Noise effects on robust synchronization of a small pacemaker neuronal ensemble via nonlinear controller: electronic circuit design

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Abstract In this paper, we report on the synchronization of a pacemaker neuronal ensemble constituted of an AB neuron electrically coupled to two PD neurons. By the virtue of this electrical coupling, they can fire synchronous bursts of action potential. An external master neuron is used to induce to the whole system the desired dynamics, via a nonlinear controller. Such controller is obtained by a combination of sliding mode and feedback control. The proposed controller is able to offset uncertainties in the synchronized systems. We show how noise affects the synchronization of the pacemaker neuronal ensemble, and briefly discuss its potential benefits in our synchronization scheme. An extended Hindmarsh–Rose neuronal model is used to represent a single cell dynamic of the network. Numerical simulations and Pspice implementation of the synchronization scheme are presented. We found that, the proposed controller reduces the stochastic resonance of the network when its gain increases.

Keywords Noise · Synchronization · Pacemaker neuronal ensemble · Sliding mode · Feedback control · Pspice simulation

Introduction

In all animal species, intercellular communication represents one of the most important characteristics, because it allows many components of such systems to spread together. There exist many type of intercellular communication, but brain cells (neurons) communication from which information are mediated by electrical synapses, are the main topic of our study.

Several intracellular mechanisms exist and they influence directly or indirectly the electrical activity of neurons in different time scales. The dynamics of the ionic channels present in the cell are generally described by realistic models of neurons. Among these channels, there are some depending on the concentration of cytoplasmic calcium, for which its dynamics is frequently described by the first order kinetic equation (Varona et al. 2001).

Many phenomena such as synchronization, competition, intermittency and resonance are used by neuronal systems (Rabinovich et al. 2006). In fact, the dynamics of these neuronal modules are generally non autonomous and for instance are forced by different kind of signals. Several experimental studies showed that synchronization plays an important role in information transmission by a large ensemble of neurons (Gray et al. 1989; Meister et al. 1991; Kreiter and Singer 1996; Elson et al. 1998). Recently, Baptista et al. (2008), proposed a theoretical approach to quantitatively relate information, synchronization and network topology; their study were based in active networks. Since internal or external input can produce synchronous

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behaviors in neuronal networks, it is of great interest to study the stimuli effect on intrinsic physiological rhythms (Gass 2001).

Networks specialized for the production of rhythmic motor patterns are called central pattern generators (CPGs). They are found in all nervous systems, and are able to produce independently, rhythmic spacio-temporal motor patterns. This behavior is observed in systems without rhythmic sensory feedback (Selverston et al. 2000; Delcomyn 1980). The patterns are highly influenced by sensory feedback and by chemical neuromodulatory substances. The fundamental structure of the generated patterns is fixed by biophysical properties and the synaptic connections of the individual neuron. These properties are important in finding the timing and organization of the motor patterns. They can be distorted chemically, involving the fact that, exposition of CPGs to chemical modulators can reconfigure their functionalities. Hence, many different stable modes of activity can be reproduced by one anatomical circuit. This involves an analytic challenge because in healthy animals, the central nervous system (CNS) neuron's properties remains in constant state of flux (Harris-Warrick et al. 1992). CPGs in general, need the presence of one or more modulators to function normally; so if the modulator are influenced, they will usually be inoperative. One of the neuroscience challenge is to provide CPGs with more modulation, so that they stay rhythmically active, while the chemical environment remains constant and the rhythms stay stable for extended period. Interpreting neurodynamics: concepts and facts has been reviewed by Harald and Stefan (2008). They discussed conceptual issues in neurodynamics that are helpful for an appropriate interpretation and evaluation of its results. Their importance for selected topics of theoretical and empirical works was demonstrated. Particularly, notion such as stochasticity and determinacy in neurodynamics were analyzed from microscopic, mesoscopic to macroscopic descriptions. They also propose a rich discussion of conceptual foundations in neurobiological, results as a good step to a profitable future neuroscience's philosophy. Moreover, in 2012, Kryukov gave a short review on a unified model of pavlovian conditioning. In his paper and in a brief form, eight representatives models of trace conditioning are analyzed (Kryukov 2012). It results that one of them presenting the most important characteristics of its forerunner can be considered as a real candidate for a unified model of trace conditioning.

Generally, more or less independent microcircuits (MCs) must synchronize in order to improve some coordination functions. Most of these microcircuits have been studied, from which there are some generating oscillatory dynamics and rhythmic behavior (central pattern generators). They represent one of the two subnetworks of the

stomatogastric ganglion (STG) of the lobster. The later is known as the basis model, used for several types of analysis (Szucs et al. 2001; Elson et al. 2002; Cazalets et al. 1990; Sto-Trevino et al. 2005). A small ensemble of fourteen coupled neurons that communicate with each other via both electrical and chemical interactions, represents the main group of the lobster's CPGs. Between these fourteen coupled neurons, we have one ventricular dilator (VD), two pyloric dilators (PD), one lateral pyloric (LP), one inferior cardiac (IC), eight pyloric neurons (PY) and one anterior burster (AB). AB and PD neurons constitute a subset of three electrically coupled neurons, and are considered as the main pacemaker group of the CPG. Using the pharmacological tools, this small group of neurons can be dissociated from all synaptic inputs coming from their surrounding. Recently, some interactions between the gastric circuit and the pyloric network, and between the anterior ganglia and pyloric network were simulated in some respects, by the periodic forcing of the pacemaker at low frequencies (Szucs et al. 2001). In 2000, the synchronous behavior of a hybrid network composed of an electronic neuron bidirectionally coupled to a PD neuron in the pacemaker group of the pyloric CPGs was investigated by Pinto et al. In their study, the procedure used to separate the STG from its associated anterior ganglia was to cut the stomatogastric nerve. They found that the electronic and the biological neurons, behave similarly when coupled in the same manner (Pinto et al. 2000).

Due to its diverse applications, synchronization phenomenon in complex network has attracted great attentions (Motter et al. 2006; Restrepo et al. 2006; Arenas et al. 2006; Chen and Zhou 2006; Bowong and Kakmeni 2004, 2003; Fotsin and Daafouz 2005; Louodop et al. 2013; Tchitnga et al. 2013; Louodop et al. 2014; Megam et al. 2014b, a). Controlled synchronization, with the aim of imposing a desired behavior to a member of a network and then inferring the same behavior in the whole network, represents a recent problem in complex networks (Cornejo-Perez et al. 2012). The main goal of this work is to study the synchronized behavior of an external neuron and a complex network constituted of the pacemaker group neurons of the lobster's pyloric CPG network, with linearly coupled nodes. The synchronization dynamics is achieved by controlling one neuron of the sub-network. The desired dynamics to induce into the whole network is described by an external neuron. Frequency or energy dissipation or absorption between the master and the controlled neurons of the system should be done with some rhythm in order to infer synchronization; moreover, the required rhythm is ruled by the controller. The controlled neuron in this way is equipped with the particular rhythm and the particular frequencies of the reference neuron; though, the uncoupled neurons fire with the rhythm from the interconnection with

other sub-network's neurons. We also presented in this work the Pspice implementation of the synchronization process of the controlled system to prove the experimental design of the synchronization scheme.

Going back to the work of Stratonovich (1965, 1961) where the interest was on how noise could disrupt oscillatory radio circuits, there is a long history of the study of noise in oscillators. From the Oxford English Dictionary, noise can be defined as a “random or irregular fluctuations or disturbances which are not part of a signal or which interfere with or obscure a signal or more generally any distortions or additions which interfere with the transfer of information”. Our second focus in this paper, concerns how noise affects neural oscillators, when coupled to each other. Furthermore, we will mainly consider the activity when the noise and coupling are weak and the oscillators are nearly identical. This allows one to treat each oscillator as a single variable coding its phase and to significantly reduce the dimensionality of the problem. Notice that, noise is not only a problem for neurons, other information issues can find solution when studying its effects on the network synchronization (Faisal et al. (2008)). There exist several strategies to employ noise to this end. It has been shown for example that, stochastic resonance represents a process by which the ability of threshold-like systems to detect and transmit periodic signal can be improved by the presence of certain level of noise (Faisal et al. 2008; Shu et al. 2003; Benzi et al. 1981). In 2009, Hatsuo and Jun studied LTD windows of the STDP learning rule and synaptic connections having a large transmission delay (Hatsuo and Jun 2009). They found that, the LTD windows of the STPD rules enable robust sequence learning amid background noise in cooperation with a large signal transmission delay between neuron and theta rhythm. In their research, they considered a network model of the entorhinal cortex layer II with entorhinal–hippocampal loop connections. Walter J. did in 2009 the deep analysis of perception through dynamic structures that emerge in cortical activity from self-regulated noise Walter (2009). Starting with random numbers, containing the distributions and going through the characteristic deviations from randomness in electrocorticogram (ECoG) of subjects at rest and during intentional behaviors, the statistical properties of the spontaneous background electrocorticogram were modeled. They found at the presence of noise that, the beats in dynamics theory are deemed essential for perception, by gating beta and gamma bursts at theta rates via enhancement of the cortical signal-to-noise ratio. Few years later, Martin et al. proposed to combine histogram equalization and multi-condition training for robust keyword detection in noisy speech (Martin et al. 2011). They found that contextual information can effectively be exploited in a multi-stream automatic speech recognition

(ASR) framework. The latter usually models dynamically, context-sensitive phoneme estimates generated by a long short-term memory neural network.

The simple neural dynamics, in each neuron of the network is represented by the extended four-dimensional Hindmarsh–Rose (eHR) neuronal model (Pinto et al. 2000). This model have been recently used to study synchronous behavior of coupled electronic neurons and the dynamics of interacting biological and electronic neurons (Pinto et al. 2000; Szucs et al. 2001; Megam et al. 2014b, 2015, 2016). It can reproduce the observed behavior of realistic neuronal membrane voltage activities (Pinto et al. 2000; Fakle et al. 2000). The overall organization of the paper is as follows. We start in section “[Description of the neuronal model and the pacemaker network](#)” by the presentation of the mathematical model of the eHR neuron, followed by the description of the lobster's pyloric network and its pacemaker group neurons. The synchronization problem of the external neuron and the complex network is presented in section “[Robust synchronization problem formulation and design of synchronization scheme](#)”. Robust synchronization is got through the construction of a high gain observer. In section “[Numerical simulations and Pspice implementation of the synchronization scheme](#)”, Pspice implementation and numerical simulations of the synchronization scheme are shown. We then look at how correlated noises can affect the synchronization of the coupled oscillators in section “[Noise effects on the synchronization](#)”. Concluding remarks are made in last section.

