulated subsystem decreases or if the mean frequency Ω increases. Numerical simulations of eq. (3) with different values of the parameters N_1, N_2, and Ω shown in fig. 3 conform this theoretical prediction as well.

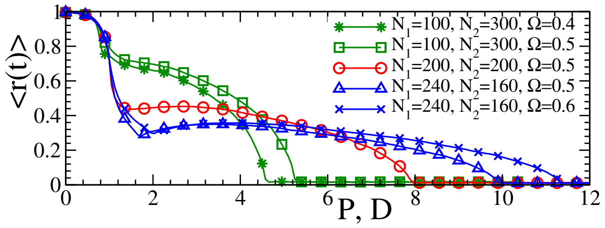

Fig. 3: The time-averaged total order parameter $\langle r(t)\rangle$ of the Kuramoto model (3) is depicted *vs.* parameters $P=D$ for different number of oscillators N_1 and N_2 and mean frequencies Ω, as indicated in the legend. The total number of oscillators is $N=N_1+N_2=400$ and $K=1$. The natural frequencies $\{\omega_j\}$ are Gaussian distributed with deviation $\sigma=0.1$.

PID control of globally coupled van der Pol oscillators. – As a last example we consider an ensemble of globally coupled van der Pol oscillators:

$$\begin{aligned}\dot{x}_j &= y_j,\\ \dot{y}_j &= -\omega_j^2 x_j - \varepsilon(x_j^2-1)y_j + KY\\ &\quad -H(j-N_1)(PY_1+IX_1+D\dot{Y}_1),\end{aligned}\tag{20}$$

where the parameter ε defines the strength of nonlinearity of each oscillator, $Y_1=N_1^{-1}\sum_{k=1}^{N_1} y_k$, $X_1=N_1^{-1}\sum_{k=1}^{N_1} x_k$ are the mean fields of the observed subsystem, and $Y=N^{-1}\sum_{k=1}^{N} y_k$ is the total mean field. In contrast to the previous examples, we here use a more general (and even more efficient) stimulation protocol, the proportional-integro-differential (PID) control algorithm. We suppose that the observable is the mean field Y_1. The variable X_1 is the integral of Y_1, the constant I denotes the strength of integral feedback component, and the other parameters are as above. For small parameters ε, K, P, I, and D:

$$\varepsilon \ll \Omega, \quad K \ll \Omega, \quad P \ll \Omega, \quad I \ll \Omega^2, \quad D \ll 1, \tag{21}$$

eqs. (20) can be transformed to the system of the PD controlled Landau-Stuart oscillators. Indeed, substituting $x_j=z_j\mathrm{e}^{\mathrm{i}\Omega t}+\text{c.c.}$, $y_j=\mathrm{i}\Omega z_j\mathrm{e}^{\mathrm{i}\Omega t}+\text{c.c.}$, for slowly varying amplitudes z_j, we obtain

$$\begin{aligned}\dot{z}_j &= \frac{\varepsilon}{2}\left(\mathrm{i}\frac{\omega_j^2-\Omega^2}{\varepsilon\Omega}+1-|z_j|^2\right)z_j+\frac{K}{2}Z\\ &\quad -H(j-N_1)\left[\frac{1}{2}\left(P+\frac{I}{\mathrm{i}\Omega}+\mathrm{i}\Omega D\right)Z_1+D\dot{Z}_1\right].\end{aligned}$$

The main difference of this equation from eqs. (1), (2) is that here the constant $(P+I/\mathrm{i}\Omega+\mathrm{i}\Omega D)/2$ responsible for the proportional feedback strength (in the representation of Landau-Stuart oscillators) is complex valued. Similarly as above this system can be further reduced to the PD controlled Kuramoto model. Substituting $z_j=\rho_j\mathrm{e}^{\mathrm{i}\theta_j}$ and neglecting the dynamics of amplitudes ρ_j one obtains for the phases θ_j a system

$$\dot{\theta}_j = \frac{\omega_j^2-\Omega^2}{2\Omega}+\frac{K}{2N}\sum_{k=1}^{N}\sin(\theta_k-\theta_j)-H(j-N_1)F_j,$$

$$F_j=\frac{P}{2N_1}\sum_{k=1}^{N_1}\sin(\theta_k-\theta_j)+\frac{D}{N_1}\sum_{k=1}^{N_1}\left(\dot{\theta}_k+\frac{\beta}{2}\right)\cos(\theta_k-\theta_j),$$

