Deterministic Chaos in Mathematical Model of Pacemaker Activity in Bursting Neurons of Snail, *Helix Pomatia*

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Chaotic regimes in a mathematical model of pacemaker activity in the bursting neurons of a snail, *Helix pomatia*, have been investigated. The model includes a slow-wave generating mechanism, a spike-generating mechanism, an inward Ca current, intracellular Ca ions, [Ca$^{2+}$]_{in}, their fast buffering and uptake by intracellular Ca stores, and a [Ca$^{2+}$]_{in}-inhibited Ca current. Chemosensitive voltage-activated conductance, $g_{\text{B}}$, responsible for termination of the spike burst, and chemosensitive sodium conductance, $g_{\text{Na}}$, responsible for the depolarizing phase of the slow-wave, were used as control parameters. These conductances in intact snail bursting neuron are regulated by neuropeptides. Time courses of the membrane potential and [Ca$^{2+}$]_{in} were employed to analyse different regimes in the model. Histograms of interspike intervals, autocorrelograms, spectral characteristics, one-dimensional return maps, phase plane trajectories, positive Lyapunov exponent and especially cascades of period-doubling bifurcations demonstrate that approaches to chaos were generated. The bifurcation diagram as a function of $g_{\text{B}}$ and the ([Ca$^{2+}$]_{in}-V) phase diagram of initial conditions reveal fractal features. It has been observed that a short-lasting depolarizing current or elevation of [Ca$^{2+}$]_{in} may evoke transformation of chaotic activity into a regular bursting one. These kinds of transitions do not require any changes in the parameters of the model. The results demonstrate that chaotic regimes of neuronal activity modulated by neuropeptides may play a relevant role in information processing and storage at the level of a single neuron.

1. Introduction

It is established that some excitable cells possess different types of electrical pacemaker activity. Well-known bursting neurons of different mollusks (*Aplysia* R15 neuron, *Tritonia* 22 cell, *Otala* 11 cell, *Helix* F1 or RPa1 neuron) can exhibit bursting or beating pacemaker activity or have no intrinsic electrical activity (Frazier *et al*., 1967; Gainer, 1972; Ifshin *et al*., 1975; Kerkut *et al*., 1975; Kononenko, 1993; Sakharov & Salanki, 1969; Smith & Thompson, 1987; Willows & Hoyle, 1968). Sometimes the bursting neurons spontaneously (see Section 3) or under a polarizing current (Hayashi & Ishizuka, 1992) or drug application (Labos & Lang, 1978; Haydon *et al*., 1982; Holden *et al*., 1982), demonstrate an irregular electrical activity which can be interpreted as chaotic or a chaotic-like one (see for review Elbert *et al*., 1994). Single cells are hypothesized to be able to exhibit a variety of behaviour due to their strong nonlinearity. Unfortunately, the assessment of chaos in most cases studied was determined mainly by visual inspection of the time series, and the cogent experimental arguments were presented for only the *Onchidium* pacemaker bursting neuron (Hayashi & Ishizuka, 1992). The chaotic behaviour of *Onchidium* bursting pacemaker neuron caused by a d.c. bias current is suggested by the presence of a strange

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attractor, and the Poincare map shows a non-invertible function with an unstable fixed point. Transformation of the silent state of a burster to beating or bursting activity can take place spontaneously or can be evoked by stimulation of appropriate peptidergic interneurons forming synaptic contacts with bursting neuron (Pin & Gola, 1983; Kononenko & Osipenko, 1986).

At present, the ionic basis for the generation of the MP wave in bursting neurons is not well understood and there are some physical models and corresponding mathematical versions describing a slow wave of MP on the crest of which a burst of spike arises (Canavier et al., 1990, 1991, 1993; Chay, 1985, 1990, 1993; Hayashi & Ishizuka, 1992; Plant, 1981; Rinzel & Lee, 1987; Wang, 1994). These models demonstrate beating or bursting activity that depends on model parameters. Transformation of beating activity into the bursting one in the models, which consist of slow and fast subsystems, occurs through cascades of period doubling and arising chaotic regimes (Canavier et al., 1990; Chay, 1984, 1993; Hayashi & Ishizuka, 1992).

