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A proportional mean-field feedback for the desynchronization and inhibition of Kuramoto oscillators

A. Franci, A. Chaillet, W. Pasillas-Lépine, E. Panteley, F. Lamnabhi-Lagarigue

Abstract—In this paper we summarize recent advances in the controlled alteration of synchronization in networks of interconnected Kuramoto oscillators. Motivated by neuroscience applications, and in particular by the deep brain stimulation treatment for Parkinson’s disease, we show that a scalar signal, proportional to the mean behaviour of the oscillators population, may either desynchronize or inhibit the resulting oscillations.

I. INTRODUCTION

Neuronal oscillations play a central role in brain functioning. On the one hand, neuronal synchronous oscillations are at the basis of fundamental functions like memory, cognition and movement path generation. On the other hand, a too strong neuronal synchronization can lead to pathological states such as Parkinson’s disease (PD).

Parkinsonian patients exhibit an intense oscillatory synchronous activity in some deep brain areas. This synchrony is tightly correlated to PD physical symptoms [1]. A successful treatment of PD symptoms is called deep brain stimulation (DBS), which consists in a permanent electrical stimulation of deep brain areas through implanted electrodes [2].

Despite its impressive therapeutic results, DBS still suffers from considerable limitations. DBS relies, in its present form, on no cerebral measurement but rather permanently injects a square signal in deep brain structures, regardless of the neurological activity of the patient (open-loop stimulation). This has negative consequences in terms of side effects and energy consumption.

The attempts to develop a closed-loop DBS face several inherent obstacles. First, due to intrinsic heterogeneities, uncertainties, dimensions, and non-linearities, the dynamics ruling the neuronal population’s behaviour is complex. In addition, due to the size of the electrode with respect to the neuronal scale, the only measurement one may rely on is the mean electrical activity of the neuronal population around the electrode head (local field potential). In the following we refer to this scalar measurement as mean-field. Thus, for the same reason, only one input signal is available for stimulation. From a control perspective, closed-loop DBS

consists in altering synchronization of a complex large-scale scale based on single measurement and a single control input.

Another important limitation in the present DBS stands in the little understanding of its underlying mechanisms: the debate is still open in the medical community about the exact functioning of DBS and its relationships with neuronal synchronization [3]. More precisely, it is not yet clear whether DBS acts by desynchronizing neuronal cells, inhibiting their behaviour, or modify the frequency of the resulting oscillations.

In this paper, we develop a simple scalar control law and test the two former hypotheses. We show, based on a simplified model of phase oscillators, that a stimulation signal taken proportional to the measured mean-field may induce either desynchronization or oscillation inhibition in the stimulated neuronal population.

Apart from its simple nature, which ensures mathematical treatability and easy practical implementation, the proportional feedback approach is particularly tempting for DBS for energetic concerns. Energy efficiency is a crucial issue in DBS for side effects reduction and diminution of surgical operations for battery replacement. Since the population’s mean electrical activity is small in both the desynchronized and inhibited states, a proportional feedback approach ensures a DBS signal of small amplitude.

The model we rely on is derived from the complex Landau-Stuart oscillator. After classical simplifications, we show that this model boils down to a modified version of a network of Kuramoto oscillators [4]. This model is presented in Section II. We then formally show that exact phase-locking is generically impossible under proportional mean-field feedback, regardless of the oscillators interconnection topology. Nonetheless, by establishing some robustness properties of phase-locking, we explain in Section III why this feature may not be sufficient for practical DBS concerns. Focusing then on all-to-all interconnection topologies, we propose values of the feedback gain to ensure desynchronization (Section IV) and inhibition (Section V). Some conclusions and perspectives are provided in Section VI.

Notation. For a set $A \subset \mathbb{R}$ and $a \in \mathbb{R}$, $A_{\geq a}$ denotes the set $\{x \in A : x \geq a\}$. Given a vector $x \in \mathbb{R}^n$, $|x|$ denotes its Euclidean norm, that is $|x| := \sqrt{\sum_{i=1}^n x_i^2}$. For a set $\mathcal{A} \subset \mathbb{R}^n$ and $x \in \mathbb{R}^n$, $|x|_{\mathcal{A}} = \inf_{y \in \mathcal{A}} |y - x|$ denotes the point-to-set distance from x to \mathcal{A} . $\mathcal{B}(x, R)$ refers to the closed ball of radius R centered at x in the Euclidean norm, i.e. $\mathcal{B}(x, R) := \{z \in \mathbb{R}^n : |x - z| \leq R\}$. \mathbb{T}^n denotes the n -Torus. $\|u\|$ is the L^1 norm of the signal $u(\cdot)$, that