which is equivalent to eqs. (3a), (6); only the coefficients are different. Here instead of Ω in eq. (6) we have the coefficient $\beta/2$, where $\beta=\Omega-I/D\Omega$. Thus we can use the previous results obtained for the Kuramoto model in order to derive the desynchronization thresholds of the PID controlled van der Pol oscillators. After a simple redefinition of the parameters it appears that the inequalities (18) defining the stability of the incoherent state remain valid for the PID controlled van der Pol oscillators (20) with the only difference that the parameter Ω in eq. (18b) is replaced by the parameter β:

$$D>\tilde{D}\equiv\frac{2}{n_2}\left(1-\frac{2K_0}{K}\right), \tag{22a}$$

$$P>\tilde{P}\equiv\frac{K_0}{n_2}\left(1-\frac{K_0}{K}\right)+\frac{4\beta^2D^2}{n_2K(D-\tilde{D})^2}. \tag{22b}$$

The parameter K_0 now denotes the critical coupling of the stimulation-free van der Pol oscillators (20) with the Lorentzian distributed frequencies ω_j, which is $K_0=4\gamma$.

We now recall that the desynchronization threshold $\tilde{P}$ of the previously considered PD controlled Kuramoto model (3) increases rapidly with the increase of the mean frequency Ω. This is related to the last term in eq. (18b), which is proportional to Ω^2. For the PID controlled van der Pol oscillators, the parameter Ω is replaced by β and the additionally involved integral feedback allows us to manipulate with this term. By setting the following relationship between the integral and differential feedback strength components

$$I=\Omega^2 D, \tag{23}$$

we can set the parameter β to zero. As a result we can remove the undesirable last term in eq. (22b). The relationship (23) is optimal in the sense that it makes the conditions (22) defining the stability of the incoherent state independent of the mean frequency Ω.

Although the conditions (22) are derived for small values of the parameters ε, K, P, I, and D, the numerical analysis of eqs. (20) shows that the PID control algorithm works well even when the inequalities (21) are not fulfilled, provided the relationship (23) is satisfied. In fig. 4 we demonstrate the successful suppression of synchrony in the ensemble of globally coupled van der Pol oscillators (20) via the PID algorithm when the uncontrolled coupled oscillators exhibit relaxation oscillations.

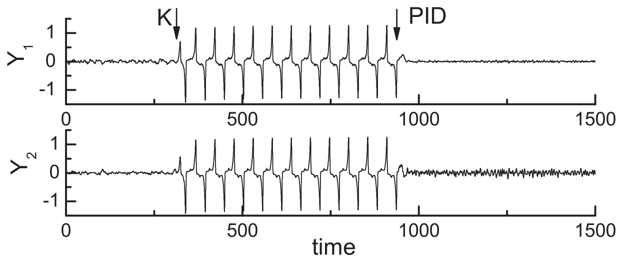

Fig. 4: Desynchronization of the relaxation van der Pol oscillators (20) by PID feedback. Mean fields of the observed (Y_1) and stimulated (Y_2) subsystem of ensemble (20) are shown for $N_1 = N_2 = 100$, $\varepsilon = 0.5$, and $K = 1$. In eq. (20) $\{\omega_j\}$ are Gaussian distributed with mean $\Omega = 0.2$ and deviation $\sigma = 0.1$. The parameters of the PID controller are $P = 2$, $D = 5$, and $I = \Omega^2 D = 0.2$. Arrows indicate when coupling (K) and control (PID) are switched on, respectively.

Conclusion. – We have proposed an efficient control algorithm to suppress the synchrony in ensembles of globally coupled oscillators for a difficult control situation, when the simultaneous registration and stimulation of the whole network is not possible. In this situation previously designed control techniques [15,16,18,22] fail. In this paper we have used a linear combination of the proportional, differential, and integral feedback. A more general approach can be based on the design of a suitable adaptive filter [28,29].

Our control technique utilizing the separate stimulation-registration setup is noninvasive and robust. It may contribute to a novel effective electrical stimulation therapy for brain diseases characterized by abnormal synchrony. In Parkinsonian and essential tremor separate stimulation and recording sites might be implanted in one (*e.g.*, thalamic) DBS target population. Alternatively, for PID control of both depth and cortical activity only the stimulation site might be implanted in a target population in the depth, whereas an epicortical electrode might register cortical activity of a network coupled to the pacemaker network in the depth. Analogously, our approach might be applied to an epileptic focus or to two interacting foci.

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