Bursting

From Scholarpedia

Eugene M. Izhikevich (2006), Scholarpedia, 1(3):1300. doi:10.4249/scholarpedia.1300 revision #47177 [link to/cite this article]

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Bursting is a dynamic state where a neuron repeatedly fires discrete groups or **bursts** of spikes. Each such burst is followed by a period of quiescence before the next burst occurs. A burst of two spikes is called a *doublet*, of three spikes is called a *triplet*, four - *quadruplet*, etc. Neuronal bursting can play important roles in communication between neurons. In particular, bursting neurons are important for motor pattern generation and synchronization.

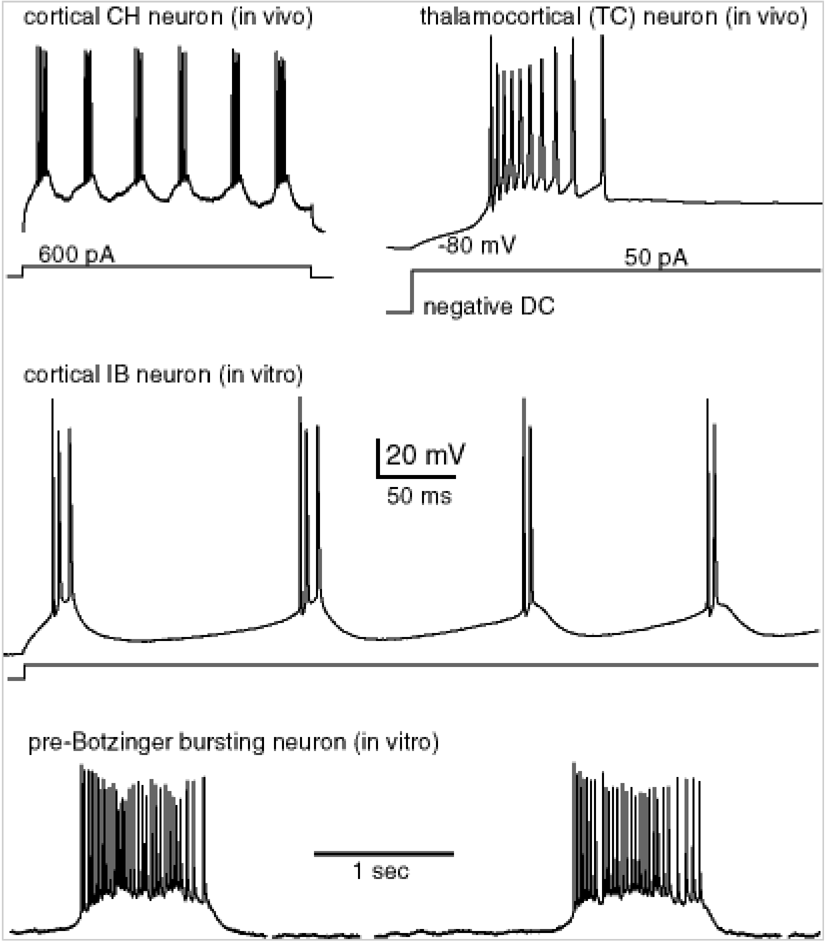


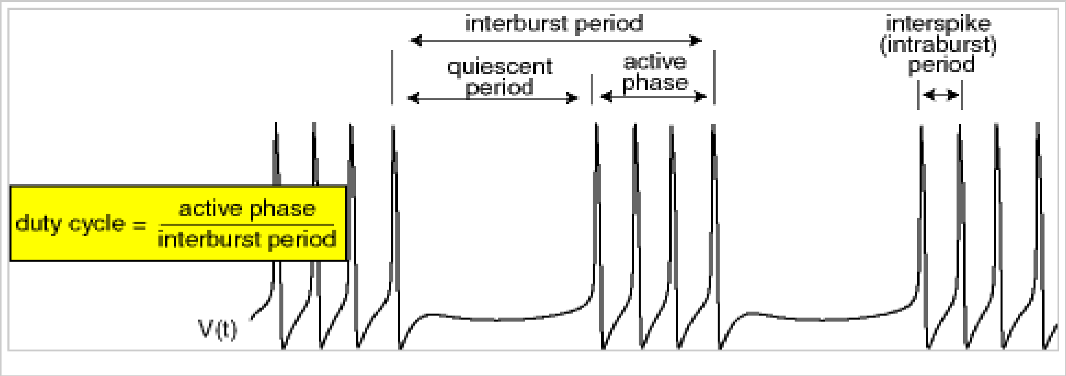
Figure 1: Modified from Izhikevich (2007) with permission. Data were provided by D. McCormick (CH cell), C.L. Cos and S.M. Sherman (TC cell), G. Stuart and M. Kole (IB cell), and C. Del Negro (pre-Bot cell).

