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Chaotic dynamics in neural systems

Krishna Pusuluri, Huiwen Ju and Andrey Shilnikov

Abstract Several basic mechanisms of chaotic dynamics in phenomenological and biologically plausible models of individual neurons are discussed. We show that chaos occurs at the transition boundaries between generic activity types in neurons such as tonic spiking, bursting and quiescence, where the system can also become bi-stable. The bifurcations underlying these transitions give rise to period-doubling cascades, various homoclinic and saddle phenomena, torus-breakdown, and chaotic mixed mode oscillations in such neuronal systems.

1 Introduction

Neurons exhibit various activity regimes and state transitions that reflect their intrinsic ionic channel behaviors and modulatory states. The fundamental types of neuronal activity can be broadly defined as quiescence, subthreshold and tonic spiking oscillations, as well as bursting composed of alternating periods of spiking activity and quiescence. A single neuron can endogenously demonstrate various bursting patterns, varying in response to the external influence of synapses, or to the intrinsic factors such as channel noise. The co-existence of bursting and tonic spiking, as well as several different bursting modes have been observed in modeling [1, 2, 3, 4, 5]

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and experimental [6, 7, 8] studies. This complexity enhances the flexibility of the nervous and locomotive systems [9].

The functional role of chaotic behaviors, and the dynamical and bifurcational mechanisms underlying their onset at transitions between neural activity types like spiking, bursting and quiescence, have been the focus of various theoretical and experimental studies. Bursting is a manifestation of multiple time scale dynamics, composed of repetitive fast tonic spiking and a slow quiescent phase. It has been observed in various fields of science as diverse as food chain ecosystems [10], nonlinear optics [12], medical studies of the human immune system [11], and neuroscience [13]. Various bursting patterns, whether regular or chaotic, endogenous or as emergent network phenomena, are the natural rhythms generated by central pattern generators (CPG) [14, 15, 16, 17, 18]. CPGs are neural networks made up of a small number of constituent neurons that often control various vital repetitive locomotive functions [16] such as walking and respiration of humans, or the swimming and crawling of leeches [19, 20, 21, 22]. Polyrhythmic bursting dynamics have also been observed in multifunctional CPG circuits that produce several coexisting stable oscillatory patterns or bursting rhythms, each of which is associated with a particular type of locomotor activity of the animal [23, 24, 25]. Bursting has also been frequently observed in pathological brain states [26, 27], in particular, during epileptic seizures [28, 29]. Neurons in bursting modes differ in their ability to transmit information and respond to stimulation from those in tonic spiking mode and therefore play an important role in information transfer and processing in normal states of the nervous system.

Understanding and modeling the generic mechanisms regulating the neuronal connectivity and the transitions between different patterns of neural activity, including global bifurcations occurring in neuron models and networks, pose fundamental challenges for mathematical neuroscience, with a number of open problems [30]. The range of bifurcation and dynamical phenomena underlying bursting *transcends* the existing state of the theory [31, 32, 33, 34, 35, 36, 37, 39, 38, 40, 41]: this includes the blue sky catastrophe [42, 43], torus-canard formation and breakdown, and homoclinic inclination/orbit-flip bifurcations, all of which can occur on the transition route to bursting in most square-wave and elliptic bursters. Studies of bursting require nonlocal homoclinic bifurcation analysis, which is often based on the Poincaré return mappings [44]. Return mappings have been employed for computational neuroscience in [45, 46, 47, 48]. A drawback of mappings constructed from time series is sparseness, as they reflect only the dominating attractors of a system. In some cases, feasible reductions to one or two dimensional mappings can be achieved through slow–fast scale decomposition of the phase variables for the system [51]. A new, computer assisted method for constructing a complete family of *onto* mappings for membrane potentials, for a better understanding of simple and complex dynamics in neuronal models, both phenomenological and of the Hodgkin–Huxley type [52], was proposed in [53]. With this approach one can study, for example, the spike-adding transitions in the leech heart interneuron model, and how chaotic dynamics in between is associated with homoclinic tangle bifurcations of some threshold saddle periodic orbits [54]. Qualitative changes in a system's ac-

tivity at transitions often reveal the quantitative information about changes of certain biophysical characteristics associated with the transition. This approach has proven to be exemplary in neuroscience for understanding the transitions between silence and tonic-spiking activities [55]. Moreover, knowledge about the bifurcation (transition) predicts cooperative behavior of interconnected neurons of the identified types [56].