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is, if $u : \mathbb{R}_{\geq 0} \rightarrow \mathbb{R}^n$ denotes a measurable signal, locally essentially bounded, $\|u\| := \text{esssup}_{t \geq 0} |u(t)|$. A continuous function $\alpha : \mathbb{R}_{\geq 0} \rightarrow \mathbb{R}_{\geq 0}$ is said to be of class \mathcal{K} if it is increasing and $\alpha(0) = 0$. It is said to be of class \mathcal{K}_∞ if it is of class \mathcal{K} and $\alpha(s) \rightarrow \infty$ as $s \rightarrow \infty$. A function $\beta : \mathbb{R}_{\geq 0} \times \mathbb{R}_{\geq 0} \rightarrow \mathbb{R}_{\geq 0}$ is said to be of class \mathcal{KL} if $\beta(\cdot, t) \in \mathcal{K}$ for any fixed $t \geq 0$ and $\beta(s, \cdot)$ is continuous decreasing and tends to zero at infinity for any fixed $s \geq 0$. The vector with all unitary components in \mathbb{R}^n is denoted by $\mathbf{1}_n$. Given $N \in \mathbb{N}$, the set \mathbb{N}_N^\neq denotes the set $\{(i, j) \in \mathbb{N}_{\leq N} : i \neq j\}$.

II. MODEL DERIVATION AND PHASE-LOCKING UNDER MEAN-FIELD FEEDBACK

A. Kuramoto under mean-field

This note focuses on periodically spiking neurons, that is neurons generating an infinite regular train of action potentials. Even though a rich variety of behaviors exist beside this, periodic neurons are commonly considered for the analysis of neuronal synchronization. See for instance [5, Chapter 8] and [6, Chapter 10], and references therein. The dynamics underlying periodic spiking behavior can be extremely complicated. Even basic physiological models, such as the Hodgkin-Huxley model [7], consist of several coupled nonlinear differential equations. With the aim of establishing analytical results on networks with an arbitrary number of neurons controlled via electrical stimulation, we look for a simpler model still exhibiting some of the peculiarities of its more detailed counterparts.

Consider the following complex oscillator, known as Landau-Stuart oscillator, and representing a normal form of a supercritical Andronov-Hopf bifurcation:

$$\dot{z} = (i\omega_o + \rho^2 - |z|^2)z, \quad z \in \mathbb{C}, \quad (1)$$

where $\omega_o, \rho \in \mathbb{R}$. In its simple form this model captures two fundamental properties of periodically spiking neurons. First, it exhibits a stable oscillation of radius $|\rho|$ and frequency ω_o [8, Theorem 3.4.2], modeling the periodic spiking activity. Secondly, we can associate its real part to the membrane voltage, representing the measured output, and its imaginary part to a recovery variable, embedding the effects of the other variables of physiological neuron models. A similar simplification of the neural rhythm has been extensively used in the synchronization and desynchronization literature. See for instance [4], [9], [10], [11], [12], [13], [14].

While the coupling between real neurons can rely on different physical mechanisms, we focus here diffusive coupling between the oscillators, in order to derive a mathematically treatable model. The same approach has been exploited in [15], [14], [16], [17]. The model for $N \in \mathbb{N}_{\geq 1}$ coupled oscillators is then given by

$$\dot{z}_i = (i\omega_i + \rho_i^2 - |z_i|^2)z_i + \sum_{j=1}^N \kappa_{ij}(z_j - z_i), \quad \forall i = 1, \dots, N,$$

where κ_{ij} , $i, j = 1, \dots, N$, denotes the coupling gain from oscillator j to oscillator i . We denote $\omega := [\omega_i]_{i=1, \dots, N} \in \mathbb{R}^N$ as the vector of natural frequencies.

As in practice the neuronal interconnection is poorly known, we allow κ_{ij} , $i, j = 1, \dots, N$, to be arbitrary for the time being. The presence of a limited number of electrodes and their large size with respect to the neuronal scale, makes the mean-field (*i.e.* the mean neurons membrane voltages) the only realistic measurement for DBS. In the same way, the unknown distances from the neurons to the electrodes and the unknown conductivity of nearby tissues make the contribution of each neuron to the overall recording both heterogeneous and unknown. Consequently the only measurement assumed to be available for DBS is the weighted sum of the neuron membrane voltages. Associating the real part of (1) to the voltage, the output of our system is therefore

$$y := \sum_{j=1}^N \alpha_j \text{Re}(z_j), \quad (2)$$

which is referred to as the *mean-field* of the ensemble, where $\alpha := [\alpha_j]_{j=1, \dots, N} \in \mathbb{R}_{\geq 0}^N$ describes the influence of each neuron on the electrode's recording. Similarly, we define $\beta := [\beta_j]_{j=1, \dots, N} \in \mathbb{R}^N$, as the gain of the electrical input on each neuron. It is assumed to be unknown. The pair (α, β) thus defines the stimulation-registration setup. The dynamics of N coupled oscillators under mean-field feedback then reads, for all $i = 1, \dots, N$,

$$\dot{z}_i = (i\omega_i + \rho_i^2 - |z_i|^2)z_i + \sum_{j=1}^N \kappa_{ij}(z_j - z_i) + \beta_i \sum_{j=1}^N \alpha_j \text{Re}(z_j). \quad (3)$$