Description of the neuronal model and the pacemaker network

As we have said, an extended Hindmarsh–Rose (HR) model is considered in this manuscript (Fakle et al. 2000; Pinto et al. 2000; Hindmarsh and Rose 1984; Megam et al. 2014b, 2015, 2016). It is described with four order dynamical variables:

$$\begin{aligned}\dot{x} &= ay + bx^2 - cx^3 - dz + I_{DC}, \\ \dot{y} &= e - fx^2 - y - gw, \\ \dot{z} &= \mu[-z + s(x + h)], \\ \dot{w} &= v[-kw + r(y + l)].\end{aligned}\tag{1}$$

Here, $a, b, c, d, e, f, g, \mu, s, h, v, k, r$ and l , are constants which express the current and conductance based dynamics. I_{DC} represents the injected current. We have chosen this model since it reproduces the observed aspect of neuronal membrane voltage activity and the complicated current voltage relationships of the conductance-based model are replaced by polynomials (Fakle et al. 2000;

Pinto et al. 2000). x represents the membrane voltage, and y a fast current. z is a slow current since $\mu \ll 1$. w is a slower dynamical process as $v < \mu \ll 1$. It is considered, because slow processes representing the calcium exchange between intracellular warehouse and the cytoplasm are required in the HH model to completely produce the chaotic behaviors of the stomatogastric ganglion neurons (Chen and Zhou 2006; Megam et al. 2015, 2016). The fourth dimensional model presents much larger region of parameters' space where chaos appears than the three-dimensional one, even if both behave chaotically. Thus, the following parameters' values were considered: $a = 1.0$; $b = 3.0$; $c = 1.0$; $d = 0.99$; $e = 1.01$; $f = 5.0128$; $g = 0.0278$; $\mu = 0.00215$; $s = 3.966$; $h = 1.605$; $v = 0.0009$; $k = 0.9573$; $r = 3.0$ and $l = 1.619$. Figure 1 presents a chaotic time series of bursting activity of the model using the above variables with $I_{DC} = 3.0249$. For this value of the injected current, the neuron's dynamics is chaotic since the corresponding maximal Lyapunov exponent is positive ($\lambda_{max} = 0.000327$). The complexity presented by the consideration of the fourth dynamical variable w can be observed in different three dimensional phase spaces: (x, y, z) , (x, y, w) and (x, z, w) .

CPGs represent neural circuits, consisting of hundreds and thousands of neurons with unknown detailed connectivities. On the other hand, the simplest one such as the gastric and pyloric CPGs in crustaceans contain dozens of cells. Modeling CPGs in this situation, becomes a challenging problem. Nevertheless, phase relationships between the different elements of the pattern simplified models can be used to study these properties, with the price of ignoring the others, when only some particular properties of the circuit are under consideration. From these perspectives, Selverston and Moulins described the connectivities in the crustaceans CPG circuit as represented in Fig. 2a (Selverston and Moulins 1987). In this circuit, we have one AB, two PDs, one LP, one IC, one VD and eight PYs, giving an ensemble of fourteen connected neurons (Selverston and Moulins 1987). Furthermore, from this circuit, one can identify the pacemaker group (AB/PDs neurons), coupled through electrical connections, while the other neurons are weakly connected through inhibitory chemical synapses. These synapses organize the fourteen neurons into a functional unit given as a chain of four reciprocally inhibitory types of neurons. Moreover, in this network the AB is an interneuron, while the rest are motor neurons. Our aim here, is to synchronize this pacemaker group when it is isolated from other networks neurons using pharmacological tools, with a master neuron through a control command. Since, AB and PDs neurons are coupled via electrical synapses (see Fig. 2b), we consider that this small network can be represented as:

Fig. 1 **a** Time evolution of the membrane potential $x(t)$ of the four dimensional HR model; **b–d** the corresponding three dimensional phase diagrams

$$\dot{x}_i = F(x_i) + g_E \sum_{j=1, i \neq j}^N G_{ij} H(x_i, x_j). \quad (2)$$

N is the total number of nodes ($N = 3$), $i, j = 1, 2, \dots, N$, x_i are the state variables of the i th (respectively the j th) neuron, $F: \mathbb{R}^4 \rightarrow \mathbb{R}^4$ represents the vector field. The way neurons are electrically connected is described by the matrix G_{ij} . $G_{ij} = 1$ if neuron i and j are connected, and

Fig. 2 **a** The electronic equivalent diagram of the pyloric circuit. It consists of fourteen neurons: one AB, two PDs, one LP, one IC, one VD, and eight PYs. Every synapse between different neurons is shown; Resistors represent electrical couplings, while dots represent inhibitory chemical couplings. **b** The pacemaker ensemble of the crustaceans pyloric CPG. It consists of three electrically coupled neurons, the AB neuron and two PD neurons. This group is isolated from others through pharmacological tools

$G_{ij} = 0$ otherwise (notice that $G_{ii} = 0$ here). The coupling function: $H(x_i, x_j)$ which represents the gap junction between neurons, is given by:

$$H(x_i, x_j) = (x_j(t) - x_i(t)). \quad (3)$$

Robust synchronization problem formulation and design of synchronization scheme

The uncontrolled nodes in the network are seen as perturbations entering the controlled node. The principal role of the control command is to offset for such perturbations so that the error system always stabilizes to the origin. Due to the fact that the control command dynamics is considered as uncertain, it has been demonstrated that the subsystem

constituted of the non controlled nodes does not affect its design. We use in this work, a robust synchronization scheme, which is combined with high-order sliding mode-based time differentiator, used to estimate and linearize feedback control. To illustrate the main results, we firstly revisit some preliminaries. Let us consider the following lemma.

Lemma 1 We consider the following single input single output nonlinear system (Isidori 1989):

$$\begin{aligned} \dot{x} &= f(x) + g(x)u, \\ y &= h(x), \end{aligned} \quad (4)$$

x , represents the state vector, $f: \mathbb{R}^n \rightarrow \mathbb{R}^n$ is a smooth function representing the vector field of the uncontrolled system. u is a control command and y represents the system output or the system measured state. We suppose that there exists a smallest ρ for which the following conditions hold:

1. $L_g L_f^i h(x) = 0, i = 1, 2, \dots, \rho - 2$,
2. $L_g L_f^{\rho-1} h(x) \neq 0$.

$L_f^i h(x) = L_f(L_f^{i-1} h(x))$ represents the Lie derivative of $h(x)$ with respect to $f(x)$. The guarantee that the smoothness assumption and the above conditions are satisfied, induce that it is possible to obtain $(n - \rho)$ functions $\psi_{\rho+1}, \psi_{\rho+2}, \dots, \psi_{\rho+n}$ satisfying $L_g \psi_i \neq 0, i = \rho + 1, \dots, n$ and such that the error system associated to the dynamics (4) can be written in the following canonical form:

$$\begin{aligned} \dot{p}_i &= p_{i+1}, i = 1, 2, \dots, \rho - 1, \\ \dot{p}_\rho &= \kappa(p, \zeta) + \beta(p, \zeta)u, \\ \dot{\zeta} &= \phi(p, \zeta), \\ y &= p_1, \end{aligned} \quad (5)$$

with $\beta(p, \zeta) \neq 0$ and $\zeta \in \mathbb{R}^{n-\rho}$. $\kappa(p, \zeta) = L_f^\rho h(x)$, $\beta(p, \zeta) = L_g L_f^{\rho-1} h(x) \neq 0$. The matrix $\phi(p, \zeta) = [\psi_{\rho+1}, \psi_{\rho+2}, \dots, \psi_{\rho+n}]$, is such that the coordinate transformation $\psi(x) = [h(x), L_f h(x), \dots, L_f^{\rho-1} h(x), \psi_{\rho+1}, \psi_{\rho+2}, \dots, \psi_{\rho+n}]$ is globally invertible. The zero dynamics of the system is given by $\dot{\zeta} = \phi(0, \zeta)$. Note that the system is fully linearizable when $\rho = n$, and when $\rho < n$, it is called partially linearizable. ρ represents here the relative degree of the system.

Consider the chaotic master system:

$$\begin{aligned} \dot{x}_m &= f(x_m, \pi_m), \\ y_m &= h(x_m). \end{aligned} \quad (6)$$

where, $x_m = [x_{1m}, x_{2m}, \dots, x_{nm}]^T \in \mathbb{R}^n$ represents the state vector, $y_m = h(x_m) \in \mathbb{R}$ is the system output, π_m is the vector of parameters and f is a smooth nonlinear function. Let us choose the slave system with the same order as the master and defined as:

$$\begin{aligned}\dot{x}_s &= f(x_s, \pi_s) + g(x_s)u, \\ y_s &= h(x_s).\end{aligned}\quad (7)$$

where $x_s = [x_{1s}, x_{2s}, \dots, x_{ns}]^T \in \mathbb{R}^n$, defines the state vector, π_s the vector of parameters, $g(x_s)$ a vector of proper length which define the control channel. $u(t)$ is the control action and $y_s = h(x_s)$ the output.

We consider that the measurement of the nearness of the master system, to the slave system is given by the variable $e = x_s - x_m$. Subtracting equation (7) from equation (6), we obtain the following error dynamics:

$$\begin{aligned}\dot{e} &= f(e + x_m, \pi_s) - f(x_m, \pi_m) + g(x_s)u, \\ y &= h(e).\end{aligned}\quad (8)$$

The difference between the master and the slave systems is denoted by: $\Delta f = f(e + x_m, \pi_s) - f(x_m, \pi_m)$, while $y = h(e)$ represents the error output. The synchronization between the master (6) and the slave (7) is reduced to the stabilization of the error system (8). The problem is to design a suitable feedback control command $u(t)$, to guarantee robust global stability of the error system (8) to the origin. To ensure the synchronization's objective, the following hypothesis are made.

Assumptions:

- (i) Only the first system state is measurable i.e. $y = h(x) = x_1$.
- (ii) Some unknown model mismatches between the master and the slave exist: $\Delta f = f(x_s, m) - f(x_m, q) \neq 0$, where subscript s and m stand for the slave and the master systems respectively.
- (iii) The error system can be transformed into a canonical form, which means that there exist a diffeomorphism transformation of coordinates given by system (8).
- (iv) The error system (8) is a minimum phase system.

Remark 1 In real communication systems, the improvement of the security is reached when only a scalar time series is transmitted to build the synchronization between the master and the slave. Due to the restriction of measuring condition, only the membrane potential is available for a neuron. Hence, the assumption (i) is suitable in practical cases.

Remark 2 In practical, mismatches between two electronic circuits is unavoidable, since there always exist some differences between two components, thus mismatches between the master and the slave exist in real cases i.e. Δf always exist.

Remark 3 Through a mathematical transformation, many chaotic systems can be changed into a canonical form. Since we deal in our work with chaotic dynamical systems, and from the fact that the error dynamics are obtained from the latter, it can also be transformed into this canonical form.