In this Chapter, we discuss nonlocal bifurcations in generic, representative models of neurodynamics, described by high order differential equations derived through the Hodgkin-Huxley formalism. We consider a number of neuroscience-related applications to reveal a multiplicity of causes and their bifurcation mechanisms leading to the onset of complex dynamics and chaos in these models.

2 Neuronal activities and transition mechanisms

This Chapter deals with neuronal models, both biologically plausible and phenomenological, that can produce complex and distinct dynamics such as tonic spiking, bursting, quiescence, chaos, and mixed-mode oscillations (MMOs) representing fast spike trains alternating with subthreshold oscillations. MMOs are typical for many excitable systems describing various (electro)chemical reactions, including the famous Belousov-Zhabotinky reaction, and models of elliptic bursters [57]. Geometrical configurations of slow-fast neuron models for bursting were pioneered in [49, 50, 55] and further developed in [58, 59, 60]. Dynamics of such singularly perturbed systems are determined by and centered around the attracting pieces of the slow motion manifolds. These are composed of equilibria and limit cycles of the fast subsystem [61, 62, 63, 64, 65, 66, 67, 68], that in turn constitute the backbones of bursting patterns in a neuronal model. Using the geometric methods based on the slow-fast dissection, where the slowest variable becomes a control parameter, one can detect and follow the branches of equilibria and limit cycles in the fast subsystem. The slow-fast decomposition allows for drastic simplification, letting one clearly describe the dynamics of a singularly perturbed system. A typical Hodgkin-Huxley model possesses a pair of such manifolds [50, 67]: quiescent and tonic spiking, respectively. The slow-fast dissection has been proven effective in low-order mathematical models of bursting neurons far from the bifurcation points. However, this approach does not account for the reciprocal, often complex interactions between the slow and fast dynamics, leading to the emergence of novel dynamical phenomena and bifurcations that can *only* occur in the whole system. Near such activity transitions, the bursting behavior becomes drastically complex and can exhibit deterministic chaos [32, 37, 39, 71, 72, 73, 74, 75, 76].

Slow-fast decomposition

Many Hodgkin-Huxley type models can be treated as a generic slow-fast system

$$\mathbf{x}' = \mathbf{F}(\mathbf{x}, z) \quad z' = \mu G(\mathbf{x}, z, \alpha), \tag{1}$$

where $0 < \mu \ll 1$, $\mathbf{x} \in R^n$, $n \geq 2$, and z is a scalar, or can be a vector in R^2 (as in the extended Plant model with two slow variables below); α is a control parameter shifting the slow nullcline, given by $G(\mathbf{x}, \alpha) = 0$, in the phase space. In the singular limit $\mu = 0$, the z-variable becomes a parameter of the fast subsystem to detect and continue the equilibrium state (ES), given by $\mathbf{F}(\mathbf{x}, z) = 0$, and the limit cycles (LC) of the fast subsystem. As long as they (ES/LC) remain exponentially stable, by varying z one can trace down the smooth invariant manifolds in the phase space of (1) such as M_{eq} with the distinct Z-shape typical for many Hodgkin-Huxley type models (see Fig. 1), while the limit cycles form a cylinder-shaped surface M_{lc}. Locally, either is a center manifold for (1) persisting in a closed system, in virtue of [61, 62, 63]. The stable upper and lower branches of M_{eq} correspond to the de- and hyperpolarized steady states of the neuron, respectively. Folds on M_{eq} correspond to the saddle-node equilibrium states of the fast subsystem. The unstable de-polarized branch of M_{eq} can be enclosed by the tonic-spiking manifold M_{lc} typically emerging through an Andronov-Hopf bifurcation and terminating through a homoclinic bifurcation, which are the key features of the fast-subsystem of the square-wave bursters [77], like the Hindmarsh-Rose model [78] and the Chay model [47] (discussed below).