Relying on the assumption that the magnitude of each z_i remains equal to a constant $r_i > 0$, which is supported by a detailed argumentation in [18] and commonly made in synchronization studies [19], [20], [21], [22], [23], [4], [24], we have shown in [25] that the dynamics ruling its phase θ_i is given by

$$\dot{\theta}_i = \omega_i + \sum_{j=1}^N (k_{ij} + \gamma_{ij}) \sin(\theta_j - \theta_i) - \sum_{j=1}^N \gamma_{ij} \sin(\theta_j + \theta_i), \quad (4)$$

for all $i = 1, \dots, N$, where

$$k = [k_{ij}]_{i,j=1, \dots, N} := \left[\kappa_{ij} \frac{r_j}{r_i} \right]_{i,j=1, \dots, N} \in \mathbb{R}^{N \times N} \quad (5)$$

is referred to as the *coupling matrix*, and

$$\gamma = [\gamma_{ij}]_{i,j=1, \dots, N} := \left[\frac{\beta_i}{2} \frac{\alpha_j r_j}{r_i} \right]_{i,j=1, \dots, N} \in \mathbb{R}^{N \times N} \quad (6)$$

defines the *feedback gain*. We also define the *modified coupling matrix*, $\Gamma \in \mathbb{R}^{N \times N}$, as

$$\Gamma := [\Gamma_{ij}]_{i,j=1, \dots, N} = [k_{ij} + \gamma_{ij}]_{i,j=1, \dots, N}. \quad (7)$$

Our study is based on the *incremental dynamics* of (4), defined, for all $i, j = 1, \dots, N$, by

$$\begin{aligned} \dot{\theta}_i - \dot{\theta}_j &= \omega_i - \omega_j - \sum_{\ell=1}^N (\gamma_{i\ell} \sin(\theta_j + \theta_i) + \gamma_{j\ell} \sin(\theta_\ell + \theta_i)) \\ &\quad + \sum_{\ell=1}^N (\Gamma_{i\ell} \sin(\theta_\ell - \theta_i) - \Gamma_{j\ell} \sin(\theta_\ell - \theta_j)). \end{aligned} \quad (8)$$

B. Phase-locking and oscillating phase-locking

Roughly speaking, a phase-locked solution can be interpreted as a fixed point of the incremental dynamics (8). We distinguish solutions that exhibit collective oscillations (pathological case) from non-oscillating ones, corresponding to neuronal inhibition.

Definition 1 A solution $\{\theta_i^*\}_{i=1,\dots,N}$ of (4) is said to be *phase-locked* if it satisfies

$$\dot{\theta}_j^*(t) - \dot{\theta}_i^*(t) = 0, \quad \forall i, j = 1, \dots, N, \quad \forall t \geq 0. \quad (9)$$

A phase-locked solution is *oscillating* if, in addition, $\dot{\theta}_i^*(t) \neq 0$, for almost all $t \geq 0$ and all $i = 1, \dots, N$. If $\dot{\theta}_i^*(t) = 0$, for almost all $t \geq 0$, then the oscillator exhibits *oscillation inhibition*.

In other words, for oscillating phase-locked solutions, the discharge rhythm is the same for each neuron, which corresponds to a synchronous (pathological) activity, while in the inhibited case the neurons are in a quiescent (non pathological) state. The above definition of phase-locking corresponds to that of “Frequency (Huygens) Synchronization” [26, Definition 5.1 and Example 5.1], which is the most widely studied in the analysis of synchronization between coupled oscillators [27], [28], [29], [19], [20], [21], [22], [23], [30], [31], [32], [26], [33], [34], [35]. On the other hand, oscillations inhibition was studied in [9].

C. Existence of oscillating phase-locking

The following result, originally presented and proven in [25], states that, for a generic neuronal interconnection, the use of a proportional mean-field feedback prevents the oscillators to all evolve at the exact same frequency.

Theorem 1 For almost all natural frequencies $\omega \in \mathbb{R}^N$, for almost all interconnection matrices $k \in \mathbb{R}^{N \times N}$, and for almost all feedback gains $\gamma \in \mathbb{R}^{N \times N}$, system (4) admits no oscillating phase-locked solution.