Remark 4 The asymptotic stability of the zero dynamic $\dot{\zeta} = \phi(0, \zeta)$, describes the minimum phase behavior of a system. Nevertheless, this condition is strong and it can be satisfied in some chaotic systems.

According to Fermat et al. (1999) and by lumping the uncertainties into a new state variable, we transform the error dynamic (8) in the following canonical form

$$\begin{aligned}\dot{p}_i &= p_{i+1}, i = 1, 2, \dots, \rho - 1, \\ \dot{p}_\rho &= p_{\rho+1} + \beta_E(p)u, \\ \dot{p}_{\rho+1} &= \Xi(p, p_{\rho+1}, \zeta, u), \\ \dot{\zeta} &= \phi(p, \zeta), \\ y &= p_1 = e_1.\end{aligned}\quad (9)$$

Note that $\text{sgn}(\beta_E(p)) = \text{sgn}(\beta_E(p, \zeta))$, $p_{\rho+1} = \Theta(p, \zeta, u)$, with $\Theta(p, \zeta, u) = \kappa(p, \zeta) + \varsigma(p, \zeta)u$ and $\varsigma(p, \zeta) = \beta(p, \zeta) - \beta_E(p)$. $\Xi(p, p_{\rho+1}, \zeta, u) = \sum_{i=1}^{\rho-1} [p_{i+1} \partial_i \Theta(p, \zeta, u)] + [p_{\rho+1} + \beta_E(p)] \partial_\rho \Theta(p, \zeta, u) + \varsigma(p, \zeta) \dot{u} + \partial_\zeta \Theta(p, \zeta, u) \phi(p, \zeta)$. The mismatches between the master and slave have been included into the nonlinear function $\Theta(p, \zeta, u)$, representing the augmented state variable $p_{\rho+1}$. Hence, the synchronization problem between systems (6) and (7) is reduced to the stabilization of the error dynamics (8). An appropriate feedback controller u , studied in many literatures must be designed to achieve this goal (Fermat et al. 1999; Fermat and Solis-Perez 2008; Yang and Shao 2002; Bowong 2004). Similar linearizing feedback controller as the one adopted in Fermat et al. (1999) and Fermat and Solis-Perez (2008), is considered in this work:

$$u = -\frac{1}{\beta_E(p)} [p_{\rho+1} + \Upsilon^T p].\quad (10)$$

where Υ 's are the feedback gains representing the coefficient of the Hurwitz polynomial: $P_{(\rho)}(s) = s^\rho + \Upsilon_\rho s^{\rho-1} + \dots + \Upsilon_2 s + \Upsilon_1 = 0$. Under such feedback control, the stabilization of system error (8) is ensured (Fermat et al. 2001). The time derivatives of the observable state error p_i are needed to construct the feedback controller u as shown in (10). In fact, these error states are unknown in practice; reason why an estimate is needed. To achieve this goal, some strategies have been developed such as the linearized feedback based controller (Fermat et al. 1999; Fermat and

Solis-Peralez 2008), the nonlinear geometric-based estimator (Lie et al. 2005) and so on. Motivated by the idea of Levent (Levent 2003; Levent and Pavlov 2008), a high-order sliding mode-based estimation strategy is used in this work, where the robust design is due to the sliding mode.

We consider the following high-order sliding mode-based time differentiator estimating system:

$$\begin{aligned}\dot{\hat{p}}_1 &= \hat{p}_2 - L A_1 | \hat{p}_1 - p_1 |^{\frac{\rho}{\rho+1}} \text{sgn}(\hat{p}_1 - p_1), \\ \dot{\hat{p}}_i &= \hat{p}_{i+1} - L^i A_i | \hat{p}_1 - p_1 |^{\frac{\rho-i+1}{\rho-i+2}} \text{sgn}(\hat{p}_1 - p_1), 1 < i < \rho, \\ \dot{\hat{p}}_\rho &= \hat{p}_{\rho+1} - L^\rho A_\rho | \hat{p}_1 - p_1 |^{\frac{1}{2}} \text{sgn}(\hat{p}_1 - p_1) + \beta_E(\hat{p})u, \\ \dot{\hat{p}}_{\rho+1} &= -L^{\rho+1} A_{\rho+1} \text{sgn}(\hat{p}_1 - p_1).\end{aligned}\quad (11)$$

Where $\hat{p}_i$ are estimated values of p_i , $L > 0$ is the high-gain parameter, $A_i, i = 1, \dots, \rho + 1$ and $\beta_E(\hat{p})$ are good fit selected positive constants.

Proof Since the synchronization problem is reduced to the stabilization of the error dynamics (8), based on idea developed in Fermat et al. (2001), it can be transform to the canonical form (9). We consider the estimation error vector $\xi \in \mathbb{R}^{\rho+1}$ defined as follows: $\xi_i = \hat{p}_i - p_i, i = 1, \dots, \rho + 1$. They are obtained by subtracting (9) from (11). Thus, we have:

$$\begin{aligned}\dot{\xi}_1 &= \xi_2 - L A_1 | \xi_1 |^{\frac{\rho}{\rho+1}} \text{sgn}(\xi_1), \\ &\dots \\ \dot{\xi}_i &= \xi_{i+1} - L^i A_i | \xi_1 |^{\frac{\rho-i+1}{\rho-i+2}} \text{sgn}(\xi_1), \\ &\dots \\ \dot{\xi}_\rho &= \xi_{\rho+1} - L^\rho A_\rho | \xi_1 |^{\frac{1}{2}} \text{sgn}(\xi_1), \\ \dot{\xi}_{\rho+1} &= -L^{\rho+1} A_{\rho+1} \text{sgn}(\xi_1) - \Xi(p, p_{\rho+1}, \zeta, u).\end{aligned}\quad (12)$$

Let us define the following variables:

$$\begin{aligned}v_1 &= L^\rho | \xi_1 |^{\frac{\rho}{\rho+1}} \xi_1, \\ &\dots \\ v_i &= L^{\rho+1-i} | \xi_1 |^{\frac{\rho-i+1}{\rho-i+2}} \xi_i, 1 < i \leq \rho, \\ &\dots \\ v_{\rho+1} &= \xi_{\rho+1}.\end{aligned}\quad (13)$$

The v_1 's first derivative of time is denoted as: $\dot{v}_1 = L^\rho [\frac{\rho}{\rho+1}] | \xi_1 |^{\frac{\rho}{\rho+1}} (\dot{\hat{p}}_1 - \dot{p}_1)$. Since chaotic systems are always bounded, p_1 is bounded. Hence the tracking dynamics p_1 implies that $\hat{p}_1$ have to be bounded too. We conclude that $[|\xi_1|^{\frac{\rho}{\rho+1}}]_{i=1}$ is also bounded. We can consider its upper bound as χ , chosen as large enough number; then, we obtain $\dot{v}_1 \leq L^\rho \chi (\dot{\hat{p}}_1 - \dot{p}_1)$. Extension to

other sub-equations of system (13) allows us to transform it as:

$$\dot{v} = LD(\chi, A_1, \dots, A_{\rho+1})\xi + \Omega_0(\cdot), \quad (14)$$

with $v = (v_1, \dots, v_{\rho+1})^T$, $\Omega_0(\cdot) = [0, 0, \dots, -\Xi]$, $\xi = (\xi_1, \dots, \xi_{\rho+1})$ and

$$D(\chi, A_1, \dots, A_{\rho+1}) = \begin{pmatrix} -A_1\chi & \chi & 0 & \dots & 0 \\ -A_2 & 0 & 1 & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ -A_\rho & 0 & 0 & \vdots & 1 \\ -A_{\rho+1} & 0 & 0 & \vdots & 0 \end{pmatrix}.$$

Taking assumption (iv) into consideration, the time derivative $\dot{p}_\rho$ is a bounded function, hence $\Xi(p, p_{\rho+1}, \zeta, u)$ is also a bounded function. The constants $A_i, i = 1, \dots, \rho + 1$ are suitably chosen such that all the eigenvalues of the matrix D are in the left half complex plane. Thus $v_i \rightarrow 0, 1 \leq i \leq \rho + 1$. Hence, the estimating error system is globally asymptotically stable at the origin, which proves that $\hat{p}_i \rightarrow p_i, 1 \leq i \leq \rho + 1$. We can clearly obtain the estimates of the measurable states $p_i, 1 \leq i \leq \rho$ and the augmented state $p_{\rho+1}$. When we introduce $\hat{p}_i$ into the control command u , the error dynamics is stabilized based on Fermat et al. (2001). Hence, $e \rightarrow 0$, which achieves the synchronization between the systems (6) and (7), which completes the proof.

Numerical simulations and Pspice implementation of the synchronization scheme

Numerical simulations

In order to state the synchronization problem of an external or master neuron and a pacemaker group of pyloric CPG, we redefine the eHR system (1). We suppose y_{i1}, y_{i2}, y_{i3} and $y_{i4}, i = 0, \dots, 3$ to be the four state variables x, y, z and w respectively. Hence we derive the following system of differential equations:

$$\begin{aligned}\dot{y}_{i1} &= a_i y_{i2} + b_i y_{i1}^2 - c_i y_{i1}^3 - d_i y_{i3} + I_{DCi}, \\ \dot{y}_{i2} &= e_i - f_i y_{i1}^2 - y_{i2} - g_i y_{i4}, \\ \dot{y}_{i3} &= \mu_i [-y_{i3} + s_i (y_{i1} + h_i)], \\ \dot{y}_{i4} &= v_i [-k_i y_{i4} + r_i (y_{i2} + l_i)].\end{aligned}\quad (15)$$

The coupling between the master neuron and the main AB neuron is considered as unidirectional. This main AB neuron represents the slave system, coupled to the PD neurons, through electrical synapses (Figs. 2b, 3).