Contents

Examples

Almost every neuron can burst if stimulated or manipulated pharmacologically. Many burst autonomously due to the interplay of fast ionic currents responsible for spiking activity and slower currents that modulate the activity. Below is the list of the more "famous" bursting neurons.

- Neocortex
  - IB: Intrinsically bursting neurons, if stimulated with a long pulse of dc current, fire an initial burst of spikes followed by shorter bursts, and then tonic spikes (Connors and Gutnick 1990). These are predominantly pyramidal neurons in layer 5.
  - CH: Chattering neurons can fire high-frequency bursts of 3-5 spikes with a relatively short interburst period (Gray and McCormick 1996). Some call them fast rhythmic bursting (FRB) cells. These are pyramidal neurons in layer 2-4, mainly layer 3.
  - Interneurons: Some cortical interneurons exhibit bursting activity in response to pulses of dc current (Markram et al. 2004).
- Hippocampus
  - LTB: Low-threshold bursters fire high-frequency bursts in response to injected pulses of current. Some of these neurons burst spontaneously (Su et al. 2001). These are pyramidal neurons in CA1 region.
  - HTB: High-threshold bursters fire bursters only in response to strong long pulses of current.
- Thalamus
  - TC: Thalamocortical neurons can fire bursts if inhibited and then released from inhibition. This rebound burst is often called a *low-threshold spike*. Some fire bursts spontaneously in response to tonic inhibition.
  - RTN: Reticular thalamic nucleus inhibitory neurons have bursting properties similar to those of TC cells.
- Cerebellum
  - PC: Purkinje cells in cerebellar slices usually fire tonically but when synaptic input is blocked they can switch to a trimodal pattern which includes a bursting phase (Womack and Khodakhah 2002).



- Other structures
  - pre-Bot: Respiratory neurons in pre-Botzinger complex fire rhythmic bursts that control animal respiration cycle.
  - MesV: Some Mesencephalic V neurons in brainstem may fire rhythmic bursts when slightly depolarized above the threshold.
  - AB: Anterior bursting neuron in lobster stomatogastric ganglion fires rhythmic bursts autonomously.
  - R15: Aplysia abdominal ganglion neuron R15 fires autonomous rhythmic bursts.
  - β-cell: Pancreatic β-cells fire rhythmic bursts that control the secretion of insulin.

Sometimes the term *bursting* is used in reference to the bursting pacemaker potential itself and not just the burst of spikes. So, in the literature, one might see references to bursts in the presence of TTX. There are also examples of one spike bursts such as in the CPGs for swimming in Clione and in Xenopus tadpoles.

Detection of Bursts

It is relatively easy to identify bursts in response to simple stimuli, such as dc steps or sine waves, especially if recording intracellularly from a quiet in vitro slice. The bursts fully evolve and the hallmarks of burst responses are clear. However, responses to sensory stimuli are often comprised of doublets or triplets embedded in spike trains. Furthermore, these responses are usually recorded extracellularly so the experimenter does not have access to the membrane potential fluctuations that are indicative of bursting. Thus, it is difficult to distinguish burst responses from random multispike events.