Generically, under mean-field feedback, only two situations may thus occur: either no phase-locking or no oscillations (inhibition). This result therefore constitutes a promising feature of mean-field feedback DBS.

On the one hand, the strength of Theorem 1 stands in the generality of its assumptions: it holds for generic interconnections between neurons, including negative weights (inhibitory synapses), and does not require any knowledge neither on the contribution α_j of each neuron on the overall measurement nor on the intensity β_j of the stimulation on each neuron. On the other hand, the disappearance of the phase-locked states does not prevent a pathological behavior. Indeed, while Theorem 1 states that the perfectly synchronized behavior is not compatible with mean-field feedback, it does not exclude the possibility of some kind of “practical” phase-locking, such as solutions whose mean behavior is near to that of a phase-locked one, but with small oscillations around it. For instance, they may correspond to

phase differences which, while not remaining constant, stay bounded at all time. From a medical point of view, such a behavior for the neurons in the STN would anyway lead to tremor. We formally characterize it in the next section.

III. PHASE-LOCKING ROBUSTNESS AND NECESSARY CONDITIONS FOR DESYNCHRONIZATION

Theorem 1 ensures the disappearance of the perfectly phase-locked states under generic proportional mean-field feedback. However, for small feedback gains, the numerical observations reported in Figure 1 highlights the persistence of “nearly” phase-locked states. Even though not phase-locked, these solutions are not yet desynchronized either. In particular, they correspond to a population of neurons that fire in an approximately synchronous, and thus still pathological, manner.

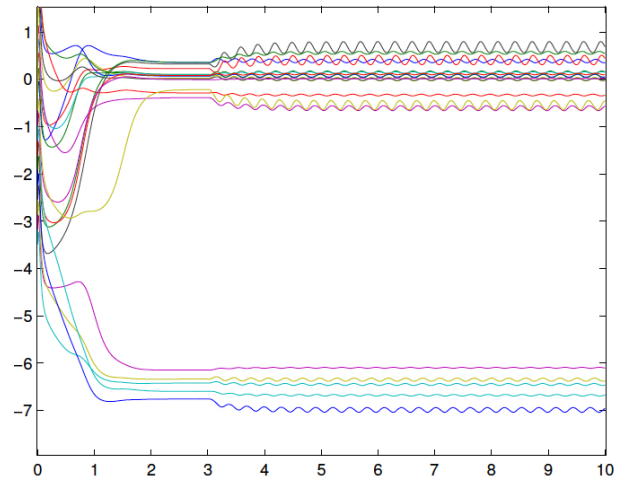


Fig. 1. Practical phase-locking in Equation (3). Each plot corresponds the evolution of a phase $\theta_i(t)$.

The evidence of this “practical” phase-locking imposes to compute necessary conditions on the feedback gain which would assure effective desynchronization. This leads us to a robustness analysis of phase-locked solutions in an oscillators population with respect to general time-varying inputs, thus including mean-field feedback as a special case.

The robustness analysis of synchronization in a finite-dimensional Kuramoto network has been the object of both analytical and numerical studies. In particular, [36] proposes a complete numerical analysis of robustness to time-varying natural frequencies, time-varying interconnection topologies, and non-sinusoidal coupling. It suggests that phase-locking exhibits some robustness to all these types of perturbations. To the best of our knowledge, analytical studies on the robustness of phase-locking in the finite Kuramoto model have been addressed only for constant natural frequencies [21], [29], [34]. The Lyapunov approach proposed in [22] for an all-to-all coupling suggests that an analytical study of phase-locking robustness can be deepened.

We start by slightly generalizing system (4) to take into

account general time-varying inputs:

$$\dot{\theta}_i(t) = \varpi_i(t) + \sum_{j=1}^N k_{ij} \sin(\theta_j(t) - \theta_i(t)), \quad (10)$$

for all $t \geq 0$ and all $i = 1, \dots, N$, where $\varpi_i : \mathbb{R} \rightarrow \mathbb{R}$ denotes the input of the i -th oscillator, and $k = [k_{ij}]_{i,j=1,\dots,N} \in \mathbb{R}_{\geq 0}^{N \times N}$ is the coupling matrix. We stress that, in this section, only nonnegative interconnection gains are considered; negative gains are assumed minority and are treated as perturbations.