Consider the following controlled network:

Fig. 3 Controlled network of pyloric CPGs ensemble. N_0 is an external master neuron. $u(t)$ represents the control law

$$\begin{aligned}\dot{Y}_0 &= K(Y_0), \\ \dot{Y}_1 &= K(Y_1) + g_E \sum_{j=2}^3 G_{1j} H(Y_1, Y_j) + Au, \\ \dot{Y}_i &= K(Y_i) + g_E \sum_{j=1, j \neq i}^3 G_{ij} H(Y_i, Y_j), i = 2, 3.\end{aligned}\quad (16)$$

Here Y_0 denotes the state vectors of the master neuron, Y_1, Y_2, Y_3 , those of the AB/PDs neurons, Y_i . A represents the control input vector taken as $A = (1, 0, 0, 0)^T$ and u is the control command applied to infer the master neuron's behavior. The synchronization in the network is identical to the stabilization of the error dynamics between the master and the controlled neuron. In the pyloric pacemaker group, the synchronous behavior can be achieved since the non controlled nodes are coupled to the controlled one through an electrical synapse. From Eqs. (16) the following synchronization error system can be defined: $E = Y_0 - Y_1$. More precisely:

$$\begin{aligned}E_1 &= y_{01} - y_{11}, \\ E_2 &= y_{02} - y_{12}, \\ E_3 &= y_{03} - y_{13}, \\ E_4 &= y_{04} - y_{14}.\end{aligned}\quad (17)$$

Hence the error dynamics is given by:

$$\begin{aligned}\dot{E}_1 &= a_1 E_2 + b_1 E_1^2 - c_1 E_1^3 - d_1 E_3 + \Delta I_{DCi} \\ &\quad + \Gamma_1(Y_0, E, Y_1) - u, \quad i = 0, 1, \\ \dot{E}_2 &= \Delta e - f_1 E_1^2 - E_2 - g_1 E_4 + \Gamma_2(Y_0, E), \\ \dot{E}_3 &= \mu_1 [-E_3 + s_1 (E_1 + h_1)] + \Gamma_3(Y_0, E), \\ \dot{E}_4 &= v_1 [-k_1 E_4 + r_1 (E_2 + l_1)] + \Gamma_4(Y_0, E), \\ \dot{Y}_i &= K(Y_i) + g_E \sum_{j=1, j \neq i}^3 G_{ij} H(Y_i, Y_j, Y_4), i = 2, 3, \\ y &= h(E_1) = E_1 = y_{01} - y_{11}.\end{aligned}\quad (18)$$

The functions $\Gamma_i (i = 1, \dots, 4)$ are given in terms of parameters of the master system, the slave system and the state variables of the controlled network. $y = h(E_1)$ represents a smooth function (error's output) made of the

difference between the master neuron and the controlled slave neuron. The stabilization of the error system (18) is noted as the synchronization of system (16). More precisely, the problem is restricted to the construction of an appropriate feedback control law $u(t)$ such that: $\lim_{t \rightarrow \infty} E \rightarrow 0$.

The existence of a diffeomorphic transformation, which allows an original system to be transformed into a canonical form, is related to the concept of relative degree of the corresponding system. Considering the preliminaries developed in section “Description of the neuronal model and the pacemaker network”, the relative degree of the error system (18) is obtained as $\rho = 1$. For instance, $L_C L_D^0 h(E_1) = L_C h(E_1) = -1$, where $C = [-1, 0, 0, 0]^T$ represents the control input vector and $\mathcal{D}$, the vector field of the error system. Besides,

$$\begin{aligned}L_D h(E_1) &= a_1 E_2 + b_1 E_1^2 - c_1 E_1^3 - d_1 E_3 + \Delta I_{DCi} \\ &\quad + \Gamma_1(Y_0, E, Y_1).\end{aligned}\quad (19)$$

Hence, a diffeomorphic transformation $p = \Phi(E) = [E_1, E_2, E_3, E_4]^T$, defined as an invertible transformation since the jacobian matrix is invertible at any point E^0 of the space is suggested. Thus we have the canonical form:

$$\begin{aligned}\dot{p}_1 &= L_D h(\Phi^{-1}(p)) + L_C h(\Phi^{-1}(p))u, \\ \dot{p}_2 &= \Delta e - f_1 p_1^2 - p_2 - g_1 p_4 + \Gamma_2(Y_0, p), \\ \dot{p}_3 &= \mu_1 [-p_3 + s_1 (p_1 + h_1)] + \Gamma_3(Y_0, p), \\ \dot{p}_4 &= v_1 [-k_1 p_4 + r_1 (p_2 + l_1)] + \Gamma_4(Y_0, p), \\ y &= p_1.\end{aligned}\quad (20)$$

Recall that the unique measurable state is the potential membrane, and the control law or the connections with other neurons of the system are applied through this state. Taking into account the preliminaries section on robust control, system (19) can also be transformed as:

$$\begin{aligned}\dot{p}_1 &= p_2 + \beta_E(p)u, \\ \dot{p}_2 &= \Xi(p_1, p_2, \zeta, u), \\ \dot{\zeta} &= \phi(p_1, \zeta), \\ y &= p_1.\end{aligned}\quad (21)$$

On the other hand, combination of robust control and high-order sliding mode gives the following linearizing feedback control:

$$\begin{aligned}\dot{\hat{p}}_1 &= \hat{p}_2 - L\Lambda_1 |\hat{p}_1 - p_1|^{\frac{1}{2}} \text{sgn}(\hat{p}_1 - p_1) + \beta_E(\hat{p})u, \\ \dot{\hat{p}}_2 &= -L^2 \Lambda_2 \text{sgn}(\hat{p}_1 - p_1), \\ u &= -\frac{1}{\beta_E(p)} [\hat{p}_2 + \Upsilon_1 \hat{p}_1],\end{aligned}\quad (22)$$

where the estimated values of (p_1, p_2) are represented by $(\hat{p}_1, \hat{p}_2)$. The error dynamics (20) asymptotically converge

to the origin, since $\dot{\zeta} = \phi(0, \zeta)$ is a minimum phase (Fermat et al. 2001). Natural variability in neuronal properties such as voltage-gated conductances and synaptic strengths are present in biological neurons and neuronal networks. Thus, numerical simulations are done using the sets of neuron's parameters presented in section “Description of the neuronal model and the pacemaker network”, in which we choose different values of the injected current for the four neurons. The estimates' initial values are taken different to zero: $\hat{p}_1 = 0.5$ and $\hat{p}_2 = -0.9$. The control gains and the high gain parameter are chosen respectively as: $L = 1$, $A_1 = 1$, $A_2 = 1$, $\beta_E = 20$ and $\mathcal{T}_1 = 100$. The control command is activated at $t = 1100$ s. Considering that only the states y_{01} in the master neuron and y_{11} in the controlled neuron are measured, we augment the controller (21) into the networks (16). Thus we obtain the following system as the controlled network:

1. Master neuron,

$$\begin{aligned}\dot{y}_{01} &= a_0 y_{02} + b_0 y_{01}^2 - c_0 y_{01}^3 - d_0 y_{03} + I_{DC0}, \\ \dot{y}_{02} &= e_0 - f_0 y_{01}^2 - y_{02} - g_0 y_{04}, \\ \dot{y}_{03} &= \mu_0 [-y_{03} + s_0 (y_{01} + h_0)], \\ \dot{y}_{04} &= v_0 [-k_0 y_{04} + r_0 (y_{02} + l_0)].\end{aligned}\quad (23)$$

2. Controlled AB neuron,

$$\begin{aligned}\dot{y}_{11} &= a_1 y_{12} + b_1 y_{11}^2 - c_1 y_{11}^3 - d_1 y_{13} + I_{DC1} \\ &\quad + \beta_E(\hat{p})u + g_E((y_{21} - y_{11}) - (y_{31} - y_{11})), \\ \dot{y}_{12} &= e_1 - f_1 y_{11}^2 - y_{12} - g_1 y_{14}, \\ \dot{y}_{13} &= \mu_1 [-y_{13} + s_1 (y_{11} + h_1)], \\ \dot{y}_{14} &= v_1 [-k_1 y_{14} + r_1 (y_{12} + l_1)].\end{aligned}\quad (24)$$

3. PD neuron 1,

$$\begin{aligned}\dot{y}_{21} &= a_2 y_{22} + b_2 y_{21}^2 - c_2 y_{21}^3 - d_2 y_{23} + I_{DC2} \\ &\quad + g_E((y_{11} - y_{21}) - (y_{31} - y_{21})), \\ \dot{y}_{22} &= e_2 - f_2 y_{21}^2 - y_{22} - g_2 y_{24}, \\ \dot{y}_{23} &= \mu_2 [-y_{23} + s_2 (y_{21} + h_2)], \\ \dot{y}_{24} &= v_2 [-k_2 y_{24} + r_2 (y_{22} + l_2)].\end{aligned}\quad (25)$$

4. PD neuron 2,

$$\begin{aligned}\dot{y}_{31} &= a_3 y_{32} + b_3 y_{31}^2 - c_3 y_{31}^3 - d_3 y_{33} + I_{DC3} \\ &\quad + g_E((y_{11} - y_{31}) - (y_{21} - y_{31})), \\ \dot{y}_{32} &= e_3 - f_3 y_{31}^2 - y_{32} - g_3 y_{34}, \\ \dot{y}_{33} &= \mu_3 [-y_{33} + s_3 (y_{31} + h_3)], \\ \dot{y}_{34} &= v_3 [-k_3 y_{34} + r_3 (y_{32} + l_3)].\end{aligned}\quad (26)$$

Figure 4 shows the behavior of the master neuron and the pacemaker nodes before and after the control action. When we apply our control command, a transition to robust synchronization dynamics is observed. It is also worth

Fig. 4 **a** The action potential of the master neuron, the AB neuron, the PD1 neuron and the PD2 neuron; $I_{DC0} = 3.024$, $I_{DC1} = 2.640$, $I_{DC2} = 3.432$ and $I_{DC3} = 2.900$. **b** Time evolution of the errors $(y_{01} - y_{11})$, $(y_{01} - y_{21})$, $(y_{01} - y_{31})$ and the control law for the robust synchronization scheme

mentioning that, the control commands u are not equal to zero, although the synchronization between the master and the whole network is established. This can be explained by the fact that the controllers have to pay the synchronization's cost with dynamics compensation. Moreover, large oscillation and a high control energy u is observed because of the swapping of the control surface. The obtained results prove that the control command can improve robust synchronization dynamics between nodes of the pyloric pacemaker group. Notice that the control gain ($\mathcal{T}_1$) and the high gain estimation (L) are decisive for the time of convergence of the trajectories.

Electronic implementation of the synchronization scheme and Pspice simulations

In this part, we present the electrical circuit of the system. An analog computer of the chaotic neuronal oscillator, defined by the set of Eq. (1) is built. (Chedjou et al. 2001; Itoh 2001; Kengne et al. 2014) Since the oscillator frequency can be modified with the help of the analog computer, it facilitates the controller design. Moreover, this analog computer is useful to verify the theoretical results obtained in the previous sections. Figure 5a, b, and figure 4.2c–4.2d represent respectively, the master neuron (Y_0), the AB neuron, the PD_1 neuron and PD_2 neuron, while in Fig. 6 we present the controller's circuit.