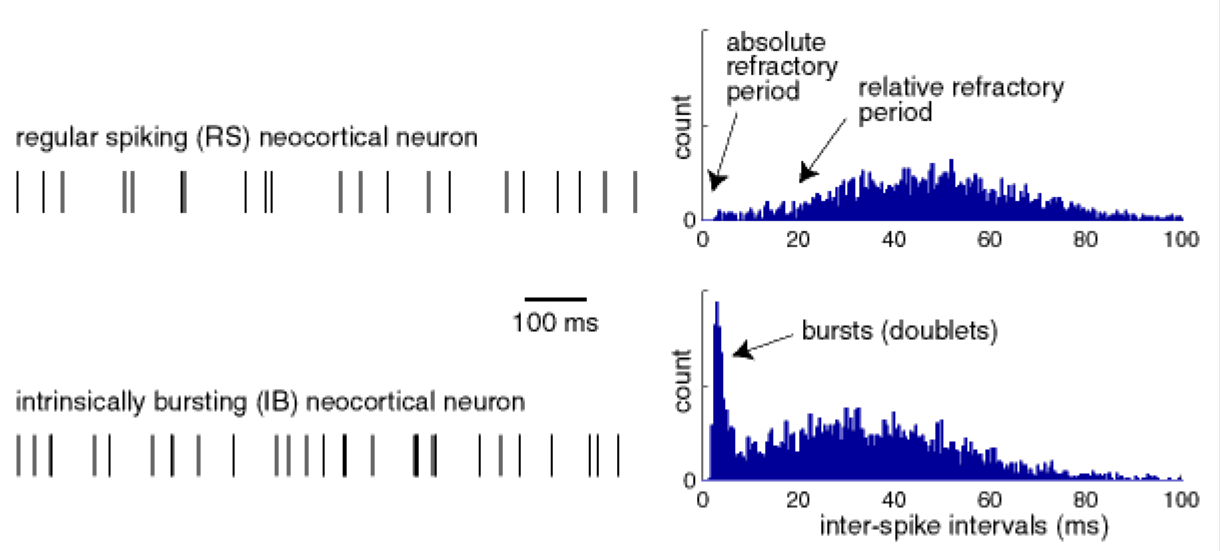


Figure 2: Interspike interval histograms of two model neurons with simulated strong in vivo input

The statistical analysis of spike trains addresses this problem. Bimodal inter-spike interval (ISI) histograms can be indicative of burst responses. The rationale is that short ISIs occur more frequently when induced by burst dynamics than would occur if predicted by Poisson firing. Burst spikes with short ISIs form the first mode while quiescent periods correspond to the longer ISIs of the second mode. This is true for intrinsic or forced (stimulus driven and network-induced) bursting. Furthermore, the trough between the two modes may correspond to the refractory period of an intrinsic burst or the timescale of the network-induced bursting (Doiron et al. 2003). This method defines a criterion for burst identification so that further analysis and experimentation can determine the mechanism and function of the bursts. See Holt et al., (1996) and Bastian and Nguyenkim (2001) for a deeper analysis into burst detection from stochastic spike data.

Bursts as a Unit of Neuronal Information

There are many hypotheses on the importance of bursting activity in neural computation.

- **Bursts are more reliable than single spikes** in evoking responses in postsynaptic cells. Indeed, excitatory post-synaptic potentials (EPSP) from each spike in a burst add up and may result in a superthreshold EPSP.
- **Bursts overcome synaptic transmission failure**. Indeed, postsynaptic responses to a single presynaptic spike may fail (release does not occur), however in response to a bombardment of spikes, i.e., a burst, synaptic release is more likely (Lisman 1997).
- **Bursts facilitate transmitter release** whereas single spikes do not (Lisman 1997). Indeed, a synapse with strong short-term facilitation would be insensitive to single spikes or even short bursts, but not to longer bursts. Each spike in the longer burst facilitates the synapse so the effect of the last few spikes may be quite strong.
- **Bursts evoke long-term potentiation** and hence affect synaptic plasticity much greater, or differently than single spikes (Lisman 1997).

- **Bursts have higher signal-to-noise ratio than single spikes** (Sherman 2001). Indeed, burst threshold is higher than spike threshold, i.e., generation of bursts requires stronger inputs.
- **Bursts can be used for selective communication** if the postsynaptic cells have subthreshold oscillations of membrane potential. Such cells are sensitive to the frequency content of the input. Some bursts resonate with oscillations and elicit a response, others do not, depending on the interburst frequency (Izhikevich et al. 2003).
- **Bursts can resonate with short-term synaptic plasticity** making a synapse a band-pass filter (Izhikevich et al. 2003). A synapse having short-term facilitation and depression is most sensitive to a burst having certain resonant interspike frequency. Such a burst evokes just enough facilitation, but not too much synaptic depression, so its effect on the postsynaptic target is maximal.
- **Bursts encode different features** of sensory input than single spikes (Gabbiani et al. 1996, Oswald et al. 2004). For example, neurons in the electrosensory lateral-line lobe (ELL) of weakly electric fish fire network induced-bursts in response to communication signals and single spikes in response to prey signals (Doiron et al. 2003). In the thalamus of the visual system bursts from pyramidal neurons encode stimuli that inhibit the neuron for a period of time and then rapidly excite the neuron (Lesica and Stanely, 2004). Natural scenes are often composed of such events.
- **Bursts have more informational content than single spikes** when analyzed as unitary events (Reinagel et al. 1999). This information may be encoded into the burst duration or in the fine temporal structure of interspike intervals within a burst.