Poincaré mappings

To elaborate on the nature of complex oscillations like bursting and their evolutions, one needs to examine nonlocal bifurcations that often require the use of Poincaré return maps [45, 46, 47, 72, 79, 80, 81, 82, 83]. An obvious drawback of maps constructed from voltage time series is in their sparseness, as they can typically reveal some point-wise attractors of the system that trajectories fast converge to, unless there is a noise or small perturbations are added to get a more complete picture of the underlying structure. In some cases, a feasible reduction to low-dimensional mapping can be achieved through slow–fast scale decomposition of slow phase variables [42, 44, 51]. We proposed and developed a new computer assisted algorithm for constructing a dense family of *onto* mappings for membrane potentials in a Hodgkin–Huxley type neuronal model [38]. Such maps let us find and examine both the stable and unstable solutions in detail; moreover, unstable points are often the primary organizing centers globally governing the dynamics of the model in question. The construction of such a map begins with the localization of the tonic spiking manifold M_{lc} in the model, using the parameter continuation technique or the slow-fast dissection, see Fig. 1. Then, a curve on M_{lc} is defined, which corresponds to minimal (maximal) voltage values, denoted, say, by V_0. By construction, the 1D map M takes all V_0 (outgoing solutions integrated numerically) on this curve back onto itself, after a single turn around M_{lc}, i.e., $M : V_0 \to V_1$ for a selected value of the parameter. Two such maps are depicted in Figs. 1 and 2. One can see that these are non-invertible [84, 85], unimodal maps with a single critical point [86, 87], which happens to be a universal feature of many other square-

wave bursters in neuroscience applications. With such maps, one can fully study the attractors, the repellers and their bifurcations, including saddle-nodes, homoclinic orbits, spike-adding, and period-doubling. We note that detection of homoclinics of a saddle periodic orbit in the phase space of a model is in general *state-of-the art* and the Poincaré map technique allows us to locate them with ease.

Classifications of bursting

The existing classifications [49, 50, 55, 58, 59, 60] of bursting are based on the bifurcation mechanisms of dynamical systems in a plane, which initiate or terminate fast trajectory transitions between the slow motion manifolds in the phase space of the slow-fast neuronal model. These classifications allow us to single out the classes of bursting by subdividing mathematical and realistic models into the following subclasses: elliptic or Hopf-fold subclass (FitzHugh-Rinzel [57] and Morris-Lecar models), square-wave bursters or fold-homoclinic subclass (Hindmarsh-Rose model [40, 88], models of pancreatic β-cells, cells in the pre-Botzinger complex, as well as intrinsically bursting and chattering neurons in neocortex); parabolic or circle-circle subclass (model of R15 cells in the abdominal ganglion of the mollusk Aplysia [4, 24], the reduced leech interneuron model at certain parameter values); and fold-fold subclass, or top hat models [69], including the reduced heart interneuron model (3) discussed below.

Transition routes

The current description of the transition routes between tonic spiking and bursting activities is incomplete and remains a fundamental problem for both neuroscience and the theory of dynamical systems. The first theoretical mechanism revealed in [71], explained chaos in the so-called square wave bursters [50] emerging between tonic-spiking and bursting. Later, two global bifurcations that occur at the loss of stability of a tonic spiking periodic orbit through quite novel homoclinic saddle-node bifurcations, were discovered and explained. The first transition, reversible and continuous, found in the reduced model of the leech heart interneuron [32, 36] and in a modified Hindmarsh-Rose model of a square-wave burster [40, 44], is based on the blue sky catastrophe [44, 89, 90, 91, 92]. This was proven in [42] to be a typical bifurcation for slow-fast systems. This striking term [93], the *blue sky catastrophe*, stands for a novel bifurcation of a saddle-node periodic orbit with a 2D unstable manifold returning to the orbit making infinitely many revolutions. After the bifurcation, this homoclinic connection transforms into a long bursting periodic orbit with infinitely many spikes. The burst duration of the orbit near the transition is evaluated by $1/\sqrt{\alpha}$, where $0 < \alpha \ll 1$ is a bifurcation parameter. The second transition mechanism is due to a saddle-node periodic orbit with non-central homoclinics [94]. An important feature of this transition is the bi-stability of co-existing tonic spiking and bursting activities in the neuron model, see Fig. 1. In this case, the