Beyond the effect of the mean-field feedback, the system (10) encompasses the heterogeneity between the oscillators, the presence of exogenous disturbances and the uncertainties in the interconnection topology (time-varying coupling, negative interconnection gains, etc.). To see this clearly, let ω_i denote the (constant) natural frequency of the agent i , let p_i represent its additive external perturbations, and let ε_{ij} denote the uncertainty on each coupling gain k_{ij} . We assume that $p_i, \varepsilon_{ij} : \mathbb{R}_{\geq 0} \rightarrow \mathbb{R}$ are bounded piecewise continuous functions for each $i, j = 1, \dots, N$. Then the effects of all these disturbances, including mean-field feedback, can be analyzed in a unified manner by (10) by letting, for all $t \geq 0$ and all $i = 1, \dots, N$,

$$\begin{aligned} \varpi_i(t) = & \omega_i + p_i(t) + \sum_{j=1}^N \varepsilon_{ij}(t) \sin(\theta_j(t) - \theta_i(t)) \\ & + \sum_{j=1}^N [\gamma_{ij} \sin(\theta_j(t) - \theta_i(t)) - \gamma_{ji} \sin(\theta_j(t) + \theta_i(t))], \end{aligned} \quad (11)$$

which is well defined due to the forward completeness of (10)¹. We define the *common drift* $\bar{\omega}$ of (10) as

$$\bar{\omega}(t) = \frac{1}{N} \sum_{j=1}^N \varpi_j(t), \quad \forall t \geq 0 \quad (12)$$

and the *grounded input* as $\tilde{\omega} := [\tilde{\omega}_i]_{i=1,\dots,N}$, where

$$\tilde{\omega}_i(t) := \varpi_i(t) - \bar{\omega}(t), \quad \forall i = 1, \dots, N, \quad \forall t \geq 0. \quad (13)$$

Noticing that $\varpi_i - \varpi_j = \tilde{\omega}_i - \tilde{\omega}_j$, the evolution equation of the *incremental dynamics* ruled by (10) then reads

$$\begin{aligned} \dot{\theta}_i(t) - \dot{\theta}_j(t) = & \tilde{\omega}_i(t) - \tilde{\omega}_j(t) + \sum_{h=1}^N k_{ih} \sin(\theta_h(t) - \theta_i(t)) \\ & - \sum_{h=1}^N k_{jh} \sin(\theta_h(t) - \theta_j(t)), \end{aligned} \quad (14)$$

for all $i, j = 1, \dots, N$ and all $t \geq 0$. We use $\tilde{\theta}$ to denote the *incremental variable*

$$\tilde{\theta} := [\theta_i - \theta_j]_{i,j=1,\dots,N, i \neq j} \in \mathbb{T}^{(N-1)^2}. \quad (15)$$

A general robustness analysis of (14) has been conducted in [37], [25]. Roughly speaking, it states that phase-locking

¹The forward completeness of (10) follows by the fact that (10) is a Lipschitz continuous periodic dynamics, and thus bounded and globally Lipschitz.

is robust (namely, locally input-to-state stable) to sufficiently small exogenous inputs, including mean-field feedback. In the case of all-to-all case, explicit bounds on the amplitude of the tolerated inputs and on the region of attraction can be provided. This is summarized by the following result.

Proposition 1 *Consider the system (10) with the all-to-all interconnection topology, i.e. $k_{ij} = k_0 > 0$ for all $i, j = 1, \dots, N$. Then, for all $\epsilon \in [0, \frac{\pi}{2}]$, and all $\tilde{\omega}$ satisfying*

$$\|\tilde{\omega}\| \leq \delta_\omega^\epsilon := \frac{k_0 \sqrt{N}}{\pi^2} \left(\frac{\pi}{2} - \epsilon \right), \quad (16)$$

the following facts hold:

- 1) the set $\mathcal{D}_\epsilon := \left\{ \tilde{\theta} \in \mathbb{T}^{(N-1)^2} : |\tilde{\theta}|_\infty \leq \frac{\pi}{2} - \epsilon \right\}$ is forward invariant for the system (14);
- 2) for all $\tilde{\theta}_0 \in \mathcal{D}_0$, the set \mathcal{D}_ϵ is attractive, and the solution of (14) satisfies

$$|\tilde{\theta}(t)| \leq \frac{\pi}{2} |\tilde{\theta}_0| e^{-\frac{k_0}{\pi^2} t} + \frac{\pi^2}{k_0} \|\tilde{\omega}\|, \quad \forall t \geq 0.$$

Proposition 1 establishes the exponential input-to-state stability (ISS, [38]) of the synchronized state in the all-to-all Kuramoto model with respect to time-varying inputs whose amplitudes are smaller than $\frac{k_0 \sqrt{N}}{2\pi}$. It holds for any initial condition lying in \mathcal{D}_0 , that is when all the initial phase differences lie in $[-\frac{\pi}{2}, \frac{\pi}{2}]$. Moreover, if the inputs amplitude is bounded by δ_ω^ϵ , for some $0 \leq \epsilon \leq \frac{\pi}{2}$, then the set \mathcal{D}_ϵ is forward invariant and all the solutions starting in \mathcal{D}_0 actually converge to \mathcal{D}_ϵ .