Associated circuitry and operational amplifier (TL084) implement the operations of integration, addition and subtraction. We use the Pspice ideal multiplier “Mult” to

ensure the multiplication operation. Taking into account an appropriate time scaling, the analog computers’ outputs can be displayed on an oscilloscope. This action will be done

Fig. 5 Circuit diagram of **a** the master neuron, **b** the AB neuron, **c** the PD1 neuron and **d** the PD2 neuron

Fig. 5 continued

by connecting the latter's inputs with the output voltage of different integrators. According to J. C. Sprott, the part of the controller in brown box (Fig. 6), simulates the absolute value function; the sign function is simulated by the part in the orange box, of the same figure (Sprott 2000; Louodop et al. 2014). With the help of the Kirchhoff law, and taking into account that the operational amplifiers are ideal devices, the states of the whole network satisfy the set of nonlinear differential equations:

1. Master neuron

$$\begin{aligned}
 \dot{x}_m &= \frac{y_m}{R_3 C_x} + \frac{x_m^2}{R_1 C_x} - \frac{x_m^3}{R_2 C_x} - \frac{z_m}{R_5 C_x} + \frac{V_1}{R_6 C_x}, \\
 \dot{y}_m &= -\frac{x_m^2}{R_{11} C_y} - \frac{y_m}{R_{12} C_y} - \frac{w_m}{R_{13} C_y} + \frac{V_2}{R_{14} C_y}, \\
 \dot{z}_m &= \frac{x_m}{R_{17} C_z} - \frac{z_m}{R_{18} C_z} + \frac{V_3}{R_{19} C_z}, \\
 \dot{w}_m &= \frac{y_m}{R_{20} C_w} - \frac{w_m}{R_{21} C_w} + \frac{V_4}{R_{22} C_w}.
 \end{aligned} \quad (27)$$

Fig. 6 (Color online) Circuit diagram of the controller

2. Controlled AB neuron

$$\begin{aligned}\dot{x}_2 &= \frac{y_2}{R_{51}C_{x2}} + \frac{x_2^2}{R_{49}C_{x2}} - \frac{x_2^3}{R_{50}C_{x2}} - \frac{z_2}{R_{52}C_{x2}} \\ &+ \frac{V_5}{R_{53}C_{x2}} + \frac{(x_3 - x_2)}{R_{54}C_{x2}} + \frac{(x_4 - x_2)}{R_{55}C_{x2}} + \frac{u}{R_{56}C_{x2}}, \\ \dot{y}_2 &= -\frac{x_2^2}{R_{61}C_{y2}} - \frac{y_2}{R_{62}C_{y2}} - \frac{w_2}{R_{63}C_{y2}} + \frac{V_6}{R_{64}C_{y2}}, \\ \dot{z}_2 &= \frac{x_2}{R_{70}C_{z2}} - \frac{z_2}{R_{71}C_{z2}} + \frac{V_7}{R_{72}C_{z2}}, \\ \dot{w}_2 &= \frac{y_2}{R_{67}C_{w2}} - \frac{w_2}{R_{68}C_{w2}} + \frac{V_8}{R_{69}C_{w2}}.\end{aligned}\quad (28)$$

3. PD neuron 1

$$\begin{aligned}\dot{x}_3 &= \frac{y_3}{R_{83}C_{x3}} + \frac{x_3^2}{R_{81}C_{x3}} - \frac{x_3^3}{R_{82}C_{x3}} - \frac{z_3}{R_{84}C_{x3}} \\ &+ \frac{V_9}{R_{85}C_{x3}} + \frac{(x_2 - x_3)}{R_{86}C_{x3}} + \frac{(x_4 - x_3)}{R_{86}C_{x3}}, \\ \dot{y}_3 &= -\frac{x_3^2}{R_{91}C_{y3}} - \frac{y_3}{R_{92}C_{y3}} - \frac{w_3}{R_{93}C_{y3}} + \frac{V_{10}}{R_{94}C_{y3}}, \\ \dot{z}_3 &= \frac{x_3}{R_{97}C_{z3}} - \frac{z_3}{R_{98}C_{z3}} + \frac{V_{11}}{R_{99}C_{z3}}, \\ \dot{w}_3 &= \frac{y_3}{R_{100}C_{w3}} - \frac{w_3}{R_{101}C_{w3}} + \frac{V_{12}}{R_{102}C_{w3}}.\end{aligned}\quad (29)$$

4. PD neuron 2

$$\begin{aligned}\dot{x}_4 &= \frac{y_4}{R_{119}C_{x4}} + \frac{x_4^2}{R_{117}C_{x4}} - \frac{x_4^3}{R_{118}C_{x4}} - \frac{z_4}{R_{120}C_{x4}} \\ &+ \frac{V_{13}}{R_{121}C_{x4}} + \frac{(x_2 - x_4)}{R_{122}C_{x4}} + \frac{(x_3 - x_4)}{R_{122}C_{x4}}, \\ \dot{y}_4 &= -\frac{x_4^2}{R_{127}C_{y4}} - \frac{y_4}{R_{128}C_{y4}} - \frac{w_4}{R_{129}C_{y4}} + \frac{V_{14}}{R_{130}C_{y4}}, \\ \dot{z}_4 &= \frac{x_4}{R_{133}C_{z4}} - \frac{z_4}{R_{134}C_{z4}} + \frac{V_{15}}{R_{135}C_{z4}}, \\ \dot{w}_4 &= \frac{y_4}{R_{136}C_{w4}} - \frac{w_4}{R_{137}C_{w4}} + \frac{V_{16}}{R_{138}C_{w4}}.\end{aligned}\quad (30)$$

5. Feedback control

$$\begin{aligned}\dot{\hat{p}}_1 &= -\frac{R_{29}}{R_{33}C_{con1}R_{32}}|\hat{p}_1 - p_1|^{\frac{1}{2}}\text{sgn}(\hat{p}_1 - p_1) \\ &+ \frac{R_{29}}{R_{33}C_{con1}R_{30}}\hat{p}_2 + \frac{R_{29}}{R_{33}C_{con1}R_{31}}u, \\ \dot{\hat{p}}_2 &= -\frac{1}{R_{34}C_{con2}}\text{sgn}(\hat{p}_1 - p_1), \\ u &= -R_{38}\left[\frac{\hat{p}_2}{R_{37}} + \frac{\hat{p}_1}{R_{39}}\right].\end{aligned}\quad (31)$$

We express the parameters of the above set of Eqs. (27, 28, 29, 30, 31), in terms of the capacitors and the resistors'

Fig. 7 Pspice simulations. **a** The action potential of the master neuron, the AB neuron, the PD1 neuron and the PD2 neuron; **b** Time evolution of the errors $(x_m - x_2)$, $(x_m - x_3)$, $(x_m - x_4)$ and the control law for the robust synchronization scheme

values as described in the appendix. Hence, with the help of the parameters' values presented in section “[Description of the neuronal model and the pacemaker network](#)“, and the controller gain of section “[Numerical simulations](#)“, the corresponding capacitors and resistors' values are also found in Table 2 of the same appendix.

Moreover, the time scaling allows us to simulate the system's behavior at any given frequency, depending on the used capacitors and resistors. Figure 7 presents the results obtained through Pspice simulations. The control is activated at $t = 110$ ms. Hence, when supervising the circuit's components (resistors and capacitors), we observe a synchronous state of the whole system, when the controller is activated. We noted the similarity between these results and those obtained in Matlab (see Fig. 4). This allows us to conclude that the analog computer is a powerful tool for the design of complex nonlinear oscillators, such as eHR neuronal model.

Noise effects on the synchronization

Noisy behavior of coupled neurons is shown by many in vivo experiments. For example, the activity of neurons from the visual cortex can be recorded when a slowly moving bar is presented on a screen in the visual field of the animal (Hubel and Wiesel 1959, 1977). The firing rate goes up until the bar leaves the receptive field at the opposite border, as soon as it entered the neuron's receptive field. However, the spike train varies considerably from test to test, if the same experiments is repeated several

times. Furthermore, even if the screen is blank and no external stimulus is applied, the same neuron is spontaneously active. The intervals between one spike and the next, exhibit a large changeableness, resulting in a wide distribution of interspike intervals, during this spontaneous activity (Softky and Koch 1993).

Intrinsic noise sources that generate behavior on the level of the neuronal dynamics, and extrinsic sources that arise from network effects and synaptic transmission are distinguished in the literature (Manwani and Koch 1999). Thermal noise is the source, which is literally omnipresent. Because of the discrete nature of electric charge carriers, a fluctuation at finite temperature (Johnson noise) is observed on the voltage u across any electrical resistor R . Wulfram Gerstner and Werner M. Kistler, expressed the variance of these fluctuations at rest, proportional to the rate $RkTB$. Where k is the Boltzmann constant, T is the temperature and B is the band width of the system (Wulfram and Werner 2002). Furthermore, one knows that the neuronal dynamics is described by an equivalent electrical circuit containing resistors. Thus, the neuronal membrane potential fluctuates as well. Nevertheless, Manwani and Koch (1999) showed that these fluctuations due to Johnson noise are however of minor importance, compared to other noises. Another source of intrinsic noise, specific to neurons results from the finite number of ion trunks in a neuronal membrane's patch (Schneidman et al. 1998; White et al. 2000).

Out of intrinsic noise sources at the level of an individual neuron, there also exist sources of noise that are due to signal transmission and network effects, called extrinsic noises. The transmission failure of synapses, appear to impose a substantial limitation to signal transmission within a neuronal network. Hessler et al. (1993) and Markram and Tsodyks (1996) suggested through experiments with double electrode recordings from two synaptically connected neurons that, only 10–30 % of the presynaptic spikes generate a postsynaptic response. However, an important part of neuronal spiking's irregularity, during spontaneous activity appears to be due to the properties of the network as a whole, rather to the individual neurons. It has been shown in model studies that, highly irregular spike trains can be produced by networks of excitatory and inhibitory neurons with fixed random connectivities (Nutzelt et al. 1994; Fusi et al. 2000; Kistler and Zeeuw 2002).