In the preceding papers (Kononenko, 1994a; Berezetskaya et al., 1996) a mathematical model of pacemaker activity in bursting neurons of snail, Helix pomatia, based on experimental data (Kononenko, 1993) has been described. The present paper deals with the analysis of chaos and routes to it in this model of bursting activity. Interspike intervals (ISIs) which are a basis for integrative processes in neuronal nets were used for identification and analysis of different types of neuronal activity. A preliminary report on our findings has been published in abstract form (Komendantov & Kononenko, 1995).

2. Model and Methods

The model is described by a set of eight first-order differential equations and includes four main components: a slow-wave generation mechanism, a spike-generating mechanism consisting of both Hodgkin–Huxley type TTX-sensitive sodium and TEA-sensitive potassium currents (Hodgkin & Huxley, 1952), a voltage-activated Ca current, intracellular calcium ions, [Ca$^{2+}$]$_i$, their fast buffering and uptake by intracellular Ca stores, and a [Ca$^{2+}$]$_i$-inhibited stationary voltage-dependent Ca current (Berezetskaya et al.; Kononenko, 1994a). Equations and parameters are presented in the Appendix. Initial conditions for the membrane potential and the free internal calcium concentration have been chosen as −55 mV and 0, respectively. Integration of the equations was done mainly using a fourth-order Runge–Kutta method with a time step of 0.0001 s (Hairer et al., 1987). Occasionally 0.00025 or 0.00001 s were used to check the accuracy. So our results seem unlikely to have an artifact of numerical integration algorithms with finite precision.

The calculations were performed on IBM PC with coprocessor. Due to transient periods, the intervals occurring in the first 50 s were not used for the analysis.

The time series of the membrane potential and [Ca$^{2+}$]$_i$ have been chosen for analysis, because they are informative and observable in experiments (Gorman et al., 1981; Müller et al., 1993). Interspike intervals (ISIs) were used for identification and analysis of different types of neuronal activity. The results of computer simulation have been observed by methods of investigation of nonlinear dynamical systems: observation of evolution at the phase plane

![Discharge patterns of the identified bursting pacemaker neuron RPa1 in different preparations of snail Helix pomatia. (a) Regular beating activity. (b) Regular bursting activity. (c) Irregular activity. (d,e) Irregular bursting activity. Calibration: 60 mV, 20 s.](image-url)
Conventional methods were used to record the electrical activity from *Helix pomatia* RPa1 bursting neurons (Sakharov & Salanki, 1969; Kononenko, 1979).

### 3. Results

**Irregular Regime in Model Neuron**

Figure 1 shows the typical records of spontaneous electrical activity generated by pacemaker bursting neurons RPa1 of snail, *Helix pomatia*. These neurons can generate the beating activity (a), bursting (b), and different types of irregular activity (c–e). We are aware that in the case of spontaneous activity of intact cells we can not determine the source of irregularity, that is, the variable synaptic influx or deterministic chaos which is due to the specific interaction of the slow and fast electrical processes taking place in the bursting neuron.

The model (Kononenko, 1994a; Berezetskaya *et al.*, 1996) may demonstrate the different types of pacemaker activity depending on its parameters. As one can see in the Appendix, the model of pacemaker activity contains two types of ionic currents—fast and slow. Therefore, it can be expected that the model is able to generate the irregular discharges similar to experimental observable ones. Figure 2 demonstrates APs generated by the mathematical model of bursting neuron (upper record) and the time course of the intracellular Ca concentration (lower record) during the first 50 s. Model parameters are given in the Appendix. After a transient period of about 20 s, which is due to the fact that the initial $[\text{Ca}^{2+}]_{\text{i}}$ was zero, the generation of APs was random which is also seen from the behaviour of $[\text{Ca}^{2+}]_{\text{i}}$.