In summary, burst input is more likely to have a stronger impact on the postsynaptic cell than single spike input, so some believe that **bursts are all-or-none events, whereas single spikes may be noise**.

Ionic Mechanisms

Forced bursting

Most spiking neurons can burst if stimulated with a current that slowly drives the neuron above and below the firing threshold. Such a current could be injected via an electrode or generated by the synaptic input. Below are some examples of forced bursters.

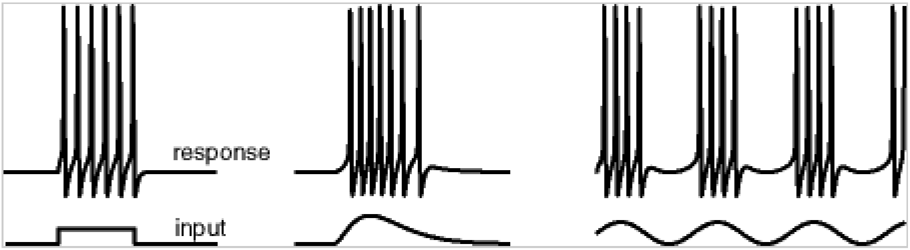


Figure 3: Forced bursting in response to injected input

- RA neurons in the songbird burst in response to drive from HVC neurons. The bursting arises as a result of either network dynamics within RA or is inherited from HVC (see review by Fee et al. 2004).
- Network induced bursts of electric fish (Doiron et al. 2003). Here bursting arises because periodic inhibitory inputs reduce firing and create intervals of quiescence.
- Electroreceptor afferents in paddlefish. Bursting occurs because of a pre-filtering of broadband stochastic stimuli that drives the receptors. The receptor dynamics can be modeled as a simple excitable system and the slow noise (filtered) pushes the neuron into periods of rapid firing and into periods of quiescence (Neiman and Russell, 2002).

Intrinsic bursting

Many neurons have slow intrinsic membrane currents that can modulate fast spiking activity. Typically, the currents build up during continuous spiking, hyperpolarize the cell and result in the termination of the spike train. While the cell is quiescent, the currents slowly decay, the cell recovers, and it is ready to fire another burst.

Much experimental research is aimed at identifying these slow currents, so that bursting can be manipulated. To produce an inhibitory effect, i.e., to stop the burst, spiking activity should either

- activate (turn on) an outward current, e.g., persistent K^+ current, or
- inactivate (turn off) an inward current, e.g., transient Ca^{2+} current.

Such action can be either

- voltage-gated or
- Ca^{2+}-gated.

Hence there are at least four different theoretical mechanisms of modulation of spiking; see table.

Examples of currents that modulate spiking

	voltage-gated	**Ca^{2+}-gated**
activation of outward	M-current	AHP-current
inactivation of inward	Ca^{2+} T-current	Ca^{2+} L-current

All four mechanisms have been documented in vitro. For example, neocortical pyramidal neurons exhibiting chattering activity have voltage-gated K^+ M-current (Wang 1999). Midbrain dopaminergic neurons have Ca^{2+}-gated K^+ current K_{Ca} (Amini et al. 1999). In both cases, continuous spiking activates the outward K^+ currents and results in burst termination. Thalamic relay neurons have voltage-gated Ca^{2+} T-current (Huguenard and McCormick 1992). Anterior bursting (AB) neurons in lobster stomatogastric ganglion have Ca^{2+}-gated Ca^{2+}-current, called L-current (Harris-Warrick and Flamm 1987). In both cases, continuous spiking inactivates the inward Ca^{2+} currents and also results in burst termination. Many neurons have a mixture of mechanisms described in the table. In all these cases, the interspike intervals within the burst is typically *increasing*.