burst duration towards the transition increases as fast as $|\ln(\alpha)|$. Another feature of this bifurcation is the transient chaos where the neuron generates an unpredictable number of burst trains before it starts spiking tonically. This phenomenon is a direct consequence of the Smale horseshoe finite shift dynamics in the system [95], which is a rather atypical phenomenon for such slow-fast systems.
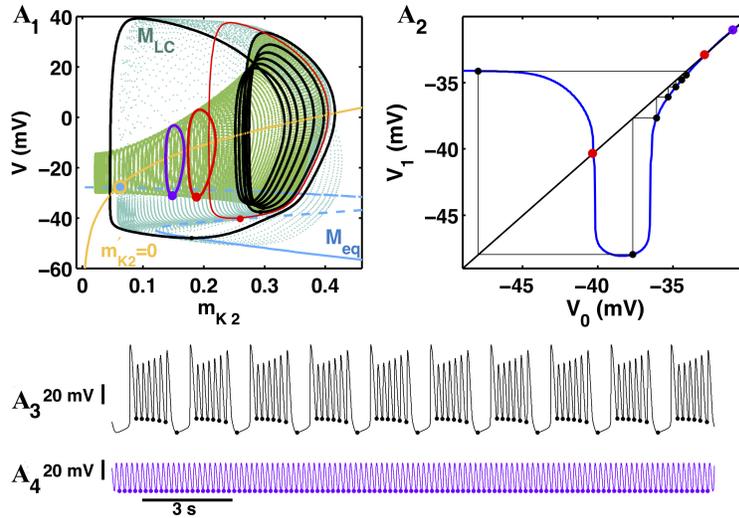

Fig. 1 (A_1) Bistability of the coexisting tonic-spiking and bursting in the 3D phase space of the leech heart interneuron model (3). Inset A_2 depicts the shape of the corresponding 1D Poincaré map with stable fixed point corresponding to the tonic spiking periodic orbit (purple) with a single voltage minima, and period-7 bursting orbit, and 2 unstable fixed points (red): the right one separates attraction basins of tonic-spiking (A_4) and bursting A_3) activities, whereas the left one causes chaotic dynamics at spike adding transitions, see Fig. 2.

3 Chaos in neuron models

In this section, we present the basic mechanisms and routes to chaos in a variety of biophysically realistic neuronal models exhibiting rich and complex dynamics including tonic spiking, bursting and quiescence. A bifurcation describing a transition between neuronal activities typically occurs near saddle (unstable) orbits and results from reciprocal interactions involving the slow and fast dynamics of the model. Such interactions lead to the emergence of new dynamical phenomena and bifurcations that can occur only in the full model, but not in either of the slow or the fast subsystem. Chaotic dynamics can be characterized by unpredictable variations in the number of spikes during the active phases of bursting and/or the subthreshold

oscillations. This phenomenon of chaotic dynamics is generally atypical in slow-fast systems as it occurs within narrow parameter windows only near the transition boundaries. Indeed, robust and regular dynamics of slow–fast neuron models contrast those of real bursting neurons exhibiting a phenomenal time dependent variability of oscillatory patterns.
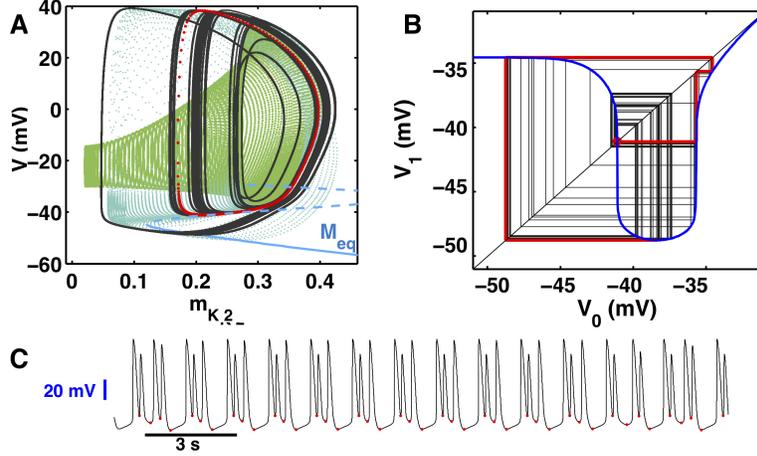

Fig. 2 (**A**) Chaotic bursting in the phase space of the leech heart interneuron model (3) and the corresponding map (**B**) at a transition between two and three spikes per burst in the voltage trace (**C**) due to proximity of the primary homoclinic orbit of the repelling fixed point (red) corresponding to a single minimum of the saddle periodic orbit (red) in (**A**).