For subsequent analysis of irregular regimes of electrical activity in the model neuron, the ISI was chosen as the basic parameter. This is because the ISI, in contrast to the gating variable $q$ (Canavier *et al.*,...
beating pacemaker activity was present for parameter variation (Kononenko, 1994a). To exclude these intervals are in the physiological regions of bifurcations and the region with chaos. One may view takes place through a cascade of period-doubling potential waves in the bursting neurons (Kononenko, 1990) or AP amplitude (Hayashi & Ishizuka, 1992), has an apparent physiological significance. It seems likely that the ISI distribution is the basis of information coding in a neuronal network.

A series consisting of about 3500 ISIs was used for the time series analysis. The decaying character of the autocorrelation function of the ISIs demonstrates the random generation of APs, and its oscillating type displays a multiple periodic process of AP generation [Fig. 3(a)]. As can be seen from the ISI histogram [Fig. 3(b)] of the electrical discharge of the model neuron, two ISI values predominate. The power spectrum [Fig. 3(c)] demonstrates the fundamental frequencies and the trace of broad-band noise that may be of a chaotic nature.

**BIFURCATION ANALYSIS**

Previously, it has been hypothesized and experimental evidence has been presented that generation of persistent bursting activity, at least in bursting neurons of Helix pomatia, is not endogenous, but is related to the activity of an interneuron(s) which constantly generates nerve impulses and releases biologically active substances of a neuropeptide nature. Subsequent activation of specific conductances $g_{Na}$ and $g_{K}$ leads to the generation of membrane potential waves in the bursting neurons (Kononenko, 1979, 1993, 1994b; Kononenko & Osipenko, 1986). Considering this, we suggest that the change between different states of electrical activity (bursting, beating and silent) and the appearance of a chaotic discharge can be evoked by long-lasting alteration of chemosensitive conductances $g_{Na}$ and $g_{K}$, participating in slow-wave generation (Kononenko, 1994a; Berezetskaya et al., 1996). Thus, it is interesting to investigate the ISI dependence of the model neuron on both the $g_{Na}$ and $g_{K}$, conductances as the control parameters. Figures 4(a) and (b) show the bifurcation diagrams obtained for these parameters.

At first, 100 second time domains were calculated for different values of both $g_{Na}$ in the interval between 0.105 µS and 0.195 µS [Fig. 4(a)] and $g_{K}$ in the interval between 0.02 µS and 0.035 µS [Fig. 4(b)]. These intervals are in the physiological regions of parameter variation (Kononenko, 1994a). To exclude the transient periods, the intervals during first 50 s are not represented on the diagrams.

The only periodic trajectory corresponding to beating pacemaker activity was present for $g_{Na}$ from 0.1 µS to 0.117 µS [Fig. 4(a)]; as one can see from the diagram, the transformation of the beating activity into the bursting one ($g_{Na}$ from 0.141 µS to 0.195 µS) takes place through a cascade of period-doubling bifurcations and the region with chaos. One may view these variations of parameters as a regulation of the neuropeptide concentration on the bursting neuron receptors by changing the interneuron activity. The period doublings are well-known route to chaos (Feigenbaum, 1978, 1979; May, 1976; Schuster, 1988).

Figure 5 illustrates time domain, one-dimensional return maps and phase plane trajectories of the beating mode ($g_{Na} = 0.1100 \mu S$) and the transitions through two period-doubling cascades ($g_{Na} = 0.1240 \mu S$, $g_{Na} = 0.1300 \mu S$) to the chaotic regime ($g_{Na} = 0.1372 \mu S$ and the bursting ($g_{Na} = 0.1650 \mu S$) mode. One-dimensional return maps were constructed by sampling the values of the previous interspike interval vs. the current one (middle column). A map under $g_{Na} = 0.1372 \mu S$ has been defined using the sequential series of 3500 spikes. The map is non-invertible and has a delicate structure that is a feature of chaos (Moon, 1987; Schuster, 1988).