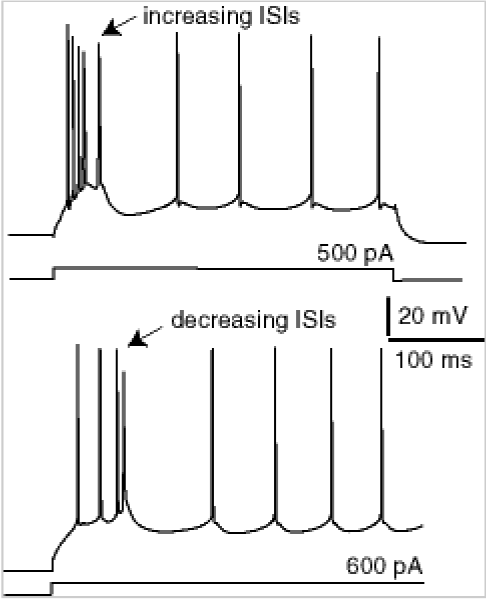


Figure 4: Bursting can be with increasing or decreasing interspike intervals (ISI). Modified from Izhikevich (2007) with permission.

In addition, bursting can result from the **somatic-dendritic interplay**: Somatic spike excites dendritic tree resulting in a delayed spike there. Dendritic spike depolarizes the soma and evokes another somatic spike, which results in another dendritic spike, and so forth. The interspike intervals of the burst is typically *decreasing* up to the point when a somatic spike falls into the refractory period of the dendritic spike, fails to evoke dendritic response, and stops the burst (Doiron et al. 2002).

Mathematical Mechanisms

Different ionic mechanisms of bursting may result in different mathematical mechanisms, which in turn determine the neuro-computational properties of bursters, i.e., how they respond to the input. Therefore, much effort is devoted to study and classify the dynamics of bursting, as reviewed in detail by Coombes and Bressloff (2005) and Izhikevich (2007).

Most mathematical models of bursters can be written in the fast-slow form

$$\begin{aligned} \dot{x} &= f(x,y) && \text{(fast spiking)} \\ \dot{y} &= \mu g(x,y) && \text{(slow modulation)} \end{aligned}$$

where vector x describes the state of the fast subsystem responsible for spiking activity, vector y describes the state of the slow subsystem that modulates spiking, f and g are some Hodgkin-Huxley-type functions, and $\mu << 1$ is the ratio of time scales.

Dissection of neuronal bursting

A standard method of analysis of fast-slow bursters, as well as of any singularly perturbed system, is to set $\mu = 0$ and consider the fast and the slow subsystems separately. This is known as **dissection** of neuronal bursting (Rinzel 1985), since it allows us to study the fast subsystem

$$\dot{x} = f(x,y)$$

and treat y as a vector of slowly changing bifurcation parameters. Typically, the fast subsystem has a limit cycle (spiking) attractor for some values of y and an equilibrium (resting) attractor for other values of y. As the slow variable oscillates between the two values, the fast subsystem, and hence the whole system, burst.

Slow-wave bursting

What makes the slow variable oscillate? In the simplest case, the slow subsystem $\dot{y} = \mu g(x,y)$ may have a limit cycle attractor, which is relatively insensitive to the value of the fast variable. In this case, the slow variable exhibits an autonomous oscillation that periodically drives the fast subsystem over the threshold. Such a bursting is called **slow-wave** bursting. The slow subsystem must be at least two-dimensional to exhibit slow-wave bursting.

Slow-wave bursting in conductance-based models is usually more interesting than the simplest case described above. In such models, the slow subsystem often consists of activation and inactivation gates of slow currents. For example, $y = (n,h)$ with

$$\dot{n} = (n_\infty(V) - n)/\tau_\mathrm{n}(V)$$
$$\dot{h} = (h_\infty(V) - h)/\tau_\mathrm{h}(V)\,.$$

Obviously, such a slow subsystem is uncoupled for any fixed value of the fast variable, i.e., in the voltage clamp ($V = \mathrm{const}$). Yet, it can still have a limit cycle attractor if the fast variable is allowed to evolve, i.e., the membrane potential V is allowed to spike.

Hysteresis-loop bursting

When the equilibrium and limit cycle attractors of the fast subsystem co-exits for the same value of y, there is a bistability of resting and spiking states. This creates a hysteresis loop for the slow variable and such a bursting is called **hysteresis-loop** bursting. The slow variable y may be one-dimensional in this case, oscillating between resting and spiking values via the hysteresis loop.