Leech heart interneuron model: period doubling cascades and the blue sky catastrophe

We first illustrate and discuss the onset of chaotic dynamics in the reduced (3D) model of the leech heart interneuron (see equations (3) of Appendix). This is a *typical* slow-fast Hodgkin-Huxley type (HH) model describing the dynamical interplay of a single slow variable – persistent potassium current, I_{K2}, and two fast variable – the sodium current, I_{Na} and the membrane voltage V that can be recast in this generic form [36, 37, 77, 96]:

$$CV_i' = -\sum_j I_j - \sum_i I_i^{\text{syn}}, \quad \tau_h h' = f_\infty(V) - h, \quad (2)$$

where C is a membrane capacitance, V is a transmembrane voltage, I_j stands for various in/outward currents including synaptic ones, $0 \le h \le 1$ stands for a gating (probability) variable, f_∞ is a sigmoidal function, and τ_h is a time scale, fast or slow, specific for specific currents.
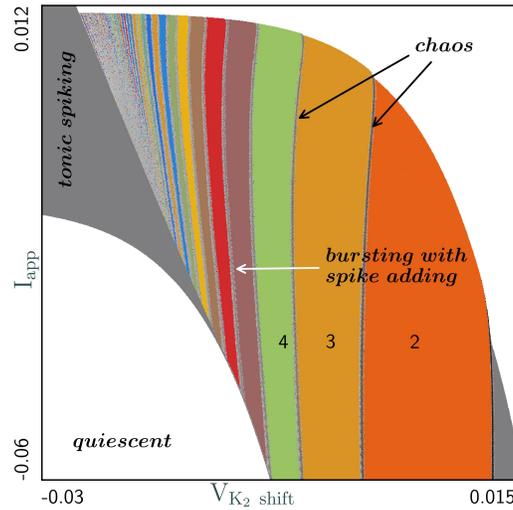
Fig. 3 Bi-parametric sweep of the leech heart interneuron model (3) using the symbolic toolkit *Deterministic Chaos Prospector* [97, 98, 99] to process wave-form traces and to reveal regions of quiescent behavior, tonic spiking, as well as bursting activity with spike adding cascades: from 2 spikes (orange zone) to 3 spikes (yellowish zone), next to 4 spikes (light green zone) and so forth. The noisy regions near the boundaries of spike addition reveal the occurrence of chaos, while the noisy boundary between tonic spiking and bursting portrays the blue sky catastrophe [32] corresponding to infinitely long bursting.

This model shows a rich set of dynamics and can produce various types of complex chaotic and bistable behaviors, including the period-doubling cascade *en a route* from tonic spiking through bursting [32, 39], as well as various types of homoclinic chaos. Following the period-doubling cascade, the model demonstrates a terminal phase of chaotic tonic spiking that coexists alongside another periodic tonic spiking activity. For a different set of parameter values compared to the period doubling cascade, the model can also exhibit the blue sky catastrophe as a continuous and reversible mechanism of the transition between bursting and tonic spiking. Fig. 1 explains the nature of bi-stability in this model as it exhibits the co-existing tonic-spiking and bursting oscillations corresponding to the stable fixed point (FP) (purple) and the period-7 orbit in the 1D map, whose basins are separated by an unstable FP representing a saddle periodic orbit (red) on the 2D manifold M_{lc} in the 3D phase space. The role of the other unstable (red) FP is revealed by Fig. 2. It is shown that the spike-adding in bursting is accompanied with an onset of chaotic dynamics orchestrated by the homoclinic orbits and bifurcations involving the other saddle orbit, see more details in [43, 54, 57, 77, 78, 88, 96]. Fig. 3 shows the bifurcation diagram of the system constructed as a parametric sweep using our previously developed symbolic toolkit called the *Deterministic Chaos Prospector* [97, 98, 99] to process symbolic sequences extracted from wave-form traces and analyze activity types and underlying bifurcations. This bifurcation diagram identifies the regions of

quiescence, tonic spiking, as well as bursting with spike adding cascades. The noisy regions near the boundaries of spike addition reveal the occurrence of chaos. In addition, the blue sky catastrophe takes place at the noisy region near the boundary between bursting and tonic spiking.