MP and [Ca$^{2+}$]$_{in}$ in the model neuron are the easily interpreted physiological parameters, and, therefore, projections of oscillations onto the phase plane ([Ca$^{2+}$]$_{in}$, V) (right column) have been investigated. In the case of the regular discharges the trajectories are a closed curve. However, in the case of chaotic regime the aperiodic trajectory fills some regions of the phase plane and resembles a strange attractor, as one can see from Fig. 5. It should be noted that the chaotic region on bifurcation diagram [Fig. 4(a)] is not uniform, but alternates with areas in which regular bursting activity takes place. Moreover, as the step of the control parameter, $g_{Na}$, decreases, new intermittent areas of chaotic regimes and regular bursting activity arise (Fig. 6). It seems likely that boundary in the space of parameters between the strange attractor and the limit cycle has fractal features. Route from the beating mode to the bursting one through chaos could also be observed upon $g_{Na}$ variation [Fig. 4(b)].

**SENSITIVE DEPENDENCE UPON INITIAL CONDITIONS**

All the above-described model investigations have been carried out under only the initial conditions ($V = -55$ mV, [Ca$^{2+}$]$_{in} = 0$). Sensitive dependence upon initial conditions (SDIC), which is a defining feature of chaos (Moon, 1987; Schuster, 1988; Wolf et al., 1985) has also been investigated in one study. As one can see in the diagram of initial conditions (Fig. 7), the boundary between regions of chaotic and bursting activity is not smooth. The left picture [Fig. 7(a)] is the result of integration of 1116 initial conditions. We have chosen a small part of the initial conditions and have magnified it. Figure 7(b) shows the results of this operation. It can be seen that the transition to a larger scale (441 initial conditions) displays the feature of a fractal structure. SDIC
means that small changes in the state of a system will grow at an exponential rate and will dominate the behaviour. SDIC is quantified with Lyapunov exponents (Wolf et al., 1985). In our model, the dominant Lyapunov exponent for the time series of 3500 ISIs has been estimated using the algorithm of Wolf et al. (1985) and is equal to 0.38 bits iteration.

**CHAOTIC BURSTING ACTIVITY IN THE MODEL NEURON**

In electrophysiological experiments intact bursting neurons sometimes exhibited a specific type of electrical activity which may be called a chaotic bursting one [Fig. 1(d,e)]. In this case, the duration of the burst or the interburst interval was not stable. Unfortunately, in the intact brain, it is almost impossible to investigate whether the instability of the
bursting activity is connected with endogenous (an intrinsic mechanism of bursting activity) or exogenous (activation of synaptic inputs) factors. It is interesting to note that the model studied allows us to obtain aperiodic solutions which are similar to experimental records (Parameters in the Appendix). Figure 8 (a) demonstrates quasi-bursting electrical activity during 100 s in the model. The projection of

![Figure 5](image-url)

**Fig. 5.** Time domains (left column), one-dimensional return maps (middle column), and phase plane representations (right column) as a function of increasing $g^\#$. Here and below, moving clockwise along the projections corresponds to the direction of increasing time. The figures to the right of each row of graphs are the values of $g^\#$ ($\mu$S).
the attractor on the phase plane ([Ca$^{2+}$]$_{in}$, V) has signs of both a typical bursting oscillator and a chaotic one [Fig. 8(b)]. In the chaotic bursting regime, the interaction of slow-waves is observed on the time domain record. Low amplitude waves (1 mV) with a period near 2 s are seen on the crest of the high amplitude wave (12 mV) with a period of about 12 s.
TRANSFORMATION OF SPONTANEOUS ACTIVITY