In this part, we study the noise effects on robust synchronization of pyloric pacemaker neurons. We split the perturbations (noises) into two parts, those coming from coupling with other oscillators and those arising from extrinsic noise. Hence, the noisy network is modeled as:

Fig. 8 Numerical simulations when noise is taken into account in the system; the control gain is $L = 1.0$. First line $R_{ij}(Y(t), t) = 0.5$, second line $R_{ij}(Y(t), t) = 1.0$ and third line $R_{ij}(Y(t), t) = 10.0$. **a, c, e** The action potential of the master neuron, the AB neuron, the PD1 neuron and the PD2 neuron; **b, d, f** time evolution of the errors $(y_{01} - y_{11})$, $(y_{01} - y_{21})$, $(y_{01} - y_{31})$ and the control law for the robust synchronization scheme

$$\dot{Y}_0 = K(Y_0),$$

$$\begin{aligned} \dot{Y}_1 = & K(Y_1) + g_E \sum_{j=2}^3 [G_{1j}H(Y_1, Y_j) \\ & + R_{1j}(Y_1, Y_j, t)\zeta_j^{OU}(t)] + Au, \end{aligned} \quad (32)$$

$$\begin{aligned} \dot{Y}_i = & K(Y_i) + g_E \sum_{j=1, j \neq i}^3 [G_{ij}H(Y_i, Y_j) \\ & + R_{ij}(Y_i, Y_j, t)\zeta_j^{OU}(t)], i = 2, 3. \end{aligned}$$

Where we consider a stochastic differential equations with colored noises. $\zeta_j^{OU}(t)$ are Ornstein–Uhlenbeck processes which are uncorrelated among them, and with different autocorrelation times

$$\langle \zeta_j^{OU}(t) \rangle = 0, \quad (33)$$

$$\langle \zeta_k^{OU}(t)\zeta_l^{OU}(s) \rangle = \delta_{kl} \frac{1}{2\tau_k} e^{-|t-s|/\tau_k}. \quad (34)$$

$Y_j(t)$ are the states variables for oscillators ($j = 1, \dots, 3$), and g_E represents the coupling strength. R_{ij} represents the noisy perturbations. Each oscillator obeys exactly the same dynamics (16) when there is no noise ($R_{ij}(Y(t), t) = 0$). Since this can be done more generally, it seems simpler to split the perturbations into parts dealing with coupling from the noisy one. This makes sense as well, in the point of view of neuronal applications. Simulations are done, using the stochastic Runge–Kutta method also called the Heun algorithm. The set of parameters of section “Numerical simulations” are considered; furthermore, $\tau_k = 0.5, k = 1, \dots, 3$ and perturbations independent of system’s states are considered. The numerical integration of the noisy system for different values of the control parameter of the intensity of the noise, give the following results which are sketched on the Figs. 8, 9 and 10.

Recall that, the pacemaker neuronal ensemble is isolated from the rest of the pyloric CPGs, by pharmacological tools. Nevertheless, neuromodulator inputs coming from anterior ganglia are left intact, while the pacemaker group can still receive inputs from the gastric network (Cornejo-Perez et al. 2012). Intense synaptic inputs coming from the cardiac sac network appear abruptly, when the aforementioned inputs are unchanged. We suppose, these gastric/oesophageal inputs as noise in the network, together with non-uniformity coming from coupling. We observed that

Fig. 9 Numerical simulations when noise is taken into account in the system; the control gain is $L = 10.0$. First line $R_{ij}(Y(t), t) = 0.5$, second line $R_{ij}(Y(t), t) = 1.0$ and third line $R_{ij}(Y(t), t) = 10.0$. **a, c, e** The action potential of the master neuron, the AB neuron, the PD1 neuron and the PD2 neuron; **b, d, f** time evolution of the errors $(y_{01} - y_{11})$, $(y_{01} - y_{21})$, $(y_{01} - y_{31})$ and the control law for the robust synchronization scheme

when the noise amplitude increases, the burst disappears in the pacemaker nodes, giving rise to a random vibration with high amplitude (see Figs. 8e, 9e, 10e). In this case, the pacemaker neurons still behave as the master neuron, showing the states coherence when the control action is activated. As illustrated in Figs. 8b, d, f, 9b, d, f, 10b, d, f, the energy needed to ensure such behavior becomes high. It can be justified by the fact that, the sub-network responses are more noisy as the noise amplitude increases. In such situation, only the AB neuron turns to synchronize with the master neuron while the PD1 and PD2 remain very noisy. This may be due to the fact that the control law is applied directly to the AB node. When the noise amplitude is small compared to the network's voltage membranes (see Figs. 8a–d, 9a–d, 10a–d), the synchronization scheme ensures the convergence of the coupled nodes to the master's behavior. In the latter case, the energy needed by the controller is small compared to the one necessary in Figs. 8e–f, 9e–f and 10e–f. Nevertheless, the control gain (L) also plays a capital role. When it increases, the error states decrease near zero as observed in Figs. 8 and 9. But, when it reaches a critical value, it turns to increase the energy necessary for the system to synchronize (see Fig. 8), since in this case the controller amplitude becomes high.

We found that, although noise effects are taken into account in the network, synchronization can be ensured using the combination of high-order sliding mode based information reconstruction technique, and the linear feedback control. Consequently, the trajectory of the master neuron can be tracked with superior performance, when the noise amplitude is small compared to different nodes' voltage membrane. When this amplitude becomes higher, although the control law allows the pacemaker to track the master behavior, its evolution becomes noisy.

Generally, noise is linked to signals transmission or reception and constitute an obstacle to such signals which become unsteady or uncontrolled perfectly. Furthermore, considering the fact that it is impossible to separate a system perfectly from its environment, noise is unavoidable in such system. Sometimes, addition of noise is useful and can make a dynamical system behaves more regularly (Wellens et al. 2004). In nonlinear systems the cooperative effect between a weak signal and noise, leads to an

Fig. 10 Numerical simulations when noise is taken into account in the system; the control gain is $L = 25.0$. First line $R_{ij}(Y(t), t) = 0.5$, second line $R_{ij}(Y(t), t) = 1.0$ and third line $R_{ij}(Y(t), t) = 10.0$. **a, c, e** The action potential of the master neuron, the AB neuron, the PD1 neuron and the PD2 neuron; **b, d, f** Time evolution of the errors $(y_{01} - y_{11})$, $(y_{01} - y_{21})$, $(y_{01} - y_{31})$ and the control law for the robust synchronization scheme

Fig. 11 SNR for the pyloric pacemaker neurons as a function of the noise amplitude. **a** The control gain is $L = 0.1$; **b** the control gain is $L = 1.0$; **c** the control gain is $L = 10.0$

enhanced response to external force and is named stochastic resonance (SR) (Wellens et al. 2004). This phenomenon was first analyzed in 1981 and was proposed as likely mechanism for many periodic occurrences of ice ages of earth (Kramers 1940). Its first experimental demonstration was due to Schmitt triggers in 1983 (Fauve and Heslot 1983). Stochastic resonance has then grown to a rapidly developing, interdisciplinary field of research, including laser, electronics, biology and other systems.

The more quantitative analysis of SR in systems is through the power spectrum of output variables of systems. The power spectrum of such output exhibits a strong peak at some signal frequency (Wellens et al. 2004). A measure of the SR is defined by the area under this peak, also called output signal power. In other case, we make use of the signal to noise ratio (SNR) to quantify the SR. SNR is defined as the height of the signal peak divided by the amplitude of the noise background at frequency ω_s .

$$SNR = \frac{P(\omega_s)}{P_N(\omega_s)}, \quad (35)$$

where, $P(\omega_s)$ is the power spectrum of the signal and $P_N(\omega_s)$ the power spectrum of the noise. Recall that, SR occurs in the system if the SNR exhibits a maximum as a function of the noise strength. In Fig. 11 we show an example obtained via the numerical simulations of system (31) for different values of the control gain (Fig. 11a: $L = 0.1$, Fig. 11b: $L = 1.0$, and Fig. 11c: $L = 10.0$). In these figures and for low values of the control gain, SNR exhibits a less pronounced local maximum at a slightly higher noise level (Fig. 11a, b). When the control gain becomes high, this local maximum disappear (Fig. 11c). This allows us to conclude that the proposed controller turns to suppress SR in the system when its gain becomes high. On the other hand, the robust synchronization reduces the stochastic resonance of the network when the controller gain increases.

Conclusion

The purpose of this paper was to investigate the noise effects on robust synchronization of a small pacemaker neuronal ensemble via a nonlinear controller, and the electronic design of the synchronization scheme. We considered the pacemaker group of the pyloric CPG in the STG of lobster, widely studied in biological preparations. Based on the combination of the high-order sliding mode and the linear feedback control, a control law is constructed by a linear function of the estimated time differentiator. We drove the dynamics of the slave neurons (constitute of coupled AB, PD1 and PD2 nodes) to follow that of the master neuron. The coupling between the master neuron and the AB node of the pacemaker group was considered as unidirectional. Electronic circuits of the control strategy and those of the whole system are proposed, together with the corresponding Pspice simulations. We modeled the pyloric pacemaker group as a network of coupled three cells, with perturbations described by the Ornstein–Uhlenbeck processes. We have considered noises coming from coupling to other oscillators, and those arising from extrinsic source. As it has been shown by neuroscientists, noise is an ineluctable consequence of brains functioning with molecular components at the scale of nanometer. Its presence in nervous systems, particularly in coupled neural networks has great effects on their synchronous behavior. In the network investigated in this paper, such sources of noise are offsetted by the controller through the dynamics compensator. The synchronization is illustrated with the help of the action potentials. The simulations show that the robust synchronization can be achieved in the whole

network and we think that it may results in interesting applications in biomimetic and robotic systems. Moreover, We found that, the proposed controller reduces the stochastic resonance of the network when its gain increases. Since the synchronization error depends on the control gain and the synchronization of the network on the coupling strength, a compromise exists between both the coupling strength and the control gain. Furthermore, time-delay is unavoidable in dynamical systems like neurons. It plays a significant role in the brain and in large-scale synchronized oscillations. However, further detailed analysis is necessary in order to study the robustness of the synchronization process when delay is taken into account. Treatment of these questions remains an open problem and is currently under investigation.