Period-doubling in the Chay model
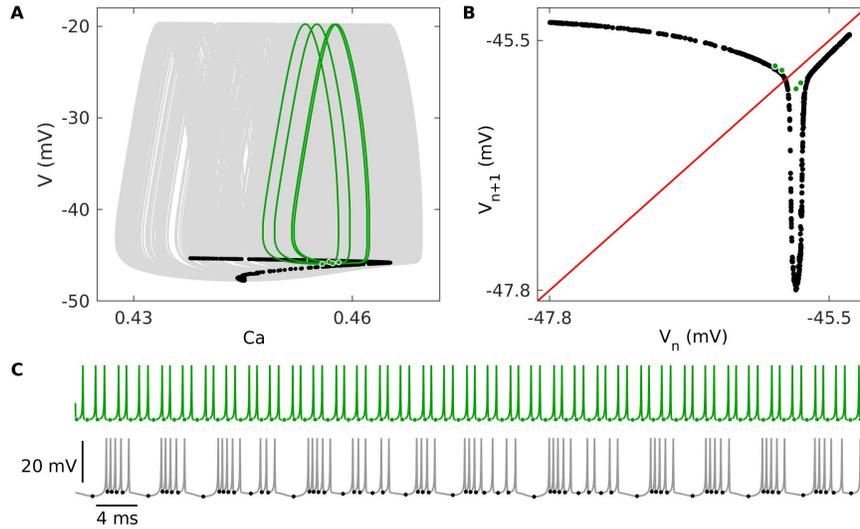
Fig. 4 A The (V, Ca) phase space projection overlaying a period-4 orbit (green, $g_{K,C} = 11.12$) and a chaotic bursting trajectory (grey, $g_{K,C} = 11.5$) generated by the Chay model. Here V_{min} – minimal values, labeled with green and black dots in he voltage traces (**C**), are used to generate 1D Poincaré return maps: $V_{min}^{(n)} \rightarrow V_{min}^{(n+1)}$ in Inset **B**.

The Chay model is a simple, realistic biophysical model for excitable cells, producing endogenous chaotic behavior (see its Eqs. (5) of Appendix). The model transitions from tonic spiking to bursting via period-doubling bifurcations, whereby chaotic dynamics can also arise. Fig. 4 shows the 2D (V, Ca)-phase space projection of the Chay model with a period-4 orbit and a chaotic bursting orbit, along with the corresponding Poincaré return map. The model goes through a period-doubling cascade and then immediate chaotic bursting, before regular bursting as the bifurcation parameter $g_{K,C}$ increases.

Torus breakdown in the bull frog hair cell model
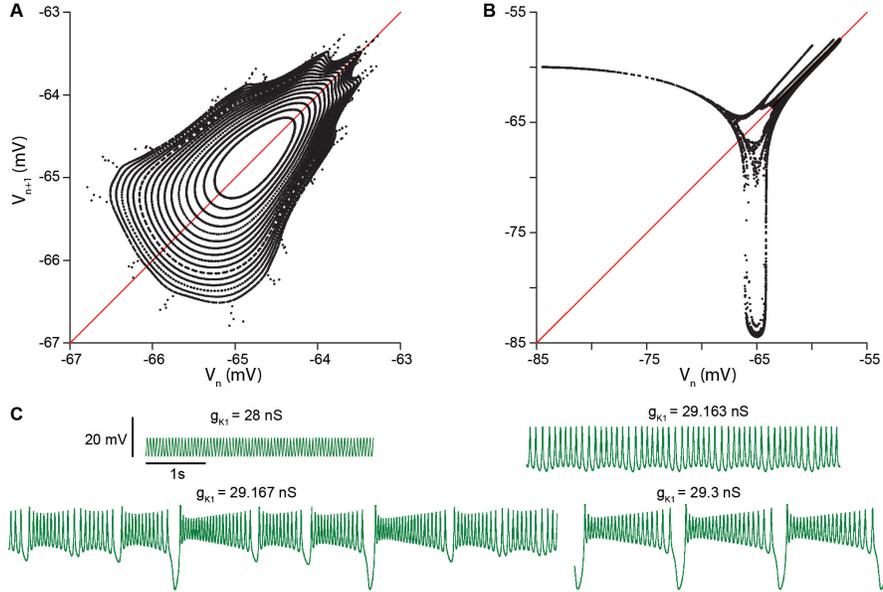
Fig. 5 Poincaré return map, $V_{\min}^{(n)} \to V_{\min}^{(n+1)}$, for the consecutive $V_{\min}$-values in voltage traces generated by the hair cell model. **A** Evolution of stable invariant circles (IC) from ergodic to resonant with further non-smooth torus breakdown as the g_{K1} parameter is increased from 29.185 through 29.2073 nS. **B** Chaotic bursting after the torus breakdown at $g_{K1} = 29.213$ nS. The flat, stabilizing section of the map corresponds to hyperpolarized quiescence, while multiple sharp folds reveal a ghost of the non-smooth IC in the depolarized range. **C** En route from tonic spiking to regular bursting, the voltage trace undergoes quasi-periodicity and chaotic bursting. This figure is adapted from [129].