In some experiments conducted on intact bursting neurons transformation of the silent state into the bursting activity evoked by short-lasting polarizing current pulse and in a opposite direction could be observed. The presence of nonlinear dynamic properties in the model studied allows us to suggest the existence of similar behaviour of the model. Figure 9(a) demonstrates that short-lasting membrane depolarization (−0.5 nA, 2 s) transforms original chaotic activity into the bursting one. Figure 9(b) shows the corresponding time course of $[\text{Ca}^{2+}]_\text{i}$. A similar result could be obtained for an increase of $[\text{Ca}^{2+}]_\text{i}$ to 120 nM. Both influences are easily interpreted physiologically as activation of excitatory synaptic inputs or liberation of Ca$^{2+}$ from intracellular calcium stores, respectively. Transformation of electrical activity from a chaotic to a regular bursting one has an apparent interpretation on the phase plane [Fig. 9(c)]. The strange attractor corresponds to

Fig. 7. Fractal features of the diagram of initial conditions. Filled circles: chaotic activity; empty circles: bursting activity. The arrow points to the initial conditions $[\text{Ca}^{2+}]_\text{i} = 0$, $V = −55$ mV. (b) corresponds to the small square in (a). The integration of the model equations was done with a time step of 0.00025 s. Parameters are given in the Appendix (chaotic mode). The incremental steps for $[\text{Ca}^{2+}]_\text{i}$ and $V$ on the left diagram are 4 nM and 1 mV, respectively. The incremental steps on the right diagram are 0.4 nM and 0.1 mV.

Fig. 8. Chaotic bursting activity in the model. Time course of membrane potential (a) and corresponding projection on to ($[\text{Ca}^{2+}]_\text{i}$, $V$) plane (b) simulated in accordance with equations and parameters presented in the Appendix.
chaotic activity, while the limit cycle corresponds to regular bursting activity. Transformation of the electrical activity evoked by the short-lasting influence corresponds to an induced change from a low-[Ca$^{2+}$]$_{in}$ region to a high-[Ca$^{2+}$]$_{in}$ one. Similarly, the model bursting activity could be transformed into the chaotic activity by decreasing [Ca$^{2+}$]$_{in}$ or hyperpolarization of the neuronal membrane by an external current (0.5 nA, 15 s) and the subsequent accumulation of [Ca$^{2+}$]$_{in}$ by intracellular calcium stores.

4. Discussion

In the present investigation, it has been shown that a deterministic model of pacemaker activity in bursting neuron of snail *Helix pomatia* (Berezetskaya et al.; Kononenko, 1994a) under certain values of the parameters exhibits non-periodic behaviour that is in good agreement with experimental observations. In this case, an irregular electrical activity is deterministic in its origin and differs from stochastic noise. Analysis of time domains, ISI histograms, autocorrelations, spectral characteristics provides support for this suggestion. One-dimensional return maps, projections of trajectories onto a phase plane, bifurcation diagrams with cascades of period-doubling show that the irregular electrical activity is deterministic chaos. Sensitive dependence upon initial conditions and a positive dominant Lyapunov exponent for ISIs suggest this also. However, the question as to what extent the mathematical model of bursting activity, in general, and its chaotic behaviour, in particular, reflects the processes taking place in the intact burster is still not clear. We are fully aware that the dynamic activity of a real neuron is likely to be more complex that the model one.

Hayashi & Ishizuka (1992) have compared reconstructed attractors and Poincare maps of discharges of both the intact bursting neuron in *Onchidium* and its mathematical model using the polarizing current as the control parameter and the peak potentials of spikes for analysis. Satisfactory agreement was observed in this study. In other studies (Labos & Lang, 1978; Haydon et al., 1982; Holden et al., 1982) the assessment of chaos was determined only by visual inspection of the time series. At present, there are no available data which could permit one to draw final conclusions concerning the origin of the irregular activity in the intact burster of *Helix pomatia*, and subsequent experimental investigations of the interspike intervals in the intact burster demonstrating a chaotic discharge is needed.