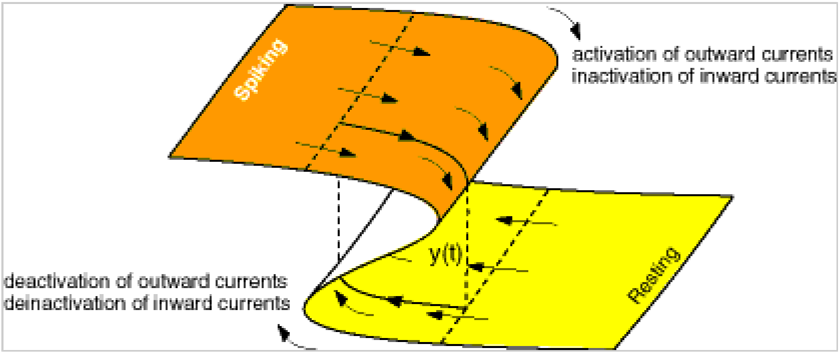
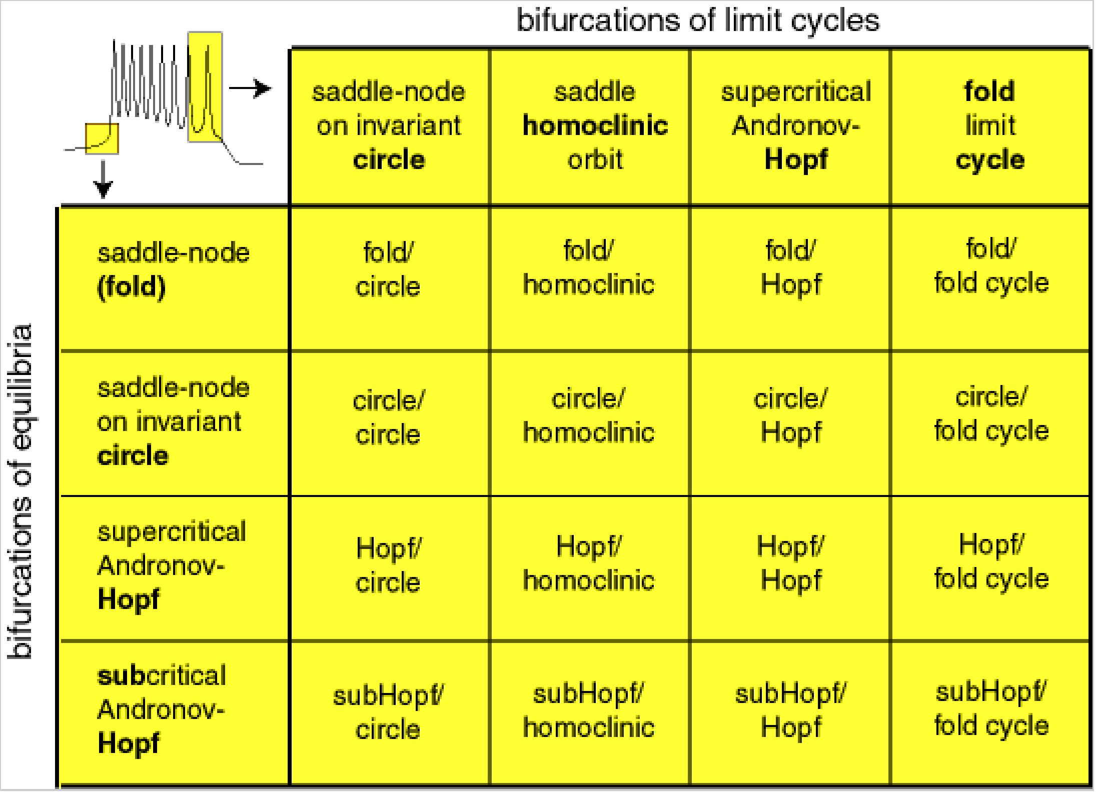


Figure 5: Hysteresis-loop bursting (modified from Izhikevich (2007) with permissions).

When the fast variable x is in the spiking state, the slow variable, governed by the equation $\dot{y} = \mu g(x,y)$, is pushed toward the region of quiescence (resting, rightward in the figure), and spiking abruptly stops. When the fast variable is quiescent, the slow variable is pushed toward the region of spiking (leftward in the figure) and after a while spiking abruptly starts. These transitions from spiking to resting and back correspond to bifurcations of the fast subsystem.