Next, we consider the hair cell model based on experimental studies of basolateral ionic currents in saccular hair cells in bullfrog [130, 131, 132, 133]. This is a further extension of the model of the Hodgkin-Huxley type developed in [132] that includes 12 coupled nonlinear ordinary differential equations, see [96] for its detailed description. In this model, the transition from bursting to tonic spiking is due to a torus bifurcation (TB) that leads to onset of quasi-periodic dynamics [129]. Closer to this bifurcation the torus breaks down causing the onset of chaotic bursting in the system. In case of a supercritical TB, through which a stable torus emerges at the fold of the tonic spiking manifold M_{LC} (like one in Fig. 1), its development – growth and breakdown can be well studied using the Poincaré return maps. For example, Fig. 5A depicts that, right after the supercritical TB in the hair cell model, a stable torus (invariant circle) emerges from a stable tonic-spiking periodic orbit and grows from smooth and ergodic to non-smooth to resonant as the bifurcation parameter g_{K1} increases. Later, when the torus breaks down (starting at $g_{K1} = 29.213$ nS),

bursting becomes chaotic as shown in the Poincaré map (Fig. 5B). Fig. 5C illustrates the route from tonic spiking to bursting with chaotic dynamics at the torus breakdown.

Chaotic mixed-mode oscillations in the extended Plant model
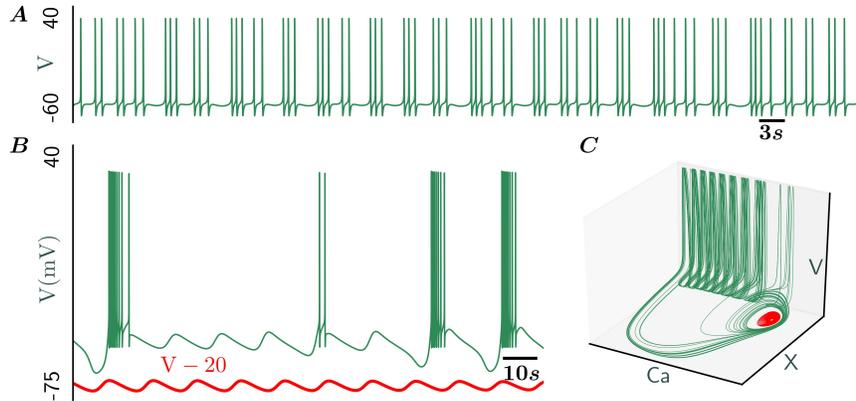
Fig. 6 The extended Plant model can exhibit chaotic bursting near the boundaries of tonic spiking and bursting with spike-adding (**A**) as well as bistability with chaotic mixed mode oscillations (green) and hyperpolarized quiescence (red) near the transitions between bursting with spike-adding and hyperpolarized quiescence (**B**). The corresponding phase space projection of the bistable states of (**B**) are shown in (**C**). Following a subcritical Andronov-Hopf bifurcation, a saddle periodic orbit (not seen) separates the chaotic mixed mode bursts (green) from the hyperpolarized quiescent state with spiral convergence (red).