Intact bursters in snail brain exhibit bursting, beating or silent activity depending on its state, stimulation of identified peptidergic interneuron having synaptic input to the burster, or application of a neuropeptide isolated from the CNS (Ifshin et al., 1975; Kononenko, 1979; Pin & Gola, 1983; Kononenko & Osipenko, 1986, 1990; Kononenko, 1993). This corresponds to changing the $g_{f}$ and $g_{a}$ in a physiologically significant region in our model of bursting neuron. The results of computational studies allow us to hypothesize that the irregular electrical discharge in the intact neuron could be due to a
specific combination of membrane conductances, or properties of intracellular calcium stores, but is not always a consequence of the convergence of excitatory and inhibitory postsynaptic potentials on burster functioning in neuronal assemblies. Thus, the single neuron with deterministic characteristics because of the interaction of the ionic current on its membrane can be a source of a "random process" in the CNS, and chaotic activity probably can play an essential role in neuronal networks. At present, it seems likely that the ability to exhibit chaotic behaviour is one of the fundamental properties of an excitable system with a complex type of electrical activity, which has slow and fast components (Canavier et al., 1990; Chay, 1990; Chay & Rinzel, 1985; Plant, 1981).

Type of chaotic activity in the model depends on its parameters. In this respect it should be noted that besides the typical chaotic discharge, the model presented with definite parameters exhibits so-called chaotic bursting activity. Similar activity could be observed in intact bursting neuron RPal from Helix pomatia [Figs. 1(d) & (e)].

The boundary in space of initial conditions or parameters between regions of a limit cycle and a strange attractor exhibits fractal structure. It is the reason that small variations in initial conditions or model parameters could produce essential changes in its behaviour. Actually, in model experiments a small short-lasting impulse of polarizing current which mimics a postsynaptic potential transforms the chaotic activity into the regular bursting one. Similar results were obtained in the model of bursting activity of Aplysia R15 neuron (Canavier et al., 1993; Byrne et al., 1994). Indeed, these mode transitions, which do not require any changes in the biochemical or physical parameters of the neuron, provide an enduring response to a transient input as well as a mechanism for phasic sensitivity (i.e. temporal specificity). In their model, the small changes of the chemosensitive conductances (anomalous rectifier conductance, \( g_k \), and the slow inward calcium conductance, \( g_{sa} \)) can abolish some of the coexisting modes of electrical activity. These conductances are known to be modulated by dopamine and serotonin (Byrne et al., 1994). The model neuronal system can make the transition between periodic and chaotic motions as the result of very small changes in the various parameters (Chay, 1984). The crisis activity has been demonstrated in excitable cell models (Fan & Chay, 1993): the sudden change in calcium concentration may give rise to the sudden switching on or off bursting activity of excitable membranes. In our model, the essential role of intracellular calcium ions in the transformation of spontaneous activity by short-lasting signals is demonstrated.

In summary, our model results provide a plausible explanation for the mechanism of the irregular electrical activity in typical bursting pacemaker neuron in molluscan brain.

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REMARKS

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APPENDIX

Mathematical Model of Pacemaker Activity in Bursting Neurons

The basic equation governing membrane behaviour is:

\[-C_m dV/dt = I_{Na(TTX)} + I_{K(TEA)} + I_k + I_{Na} + I_{Na(V)} + I_b + I_c + I_{Ca-Ca}.

Here, \(C_m\) is membrane capacity, \(V\) denotes the membrane potential (mV). The model includes:

(a) Slow-wave generating mechanism:

\[I_{Na}(V) = g_{Na}^{\infty}(V)(1/(1 + \exp(-0.2(V + 45)))(V - V_{Na});

\[I_k = g_k(V - V_k);

\[I_{Na} = g_{Na}^{\infty}(V - V_{Na});

\[I_b = g_{Na}^{\infty}m_bh_b(V - V_b); \]

\[d_m/dt = (1/(1 + \exp(0.4(V + 34))) - m_b)/0.05; \]

\[d_h_b/dt = (1/(1 + \exp(-0.55(V + 43))) - h_b)/1.5; \]