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Appendix 1: Some mathematical expressions

Here we investigate some mathematical expressions obtained during the application of the Kirchhoff law. First, we give the system parameters as a function of the components of circuits presented in Figs. 5b and 6. These equivalences are given in Table 1

Table 1 Coupled system parameters and their electronic equivalents

Parameters	Equivalences	Parameters	Equivalences
a_0	$\frac{1}{R_3 C_x}$	b_0	$\frac{1}{R_1 C_x}$
c_0	$\frac{1}{R_2 C_x}$	d_0	$\frac{1}{R_5 C_x}$
e_0	$\frac{V_2}{R_{14} C_y}$	f_0	$\frac{1}{R_{11} C_y}$
g_0	$\frac{1}{R_{13} C_y}$	$\mu_0 s_0 h_0$	$\frac{V_3}{R_{19} C_z}$
I_{DC0}	$\frac{V_1}{R_6 C_x}$	$v_0 k_0$	$\frac{1}{R_{21} C_w}$
$v_0 r_0 I_0$	$\frac{V_4}{R_{22} C_w}$	$v_0 r_0$	$\frac{1}{R_{20} C_w}$
$\mu_0 s_0$	$\frac{1}{R_{17} C_z}$	μ_0	$\frac{1}{R_{18} C_z}$
a_1	$\frac{1}{R_{51} C_{x2}}$	b_1	$\frac{1}{R_{49} C_{x2}}$
c_1	$\frac{1}{R_{50} C_{x2}}$	d_1	$\frac{1}{R_{52} C_{x2}}$
e_1	$\frac{V_6}{R_{64} C_{y2}}$	f_1	$\frac{1}{R_{61} C_{y2}}$
g_1	$\frac{1}{R_{63} C_{y2}}$	$\mu_1 s_1 h_1$	$\frac{V_7}{R_{72} C_{z2}}$
I_{DC1}	$\frac{V_5}{R_{53} C_{x2}}$	$v_1 k_1$	$\frac{1}{R_{68} C_{w2}}$
$v_1 r_1 I_1$	$\frac{V_8}{R_{69} C_{w2}}$	$\mu_1 s_1$	$\frac{1}{R_{70} C_{z2}}$
μ_1	$\frac{1}{R_{71} C_{z2}}$	g_E	$\frac{1}{R_{54} C_{x2}}$
			$\frac{1}{R_{55} C_{x2}}$
			$\frac{1}{R_{86} C_{x3}}$
			$\frac{1}{R_{123} C_{x4}}$

Table 1 continued

Parameters	Equivalences	Parameters	Equivalences
$\beta_E(\bar{p})$	$\frac{1}{R_{56}C_{x2}},$ $\frac{R_{29}}{R_{33}C_{com1}R_{31}}$	v_1r_1	$\frac{1}{R_{67}C_{w2}}$
a_2	$\frac{1}{R_{83}C_{x3}}$	b_2	$\frac{1}{R_{81}C_{x3}}$
c_2	$\frac{1}{R_{82}C_{x3}}$	d_2	$\frac{1}{R_{84}C_{x3}}$
e_2	$\frac{V_{10}}{R_{94}C_{y3}}$	f_2	$\frac{1}{R_{91}C_{y3}}$
g_2	$\frac{1}{R_{93}C_{y3}}$	$\mu_2s_2h_2$	$\frac{V_{11}}{R_{99}C_{z3}}$
I_{DC2}	$\frac{V_9}{R_{85}C_{x3}}$	v_2k_2	$\frac{1}{R_{101}C_{w3}}$
$v_2r_2l_2$	$\frac{V_{12}}{R_{102}C_{w3}}$	μ_2s_2	$\frac{1}{R_{97}C_{z3}}$
μ_2	$\frac{1}{R_{98}C_{z3}}$	v_2r_2	$\frac{1}{R_{100}C_{w3}}$
a_3	$\frac{1}{R_{119}C_{x4}}$	b_3	$\frac{1}{R_{117}C_{x4}}$
c_3	$\frac{1}{R_{118}C_{x4}}$	d_3	$\frac{1}{R_{120}C_{x4}}$
e_3	$\frac{V_{14}}{R_{130}C_{y4}}$	f_3	$\frac{1}{R_{127}C_{y4}}$
g_3	$\frac{1}{R_{129}C_{y4}}$	$\mu_3s_3h_3$	$\frac{V_{15}}{R_{135}C_{z4}}$
I_{DC3}	$\frac{V_{13}}{R_{121}C_{x4}}$	v_3k_3	$\frac{1}{R_{137}C_{w4}}$
$v_3r_3l_3$	$\frac{V_{16}}{R_{138}C_{w4}}$	μ_3s_3	$\frac{1}{R_{133}C_{z3}}$
μ_3	$\frac{1}{R_{134}C_{z4}}$	v_3r_3	$\frac{1}{R_{136}C_{w4}}$
LA_1	$\frac{R_{29}}{R_{33}C_{com1}R_{32}}$	L^2A_2	$\frac{1}{R_{34}C_{com2}}$
$\frac{1}{\beta_E(\bar{p})}$	R_{38}	$\mathcal{T}_1$	$\frac{1}{R_{39}}$
1	$\frac{1}{R_{12}C_y}$	1	$\frac{1}{R_{62}C_{y2}}$
1	$\frac{1}{R_{92}C_{y3}}$	1	$\frac{1}{R_{129}C_{y4}}$
1	$\frac{R_{29}}{R_{33}C_{com1}R_{30}}$	1	$\frac{1}{R_{37}}$

Appendix 2: Electronics components and their used values

Furthermore, the values of resistors and capacitors used in different circuits are given in the Table 2 below.

Table 2 Electronics components and their used values

Components	Value	Components	Value
V_1	3.024	R_1	3.333 k Ω
V_2	1.01 V	R_2	10 k Ω
V_3	0.0136 V	R_3	10 k Ω
V_4	0.00145 V	R_5	10.101 k Ω
V_5	3.025 V	R_6	10 k Ω
V_6	1.01 V	R_7	10 k Ω
V_7	0.0136 V	R_8	10 k Ω
V_8	0.00145 V	R_9	10 k Ω
V_9	3.021 V	R_{10}	10 k Ω
V_{10}	1.01 V	R_{11}	1.994 k Ω
V_{11}	0.0136 V	R_{12}	10 k Ω

Table 2 continued

Components	Value	Components	Value
V_{12}	0.00145 V	R_{13}	359.712 k Ω
V_{13}	3.02 V	R_{14}	10 k Ω
V_{14}	1.01 V	R_{15}	10 k Ω
R_{16}	10 k Ω	R_{17}	1.172 M Ω
R_{18}	4.651 M Ω	R_{19}	10 k Ω
R_{20}	3.703 M Ω	R_{21}	11.606 M Ω
R_{22}	10 k Ω	R_{23}	10 k Ω
R_{24}	10 k Ω	R_{25}	10 k Ω
R_{26}	10 k Ω	R_{27}	10 k Ω
R_{28}	10 k Ω	R_{29}	10 Ω
R_{30}	100 k Ω	R_{31}	5 k Ω
R_{32}	1 k Ω	R_{33}	10 k Ω
R_{34}	1 M Ω	R_{35}	10 k Ω
R_{36}	10 k Ω	R_{37}	1 Ω
R_{38}	0.05 Ω	R_{39}	0.04 Ω
R_{40}	10 k Ω	R_{41}	10 k Ω
R_{42}	1 k Ω	R_{43}	4.85 k Ω
R_{44}	13.508 k Ω	R_{45}	10 k Ω
R_{46}	10 k Ω	R_{47}	10 k Ω
R_{48}	10 k Ω	R_{49}	3.333 k Ω
R_{50}	10 k Ω	R_{51}	10 k Ω
R_{52}	10.101 k Ω	R_{53}	10 k Ω
R_{54}	1 k Ω	R_{55}	1 k Ω
R_{56}	100 Ω	R_{57}	10 k Ω
R_{58}	10 k Ω	R_{59}	10 k Ω
R_{60}	10 k Ω	R_{61}	1.994 k Ω
R_{62}	10 k Ω	R_{63}	359.712 k Ω
R_{64}	10 k Ω	R_{65}	10 k Ω
R_{66}	10 k Ω	R_{67}	3.703 M Ω
R_{68}	11.606 M Ω	R_{69}	10 k Ω
R_{70}	1.172 M Ω	R_{71}	4.657 M Ω
R_{72}	10 k Ω	R_{73}	10 k Ω
R_{74}	10 k Ω	R_{75}	10 k Ω
R_{76}	10 k Ω	R_{77}	10 k Ω
R_{78}	10 k Ω	R_{79}	10 k Ω
R_{80}	10 k Ω	R_{81}	3.333 k Ω
R_{82}	10 k Ω	R_{83}	10 k Ω
R_{84}	10.101 k Ω	R_{85}	10 k Ω
R_{86}	1 k Ω	R_{87}	10 k Ω
R_{88}	10 k Ω	R_{89}	10 k Ω
R_{90}	10 k Ω	R_{91}	1.994 M Ω
R_{92}	10 k Ω	R_{93}	359.712 k Ω
R_{94}	10 k Ω	R_{95}	10 k Ω
R_{96}	10 k Ω	R_{97}	1.172 M Ω
R_{98}	4.651 M Ω	R_{99}	10 k Ω
R_{100}	3.703 M Ω	R_{101}	11.606 M Ω
R_{102}	10 k Ω	R_{103}	10 k Ω
R_{104}	10 k Ω	R_{105}	10 k Ω

Table 2 continued

Components	Value	Components	Value
R_{106}	10 k Ω	R_{107}	10 k Ω
R_{108}	10 k Ω	R_{109}	10 k Ω
R_{110}	10 k Ω	R_{111}	10 k Ω
R_{112}	10 k Ω	R_{113}	10 k Ω
R_{114}	10 k Ω	R_{115}	10 k Ω
R_{116}	10 k Ω	R_{117}	3.333 k Ω
R_{118}	10 k Ω	R_{119}	10 k Ω
R_{120}	10.101 k Ω	R_{121}	10 k Ω
R_{122}	1 k Ω	R_{123}	10 k Ω
R_{124}	10 k Ω	R_{125}	10 k Ω
R_{126}	10 k Ω	R_{127}	1.994 M Ω
R_{128}	10 k Ω	R_{129}	359.712 k Ω
R_{130}	10 k Ω	R_{131}	10 k Ω
R_{132}	10 k Ω	R_{133}	1.172 M Ω
R_{134}	4.651 M Ω	R_{135}	10 k Ω
R_{136}	3.703 M Ω	R_{137}	11.606 M Ω
R_{138}	10 k Ω	R_{139}	10 k Ω
R_{140}	10 k Ω	R_{141}	10 k Ω
R_{142}	10 k Ω	R_{143}	10 k Ω
R_{144}	10 k Ω	R_{145}	10 k Ω
R_{146}	10 k Ω	R_{147}	10 k Ω
R_{148}	10 k Ω	R_{149}	10 k Ω
R_{150}	10 k Ω	R_{151}	10 k Ω
R_{152}	10 k Ω	$C_x, C_y,$	10 nF
		$C_z, C_w,$	
		$C_{xj}, C_{yj},$	
		$C_{zj}, C_{wj},$	
		$j = 2, \dots, 4$	
V_{15}	0.0136 V	V_{16}	0.00145 V