Topological Classification



bifurcations of limit cycles

bifurcations of equilibria

	saddle-node on invariant **circle**	saddle **homoclinic** orbit	supercritical Andronov-**Hopf**	**fold** limit **cycle**
saddle-node **(fold)**	fold/ circle	fold/ homoclinic	fold/ Hopf	fold/ fold cycle
saddle-node on invariant **circle**	circle/ circle	circle/ homoclinic	circle/ Hopf	circle/ fold cycle
supercritical Andronov-**Hopf**	Hopf/ circle	Hopf/ homoclinic	Hopf/ Hopf	Hopf/ fold cycle
subcritical Andronov-**Hopf**	subHopf/ circle	subHopf/ homoclinic	subHopf/ Hopf	subHopf/ fold cycle

Figure 6: Topological classification of fast-slow bursters having two-dimensional fast (spiking) subsystem. Reproduced from Izhikevich (2007) with permission.

At first glance, all bursters look the same - clusters of spikes separated by long periods of quiescence. A closer look reveals that bursters may be different quantitatively, but are they different qualitatively?

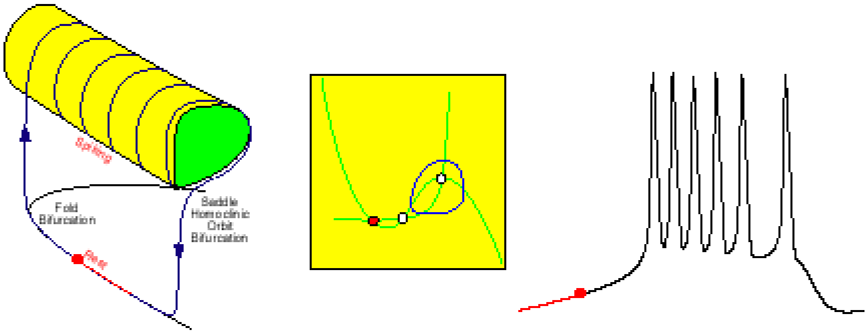
Bursters are distinguished qualitatively according to their topological type. There are two important bifurcations of the fast subsystem that determine the topological type:

- **resting to spiking**: Bifurcation of a stable equilibrium (resting) that results in the transition to limit cycle attractor (spiking).
- **spiking to resting**: Bifurcation of a limit cycle attractor that results in the transition to the equilibrium (resting).

There are only 4 possible bifurcations of co-dimension 1 of equilibrium, depicted in the first column in the figure. If the fast subsystem is two-dimensional, then there are only 4 possible bifurcations of co-dimension 1 of a limit cycle attractor, depicted in the first row of the figure. Hence, there are 16 different combinations, resulting in 16 different topological types of fast-slow bursters with 2-dimensional fast subsystems. They are named after the bifurcations involved.

The classification of bursters according to the bifurcations of the fast subsystem was first suggested by Rinzel (1987), who identified three types presented below. It was later extended by Bertram et al. (1995), who included another type. Izhikevich (2000) provided the complete classification, identifying all 16 topological types in the figure, and 120 all possible types if the constraint that the fast subsystem is two-dimensional is removed. Burst mechanisms can also be classified via unfolding of a higher co-dimension bifurcation of the fast subsystem (Golubitsky et al. 2001).

Fold/homoclinic



Probably the most frequent type of bursting encountered in simulations is the fold/homoclinic type, also known as **square-wave** bursting, because the burst profile sometimes looks like a square. While the slow variable moves to the left along the lower (resting) branch in the 3D phase portrait in the figure, the fast subsystem undergoes fold (saddle-node off limit cycle) bifurcation resulting in the transition from resting to spiking. As it spikes, the slow variable moves to the right along the cylinder corresponding to spiking state, the fast subsystem undergoes saddle homoclinic orbit bifurcation resulting in the transition from spiking to resting. The slow variable starts to move to the left again, thereby closing the hysteresis loop.

This type of bursting has been reported in many cells and cell models, including pancreatic β-cells, cells in the pre-Botzinger complex, intrinsically bursting and chattering neurons in neocortex.