Proposition 1 partially extends the main results of [29], [34] to time-varying inputs. On the one hand, it allows to consider sets of initial conditions larger than those of [29], and bounds the convergence rate by a strictly positive value, independently of the region of attraction. On the other hand the required coupling strength is comparable to the one given in [29], but more conservative than the lower bound in [34]. Finally for small regions of attraction, the bound on the convergence rate obtained in Proposition 1 is not as good as the one of [29], [34]. Details can be found in [25].

Since proportional mean-field feedback can be included in the perturbation (11), Proposition 1 provides a necessary condition, in terms of the mean-field feedback amplitude, for actual desynchronization of the neuronal population. The next section is devoted to providing sufficient conditions.

IV. DESYNCHRONIZATION

In this section, we explore the possibility of eliminating the pathological neuronal synchronization by effectively desynchronizing the population activity.

While desynchronization owns quite an intuitive meaning, its formal definition is not straightforward. One way of guaranteeing sufficient disorder in a network of oscillators is to induce chaos in the incremental dynamics of their outputs (i.e. the dynamics ruling the phase differences of each pair of oscillators). This is the approach followed by chaotification techniques, cf. e.g. [39], [40]. However, chaos may be too strong a requirement in some particular applications and most

anti-control techniques may require too much knowledge on the oscillators state to be practically implemented in a DBS device.

On the other hand, simply guaranteeing that phases are not synchronized is not enough in most practical applications. To see this, consider a pair of oscillators whose phases difference, although not constant, remains at all times in a small neighborhood of a given value (cf. Figure 1). In this case, all classical definitions of synchronization are violated as the oscillators are neither phase synchronized [41], nor phase-locked [25] or frequency-synchronized [42], as their phases difference is not constant. Nevertheless, for practical concerns, such a system cannot be considered as desynchronized since the phases difference remains “almost constant” at all times. In fact, such a situation would rather correspond to “approximative synchronization” as defined in [42]. In a nutshell, desynchronization is not simply the negation of synchronization.

In [43], two different notions of desynchronization were given for general networks of phase oscillators. The first one, called *strong desynchronization*, requires that the instantaneous frequency difference $|\dot{\theta}_i(t) - \dot{\theta}_j(t)|$ be lower bounded away from zero for each pairs of oscillators, which imposes that phases drift away from one another at all times. Nonetheless, this requirement may happen to be too harsh a constraint in practice. The permanent phase drift imposed by strong desynchronization impedes the instantaneous frequencies to be equal even on short time intervals. Intuitively, such a frequency similarity would not affect the overall desynchronization if it happens sufficiently rarely. Hence, we relax that definition by replacing the pointwise requirement by the less restrictive constraint that the difference of frequencies be bounded from below *in average*, uniformly over some moving window of length T .

Definition 2 A pair $(i, j) \in \mathbb{N}_N^\neq$ of oscillators is said to be *practically desynchronized* for (10) if there exists $\Omega_{ij}, T_{ij} > 0$ such that, for all $\theta_0 \in \mathbb{T}^N$ and $t_0 \in \mathbb{R}$,

$$\frac{1}{T_{ij}} \left| \int_t^{t+T_{ij}} \left(\dot{\theta}_i(\tau; t_0, \theta_0) - \dot{\theta}_j(\tau; t_0, \theta_0) \right) d\tau \right| \geq \Omega_{ij}, \quad (17)$$

for all $t \in \mathbb{R}$. Given $m \in \left\{ 1, \dots, \frac{N(N-1)}{2} \right\}$, the network of coupled phase oscillators (10) is said to be *m-practically desynchronized* if it contains m distinct pairs of practically desynchronized oscillators. If $m = \frac{N(N-1)}{2}$ then (10) is said to be *completely practically desynchronized*.

While Theorem 1 established that, for almost all interconnection topology and almost all value of the feedback gain, phase-locking is impossible under mean-field feedback, the next theorem states that practical desynchronization can actually be achieved. In other words, we give a sufficient condition to ensure that a given pair of oscillators is practically desynchronized.