References

- Arenas A, Diaz-Guillera A, Perez-Vicente C (2006) Synchronization processes in complex networks. *Phys D* 224:27–34
- Baptista M, Carvalho J, Hussein M (2008) Finding quasi-optimal network topologies for information transmission in active networks. *PLoS ONE* 3:e3479
- Benzi R, Sutera A, Vulpiani A (1981) The mechanism of stochastic resonance. *J Phys A Math Gen* 14:L453–L457
- Bowong S (2004) Stability analysis for the synchronization of chaotic systems with different order: application to secure communication. *Phys Rev Lett* A 36:102–113
- Bowong S, Kakmeni FM (2003) Chaos control and duration time of class of uncertain chaotic systems. *Phys Lett A* 316:206–217
- Bowong S, Kakmeni FM (2004) Synchronization of uncertain chaotic system via backstepping approach. *Chaos Solitons Fractals* 21:99–111
- Cazalets J, Nagy I, Moulins M (1990) Suppressive control of the crustacean pyloric network by pair of identified interneurons. I. Modulation of the motor pattern. *J Neurosci* 10:448–457
- Chedjou J, Fotsin H, Woafu P, Domngang S (2001) Analog simulation of the dynamics of a van der pol oscillator coupled to a duffing oscillator. *IEEE Trans Circuits Syst* 48:748–756
- Chen M, Zhou D (2006) Synchronization in uncertain complex networks. *Chaos* 16:013101
- Cornejo-Perez O, Solis-Perales G, Arenas-Prado J (2012) Synchronization dynamics in a small pacemaker neuronal ensemble via a robust adaptive controller. *Chaos Solitons Fractals* 45:861–868
- Delcomyn F (1980) Natural basis of rhythmic behavior in animals. *Science* 210:492–498
- Elson R, Selverston AI, Huerta R, Rulkov N, Rabinovich M, Abarbanel H (1998) Synchronous behavior of two coupled biological neurons. *Phys Rev Lett* 87:5692
- Elson R, Selverston A, Abarbanel H, Rabinovich M (2002) Inhibitory synchronization of bursting in biological neurons: dependence on synaptic time constant. *J Neurophysiol* 88:1166–1176
- Faisal A, Selen L, Wolpert D (2008) Noise in the nervous system. *Nat Rev Neurosci* 9:292–303
- Fakle M, Huerta R, Rabinovich M, Abarbanel H, Elson R, Selverston A (2000) Modeling observed chaotic oscillations in bursting neurons: the role of calcium dynamics and ip3. *Biol Cybern* 82:517–527
- Fauve S, Heslot F (1983) Stochastic resonance in a bistable system. *Phys Lett A* 97:5–7
- Fermat R, Solis-Perales G (2008) Robust synchronization of chaotic systems via feedback. Springer, New York
- Fermat R, Alvarez-Ramirez B, Castillo T, Gonzalez J (1999) On robust chaos suppression of nonlinear oscillators: applications to Chua's circuit. *IEEE Trans Circuits Syst I* 46:1150–1152
- Fermat R, Ortiz R, Perales G (2001) A chaos-based communication scheme via robust asymptotic feedback. *IEEE Trans Circuits Syst I* 48:1161–1169
- Fotsin H, Daafouz J (2005) Adaptive synchronization of uncertain chaotic colpitts oscillators. *Phys Lett A* 339:304–315
- Fusi S, Giudice PD, Amit D (2000) Neurophysiology of a VLSI spiking neural network. In: *Proceedings of IJCNN III*, pp 121–126
- Gass L (2001) Synchronization and rhythms processes in physiology. *Nature* 410:277–284
- Gray C, König P, Engel A, Singer W (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar synchronizations which reflects global stimulus properties. *Nature* 338:334–337
- Harald A, Stefan R (2008) Interpreting neurodynamics: concepts and facts. *Cogn Neurodyn* 2:297–318
- Harris-Warrick R, Marder E, Selverston A, Moulins M (1992) *Dynamic biological networks: the stomatogastric nervous system*. MIT Press, Cambridge
- Hatsuo H, Jun I (2009) LTD windows of the STDP learning rule and synaptic connections having a large transmission delay enable robust sequence learning amid background noise. *Cogn Neurodyn* 3:119–130
- Hessler N, Shirke A, Malinow R (1993) The probability of transmitter release at a mammalian central synapse. *Nature* 366:569–572
- Hindmarsh J, Rose R (1984) A model of neuronal bursting using three coupled first order differential equations. *Proc R Soc Lond Ser B* 221:87–102
- Hubel D, Wiesel T (1959) Receptive fields of single neurons in the cats striate cortex. *J Physiol* 148:574–591
- Hubel D, Wiesel T (1977) Functional architecture of macaque monkey visual cortex. *Proc R Soc B* 198:159
- Isidori A (1989) *Nonlinear control system*. Springer, Berlin
- Itoh M (2001) Synthesis of electronic circuits for simulating nonlinear dynamics. *Int J Bifurc Chaos* 11:605–653
- Kengne J, Chedjou J, Kom M, Kyamakya K, Kamdoun T (2014) Regular oscillations, chaos, and multistability in a system of two

- coupled van der pol oscillators: numerical and experimental studies. *Nonlinear Dyn* 76:1119–1132
- Kistler W, Zeeuw CD (2002) Dynamical working memory and timed responses: the role of reverberating loops in the olivo-cerebellar system. *Neural Comput* 14:2597–2626
- Kramers H (1940) Brownian motion in a field of force and the diffusion model of chemical reactions. *Physica* 7:284–304
- Kreiter A, Singer W (1996) Stimulus-dependent synchronization of neuronal responses in the visual cortex of the awake macaque monkey. *J Neurosci* 16:2381–2396
- Kryukov V (2012) Towards a unified model of pavlovian conditioning: short review of trace conditioning models. *Cogn Neurodyn* 6:377–398
- Levent A (2003) Higher-order sliding modes differentiation and output-feedback control. *Int J Control* 76:924–941
- Levent A, Pavlov Y (2008) Generalized homogeneous quasi-continuous controllers. *Int J Robust Nonlinear Control* 18:385–398
- Lie X, Zhao L, Zhao G (2005) Sliding mode control for synchronization of chaotic systems with structure or parameter mismatching. *J Zhejiang Univ* 6:571–576
- Loudop P, Fotsin H, Kountchou M, Bowong S (2013) Finite time synchronization of Lorentz chaotic systems: theory and circuits. *Phys Scr* 88:045002
- Loudop P, Fotsin H, Kountchou M, Megam N, Cerdeira H, Bowong S (2014) Finite-time synchronization of tunnel-diode-based chaotic oscillators. *Phys Rev E* 89:032921–1–11
- Manwani A, Koch C (1999) Detecting and estimating signals in noisy cable structures, I: neuronal noise sources. *Neural Comput* 11:1797–1829
- Markram H, Tsodyks M (1996) Redistribution of synaptic efficacy between neocortical pyramidal neurons. *Nature* 382:807–810
- Martin W, Erik M, Stefano S, Björn S (2011) Multi-stream LSTM-HMM decoding and histogram equalization for noise robust keyword spotting. *Cogn Neurodyn* 5:253–264
- Megam E, Fotsin H, Loudop F (2014a) The combined effect of dynamic chemical and electrical synapses in time-delay-induced phase-transition to synchrony in coupled bursting neurons. *Int J Bifurc Chaos* 24:1450069–1–16
- Megam E, Fotsin H, Loudop F (2014b) Implementing a memristive Van der Pol oscillator coupled to a linear oscillator: synchronization and application to secure communication. *Phys Scr* 89:035201
- Megam E, Kabong M, Kamdoun V, Fotsin H (2015) Phase synchronization of bursting neural networks with electrical and delayed dynamic chemical couplings. *Eur Phys J B* 88:299
- Megam E, Fotsin H, Loudop P, Kamdoun V, Cerdeira H (2016) Bifurcation and multistability in the extended Hindmarsh–Rose neuronal oscillator. *Chaos Solitons Fractals* 85:151–163 (in press)
- Meister M, Wong R, Baylor D, Shatz C (1991) Synchronous burst of action potentials in ganglion cells of the developing mammalian retina. *Science* 252:939–943
- Motter A, Matias M, Kurths J, Ott E (2006) Dynamics of complex networks and applications. *Phys D* 224:vii–viii
- Nutzel K, Kien J, Bauer K, Altman J, Krey U (1994) Dynamics of diluted attractor neural networks with delays. *Biol Cybern* 70:553–561
- Pinto R, Varona P, Valkovskii A, Szucs A, Abarbanel H, Rabinovich M (2000) Synchronous behavior of two coupled electronic neurons. *Phys Rev E* 62:2644
- Rabinovich M, Varona P, Selverston A, Abarbanel H (2006) Dynamical principles in neuroscience. *Rev Mod Phys* 78:1213–1265
- Restrepo J, Ott E, Hunt B (2006) Emergence of synchronization in complex networks of interacting dynamical systems. *Phys D* 224:114–122
- Schneidman E, Freedman B, Segev I (1998) Ion channel stochasticity may be critical in determining the reliability and precision of spike timing. *Neural Comput* 10:1679–1703
- Selverston A, Moulins M (1987) The crustacean stomatogastric system: a model for the study of central nervous system. Springer, Berlin
- Selverston A, Rabinovich M, Abarbanel H, Elson R, Szucs A, Pinto R, Huerta R, Varona P (2000) Reliable circuits from irregular neurons: a dynamical approach to understanding central pattern generators. *J Physiol* 94:357–374
- Shu Y, Hasenstaub A, Badoual M, Bal T, McCormick D (2003) Barrages of synaptic activity control the gain and sensitivity of cortical neurons. *J Neurosci* 23:10388–10401
- Softky W, Koch C (1993) The highly irregular firing pattern of cortical cells is inconsistent with temporal integration of random EPSPs. *J Neurosci* 13:334–350
- Sprott J (2000) A new class of chaotic circuits. *Phys Lett A* 266:19–23
- Sto-Trevino C, Rabbah P, Marder E, Nadim F (2005) Computational model of electrically coupled intrinsically distinct pacemaker neurons. *J Neurophysiol* 94:590–604
- Stratonovich R (1961) Selected problems in the theory of fluctuations in radio engineering. Sovetskoe Radio, Moscow
- Stratonovich R (1965) Topics in the theory of random noise. *SIAM Rev* 7:574–576
- Szucs A, Elson R, Rabinovich M, Abarbanel H, Selverston A (2001) Nonlinear behavior of sinusoidally forced pyloric pacemaker neuron. *J Neurophysiol* 85:1623–1638
- Tchitnga R, Loudop P, Fotsin H, Wofo P, Fomethe A (2013) Synchronization of simplest two-component hartley's chaotic circuits: influence of channel. *Nonlinear Dyn* 74:1065–1075
- Varona P, Torres J, Huerta R, Abarbanel H, Rabinovich M (2001) Regularization mechanisms of spiking-bursting neurons. *Neural Netw* 14:865–875
- Walter J (2009) Deep analysis of perception through dynamic structures that emerge in cortical activity from self-regulated noise. *Cogn Neurodyn* 3:105–116
- Wellens T, Shatokhin V, Buchleitner A (2004) Stochastic resonance. *Rep Prog Phys* 67:45–105
- White J, Rubinstein J, Kay A (2000) Channel noise in neurons. *Trends Neurosci* 23:131–137
- Wulfram G, Werner M (2002) Spiking neuron models, single neurons, populations, plasticity. Cambridge University Press, Cambridge
- Yang T, Shao H (2002) Synchronizing chaotic dynamics with uncertainties based on a sliding mode control design. *Phys Rev E* 65:046210