The conductance-based Plant model of endogenous parabolic bursters was originally developed to model the R15 neuron in the abdominal ganglion of the slug Aplysia Californica [4]. This was later extended and adapted to model the swim CPG of the sea slug *Melibe Leonina*, see [24] for details of the model and the equations. This model can produce chaotic bursting activity, as shown in Fig. 6A near the boundary between tonic spiking and bursting activity. In addition, the model exhibits complex chaotic *mixed mode oscillations* (MMOs) near the transition between bursting and the co-existing hyper-polarized quiescence state. Fig. 6B illustrates the model generating spike-varying bursts and small amplitude sub-threshold oscillations. Such chaotic MMOs coexist with a hyperpolarized quiescent state resulting in bistability due to a subcritical Andronov-Hopf bifurcation that gives rise to a saddle periodic orbit whose stable manifold separates the chaotic bursting activity (green) from the stable (spiraling) hyperpolarized quiescent state (red) as shown in Fig. 6C. As the parameters are varied gradually the system transitions from this bistable state

to the monostable hyperpolarized quiescence, or vice verse to a dominant bursting activity.

Appendix

Leech heart interneuron model

The reduced leech heart model is derived using the Hodgkin-Huxley formalism:

$$
\begin{aligned}
C\,V' &= -I_{\text{Na}} - I_{\text{K2}} - I_{\text{leak}} + I_{\text{app}}, \\
\tau_{\text{Na}} h'_{\text{Na}} &= h^\infty_{\text{Na}}(V) - h, \\
\tau_{\text{K2}} m'_{\text{K2}} &= m^\infty_{\text{K2}}(V) - m_{\text{K2}},
\end{aligned}
\tag{3}
$$

with

$$
I_{\text{leak}} = 8(V+0.046), \quad I_{\text{K2}} = 30\,m^2_{\text{K2}}(V+0.07), \quad I_{\text{Na}} = 200[m^\infty_{\text{Na}}(V)]^3\,h_{\text{Na}}\,(V-0.045),
$$

and where V is the membrane potential, $C = 0.5$; h_{Na} is a fast ($\tau_{\text{Na}} = 0.0405$ sec) activation of $I_{\text{N}a}$, and m_{K2}; I_{L} describes the slow ($\tau_{\text{K2}} = 0.25$ sec) activation of I_{K2}, I_{app} is an applied current. The steady states $h^\infty_{\text{Na}}(V)$, $m^\infty_{\text{Na}}(V)$, $m^\infty_{\text{K2}}(V)$, of the of the gating variables are given by the Boltzmann equations given by

$$
\begin{aligned}
h^\infty_{\text{Na}}(V) &= [1 + \exp(500(0.0333 + V))]^{-1}, \\
m^\infty_{\text{Na}}(V) &= [1 + \exp(-150(0.0305 + V))]^{-1}, \\
m^\infty_{\text{K2}}(V) &= [1 + \exp\left(-83(0.018 + V^{\text{shift}}_{\text{K2}} + V)\right)]^{-1}.
\end{aligned}
\tag{4}
$$

The bifurcation parameter $V^{\text{shift}}_{\text{K2}}$ of the model is a deviation from the experimentally determined voltage $V_{1/2} = 0.018$V corresponding to the half-activated potassium channel, i.e. to $m^\infty_{\text{K2}}(0.018) = 1/2$. In its range $V^{\text{shift}}_{\text{K2}}$ is $[-0.025; 0.0018]$V the upper boundary corresponds to the hyperpolarized quiescent state of the neuron, whereas the model produces spiking oscillations at the lower end $V^{\text{shift}}_{\text{K2}}$ values and bursts in between.

Chay model

The 3D Hodgkin-Huxley type Chay model reads as follows:

$$V' = -g_I m_\infty^3 h_\infty \left(V - V_I\right) - g_{K,V} n_\infty^4 \left(V - V_K\right) - g_{K,C} \frac{C}{1+C} \left(V - V_K\right) - g_L \left(V - V_L\right),$$
$$n' = \left(n_\infty\left[V\right] - n\right)/\tau_n\left[V\right],$$
$$C' = \rho\left\{m_\infty^3 h_\infty \left(V_C - V\right) - k_C C\right\},$$

(5)

where n represents the gating variable of the voltage-sensitive K^+ channel and C represents the intracellular free calcium concentration. See [47] for the detailed description.

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