Theorem 2 Suppose that there exists $(i, j) \in \mathbb{N}_N^\neq$, such that

$$|\omega_i - \omega_j| > \sum_{h=1}^N |\gamma_{ih} + \gamma_{jh}| \left(\frac{\pi\nu}{2\omega_*} + \frac{\nu^2}{6\omega_*^2} \right) + |\varepsilon_{ih} + \varepsilon_{jh}|,$$

where

$$\begin{aligned} \nu &:= 2 \max_{h=1, \dots, N} \left(|\tilde{\omega}_h| + \sum_{h'=1}^N |\gamma_{hh'} + \varepsilon_{hh'}| \right) \\ \omega_* &:= \frac{1}{N} \sum_{h=1}^N \omega_h \\ \tilde{\omega}_h &:= \omega_h - \omega_*, \quad \forall h = 1, \dots, N \\ \varepsilon_{hh'} &:= k_{hh'} + \gamma_{hh'}, \quad \forall h, h' = 1, \dots, N, . \end{aligned}$$

Then the pair of oscillators (i, j) is *practically desynchronized* for (4).

Theorem 2, proved in [43], states that, if the natural frequencies discrepancy is large enough, then practical desynchronization occurs. It is worth stressing that, from a control perspective, this result gives a hint on how to select the feedback gain in order to efficiently achieve desynchronization. Nonetheless, the gains γ_{ij} are not tunable at will since only a scalar stimulation input is available (cf. Equation (6)). The main requirement of Theorem 2 thus depends on structural properties of the oscillators population and may not be always achievable.

However, in the case when the coupling is given by the all-to-all topology, and each oscillator contributes in the same way at the measured mean-field and receives the input with same intensity, the interconnection and feedback gains become $k_{ij} = k_0$ and $\gamma_{ij} = \gamma_0$, for all $i, j = 1, \dots, N$. For all $i = 1, \dots, N$, the dynamics (4) then reduces to

$$\dot{\theta}_i = \omega_i + (k_0 + \gamma_0) \sum_{j=1}^N \sin(\theta_j - \theta_i) - \gamma_0 \sum_{j=1}^N \sin(\theta_j + \theta_i).$$

The diffusive coupling term can be eliminated by choosing $\gamma_0 = -k_0$, and (4) boils down to

$$\dot{\theta}_i = \omega_i + k_0 \sum_{j=1}^N \sin(\theta_j + \theta_i), \quad \forall i = 1, \dots, N. \quad (18)$$

In this particular situation, the following corollary provides a structural condition for practical desynchronizability by proportional mean-field feedback. See [43] for its proof.

Corollary 1 Suppose that there exists $(i, j) \in \mathbb{N}_N^\neq$, such that

$$|\omega_i - \omega_j| > 2Nk_0 \left(\frac{\pi\nu}{2\omega_*} + \frac{\nu^2}{6\omega_*^2} \right), \quad (19)$$

where ω_* and $\tilde{\omega}_h$, $h = 1, \dots, N$, are as in Theorem 2 and

$$\nu := 2 \max_{h=1, \dots, N} (|\tilde{\omega}_h| + Nk_0).$$

Then the pair of oscillators (i, j) is *practically desynchronized* for (18).

We stress that the inequality (19) is always satisfied provided that $\omega_i \neq \omega_j$ and $\tilde{\omega}$ is sufficiently large or the coupling gain k_0 is small enough.

V. NEURONAL INHIBITION

While the previous section aimed at exploiting mean-field feedback to desynchronize the neuronal population, we now investigate the possibility to inhibit the neuronal oscillations. In other words, our aim here is to use the DBS signal to inactivate the neurons, i.e. to impeded pathological bursting and spiking. Such an approach basically results as a functional lesion of the STN. This hypothesis is supported by the fact that, before the invention of DBS, the surgical PD treatment consisted in an ablation of the cerebral zone under concern, which can be seen as a radical neuronal inhibition. The aim of this section is thus to provide some insights on how the collective oscillation of a network of nonlinear oscillators, modeling a neuronal population, can be inhibited by proportional feedback when only its mean-field is measured.

Oscillation inhibition corresponds to the existence of (almost globally) attractive fixed points for the phase dynamics (4). We focus here on the all-to-all dynamics and pick the feedback gain γ_0 as $-k_0$. As seen in the previous section the dynamics (4) then boils down to (18). We start by identifying the fixed points of (18) and we study their nature in the fictitious case of zero natural frequencies. Stability properties in the case of non-zero natural frequencies are derived as a second step, by relying on robustness arguments.