Circle/circle

This type of bursting is a prominent feature of R15 cells in the abdominal ganglion of the mollusk Aplysia. It was called **parabolic** because the frequency profile within a burst looks like a parabola. The transition from spiking to resting and back to spiking of the fast subsystem occurs via saddle-node on invariant circle bifurcation. The slow subsystem of circle/circle bursters is usually (but not necessary) two-dimensional or higher exhibiting an autonomous slow-wave oscillation that drives periodically the fast subsystem over the threshold.

SubHopf/fold cycle

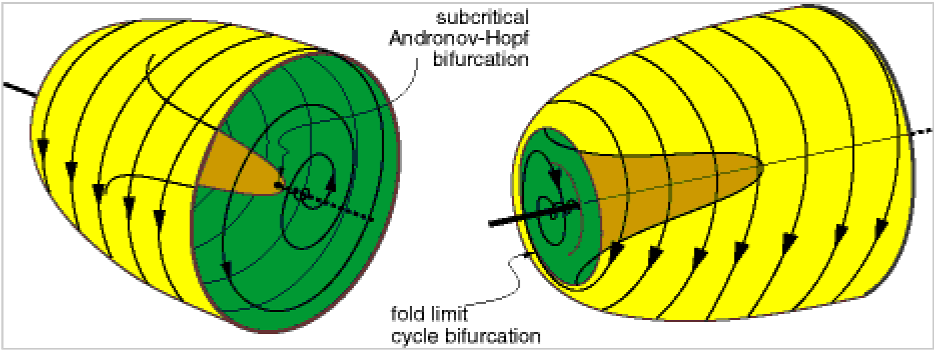


Figure 8: SubHopf/fold cycle bursting.

This type of bursting is also called **elliptic** because the amplitude profile of the burst sometimes looks like an ellipse. A three-dimensional phase portrait of the burster is depicted in the figure from two different points of view. While the fast subsystem exhibits spiking oscillations (the outer cylinder), the slow variable moves to the left and the spiking stops via fold limit cycle bifurcation. While the fast subsystem is resting, the slow variable moves along the center axis to the right and the spiking starts via subcritical Andronov-Hopf bifurcation, thereby closing the hysteresis loop.

This type of bursting has been reported in rodent trigeminal interneurons, dorsal root ganglion, and mesencephalic

V cells of the brainstem.

Neuro-computational Properties

Mathematical studies of bursters revealed that different topological types have different neuro-computational properties (Izhikevich 2007):

- Bursters that involve Andronov-Hopf bifurcation (Hopf/* and subHopf/*) act as resonators, i.e., they are sensitive to the frequency content of the synaptic input. In contrast, the other types (fold/* and circle/*) act as integrators.
- Bursters that involve fold, subcritical Andronov-Hopf, saddle homoclinic orbit, and fold limit cycle bifurcations have co-existence of resting and spiking states, and hence have a bistable or Multistable dynamics. An appropriately timed input can switch bursting activity from spiking to quiescence and back. The input does not even have to be excitatory.
- Different topological types of bursters have different synchronization properties. Some tend to synchronize in-phase, others tend to de-synchronize.

Open Problems

- The complete classification of all electrophysiological types of bursters is still missing.
- There is no methodology of how to distinguish different topological types experimentally.
- The relationship between electrophysiology of a cell and the topological type of its bursting is not worked out.
- It is not fully understood how synchronization properties of bursters depend on their topological types.
- There is no complete classification of all possible transitions between rhythmic bursting and tonic spiking, and rhythmic bursting and total quiescence. That is, what are the bifurcation mechanisms of appearance and disappearance of bursting oscillations? (see also Routes into Bursting).
- Mathematical theory suitable to study bursting activity is not fully developed yet. Most asymptotic methods break down when transitions between limit cycles are considered.

Development of early dynamical systems theory was influenced by the studies of oscillators. It is reasonable to expect that the next major developments of the theory would be coming from studies of bursting dynamics.

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External Links

- Author’s website (http://www.izhikevich.com/)
- Bursting (http://en.wikipedia.org/wiki/Bursting) in Wikipedia

See also

Bifurcations, Bistability, Burst Synchronization, Multi-stability in Neuronal Models, Neuronal Excitability, Routes into Bursting, Stability, Stomatogastric Ganglion

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Created: 8 February 2006, reviewed: 3 March 2006, accepted: 5 March 2006
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