(b) Spike-generating mechanism (the Hodgkin–Huxley sodium TTX-sensitive and potassium TEA-sensitive currents):

\[I_{Na(TTX)} = g_{Na(TTX)}m^n h(V - V_{Na}); \]

\[I_{K(TEA)} = g_{K(TEA)}n^m(V - V_k); \]

\[d_m/dt = (1/(1 + \exp(-0.4(V + 31)) - m)/0.0005; \]

\[d_h/dt = (1/(1 + \exp(0.25(V + 45)) - h)/0.01; \]

\[d_n/dt = (1/(1 + \exp(-0.18(V + 25)) - n)/0.015; \]

(c) Calcium currents (transient voltage-dependent (\(I_{Ca}\)) and stationary \([Ca^{2+}]_{in}-\)inhibited (\(I_{Ca-Ca}\))):

\[I_{Ca} = g_{Ca}^{\infty}m_C^2(V - V_{Ca}); \]

\[d_m_C/dt = (1/(1 + \exp(-0.2V)) - m_C)/0.01; \]
\[ I_{Ca-Ca} = \frac{g_{Ca-Ca}}{1 + \exp(-0.06(V + 45))} \times \frac{1}{1 + \exp(k_b([Ca] - \beta))} (V - V_{Ca}); \]

(d) Intracellular Ca ions, their fast buffering and uptake by Ca stores:

\[ \frac{d[Ca]}{dt} = \rho \left( -I_{Ca}/2Fv - k_s [Ca] \right); \quad v = 4\pi R^3/3; \]

Here, [Ca] is [Ca\(^{2+}\)](mM), F is Faraday number, 96,485 C mol\(^{-1}\); \(v\) is the volume of the cell, \(k_s\) is the rate constant of intracellular Ca-uptake by intracellular stores, \(\rho\) is the endogenous Ca buffer capacity.

### Parameters for Equations

\[ V_{Na} = 40 \text{ mV}; \quad V_K = -70 \text{ mV}; \quad V_B = -58 \text{ mV}; \]
\[ V_{Ca} = 150 \text{ mV}; \quad C_m = 0.02 \mu\text{F}; \quad R = 0.1 \text{ mm}; \]

\[ k_s = 50 \text{ s}^{-1}; \quad \rho = 0.002; \quad \beta = 15000 \text{ s}^{-1}/\text{mM}; \]

**CHAOTIC MODE**

\[ g_{Ca}^* = 0.25 \mu\text{S}; \quad g_{Na}^* = 0.0231 \mu\text{S}; \quad g_{Na}^*(V) = 0.11 \mu\text{S}; \]
\[ g_N^* = 0.1372 \mu\text{S}; \quad g_{Na(TTX)}^* = 400 \mu\text{S}; \quad g_{TEA}^* = 10 \mu\text{S}; \]
\[ g_{Ca}^* = 1.5 \mu\text{S}; \quad g_{Ca-Ca}^* = 0.02 \mu\text{S}; \]

**CHAOTIC BURSTING MODE**

\[ g_{Ca}^* = 0.25 \mu\text{S}; \quad g_{Na}^* = 0.02 \mu\text{S}; \quad g_{Na}^*(V) = 0.13 \mu\text{S}; \]
\[ g_N^* = 0.18 \mu\text{S}; \quad g_{Na(TTX)}^* = 400 \mu\text{S}; \quad g_{TEA}^* = 10 \mu\text{S}; \]
\[ g_{Ca}^* = 1 \mu\text{S}; \quad g_{Ca-Ca}^* = 0.01 \mu\text{S}; \]

**INITIAL CONDITIONS**

\[ V = -55 \text{ mV}; \quad [Ca\(^{2+}\)]_m = 0; \]

**ABREVIATIONS**

AP, action potential; MP, membrane potential; ISI, interspike interval; TTX, tetrodotoxin; TEA, tetraethylammonium; SDIC, sensitive dependence upon initial conditions; CNS, central nervous system.