A. The case of zero natural frequencies

When the natural frequencies of the oscillators are all zero, i.e. $\omega_i = 0$ for all $i = 1, \dots, N$, (18) reduces to

$$\dot{\theta}_i = k_0 \sum_{j=1}^N \sin(\theta_i + \theta_j), \quad \forall i = 1, \dots, N. \quad (20)$$

We note that (20) can be equivalently written as the gradient system

$$\dot{\theta}_i = -\frac{\partial W}{\partial \theta_i}(\theta), \quad \forall i = 1, \dots, N,$$

where the function W is given, for all $\theta \in \mathbb{R}^N$, by

$$W(\theta) := -k_0 \sum_{i,j=1}^N \sin^2\left(\frac{\theta_i + \theta_j}{2}\right). \quad (21)$$

It can be seen that the global minima of W are the elements of the set

$$\mathcal{W}_m := \left\{ \theta \in \mathbb{R}^N : \theta = \left(\frac{\pi}{2} \bmod \pi\right) \mathbf{1}_N \right\}. \quad (22)$$

The gradient dynamics nature of (20) is extensively used in [44] to establish the following result.

Proposition 2 *Given any $k_0 > 0$, the set \mathcal{W}_m defined in (22) is almost globally asymptotically stable for the dynamics (20).*

Proposition 2, whose proof can be found in [44], states that, when neglecting the natural frequencies of the oscillators, the choice $\gamma_0 = -k_0$ of the mean-field feedback gain yields oscillation inhibition for almost all initial conditions, that is the oscillators phases converge almost globally toward an asymptotically stable configuration.

B. In presence of small natural frequencies

1) Odd number of oscillators:

The following theorem, proved in [44], states that the existence of an almost globally asymptotically stable is preserved in presence of sufficiently small natural frequencies, when the number of oscillators is odd.

Theorem 3 *Let $N \in \mathcal{N}_{\geq 3}$ be odd. Then there exists $\delta > 0$ and a class \mathcal{K}_∞ function ρ such that, if the vector of natural frequencies $\omega \in \mathbb{R}^N$ satisfies $|\omega| \leq \delta$, then there exists a set of isolated points \mathcal{W}_m^ω , such that*

$$|\theta|_{\mathcal{W}_m} \leq \rho(|\omega|), \quad \forall \theta \in \mathcal{W}_m^\omega,$$

where \mathcal{W}_m is defined in (22). Moreover, this set \mathcal{W}_m^ω is almost globally attractive for (18), that is, for almost all $\theta_0 \in \mathbb{R}^N$, it holds that

$$\lim_{t \rightarrow \infty} |\theta(t, \theta_0)|_{\mathcal{W}_m^\omega} = 0.$$

Theorem 3 thus formally establishes the possibility to inhibit oscillations in presence of non-zero natural frequencies, at least when the number of oscillators is odd. Note that it guarantees, for almost all $\theta_0 \in \mathbb{R}^N$, that the solution of (18) satisfies

$$|\omega| \leq \delta \quad \Rightarrow \quad \limsup_{t \rightarrow \infty} |\theta(t, \theta_0)|_{\mathcal{W}_m} \leq \rho(|\omega|),$$

meaning that the fixed points to which θ converge is arbitrarily near from the original attractive set \mathcal{W}_m provided that the natural frequencies are sufficiently small.

A similar oscillation inhibition result is contained in [24]. In that reference the authors consider a chain of phase oscillators, and a class of coupling functions that contains the sinusoidal additive coupling considered here as a special case. The result is the existence of a unique stable inhibited solution in the chain of oscillators, provided that the natural frequencies are sufficiently small. Theorem 3 complements this analysis, by restricting to a particular coupling function and considering a different interconnection topology.

2) Even number of oscillators:

In the case of an odd number of oscillators, the set of non-isolated fixed point of the unperturbed system (20) defines a normally hyperbolic invariant manifold. This observation is instrumental in the robustness analysis conducted to establish Theorem 3 (see [44] for details). This feature no longer holds in case of an even number of oscillators. At this stage, we can only conjecture the following.

Conjecture 1 *The result of Theorem 3 holds also in the case when N is even.*

VI. CONCLUSION

Motivated by the objective of developing a closed-loop approach for deep brain stimulation, we have introduced a phase model that captures the rhythmic neuronal oscillation

of the considered population and allows to analyze its synchronization properties. We have shown that a proportional mean-field feedback can bring a pathologically synchronous neuronal population to either a desynchronized state or to a silent inhibited state. More precisely, we have characterized the pathological states in terms of oscillating phase-locked solutions. The existence of such states has been shown to be generically incompatible with any nonzero proportional mean-field feedback, thus supporting the proposed control strategy. Nonetheless, the robustness analysis we have conducted shows that the pathological states can persist as practically phase-locked solutions and provides necessary conditions for desynchronization via mean-field feedback. Under some simplifying assumptions, we have described how the presence of mean-field feedback can actually achieve neuronal inhibition. We have also derived sufficient conditions to achieve effective desynchronization via proportional mean-field feedback. Future works will aim at extending this analysis to finer neuronal models.

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