



Basic Mechanisms of Neural Coding Illustrated by Computer Simulations from Different Functional Levels*

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Abstract

In close relation to experimental data, we present simulation examples of neural coding phenomena from different levels to illustrate their functional interdependencies and to elucidate common characteristics. This goes from single ion channel recordings, to voltage and current clamp experiments, including intracellularly recorded action potentials, up to the generation of neuronal impulse pattern and their implications for neuronal network synchronization. Special attention is given to neural stochasticity emphasizing the impact on neuronal heterogeneity and noise.

Keyphrases

Ion channels, voltage clamp, action potentials, heterogeneity, noise, synchronization.

Introduction

Relating experimentally or clinically observed neural coding phenomena of to the underlying mechanisms and understanding their interdependencies can be a challenging task. Neural codes are considered to be transmitted by the temporal and spatial pattern of action potentials. Action potentials originate from the opening and closing of ion channels. Depending on the composition of ion channels the neurons can generate action potential sequences of different temporal pattern. How the neural code is interpreted also depends on the internal states of the neurons to which the information is transmitted. This again depends on how strongly the neurons are connected in neuronal networks. The collective behavior of network neurons in turn depends not only on their connectivity but likewise on the internal dynamics of their individual neurons and how they may change under specific stimulus conditions. This will be illustrated by computer simulations of neuronal dynamics at different functional levels, from ion channels to neuronal networks.

Methods

Our computer simulations are based on the classical Hodgkin-Huxley (HH) equations (Hodgkin and Huxley 1952), however, with several simplifications (Postnova et al. 2010; Tchaptchet et al. 2013). Especially, we

have replaced the complicated rate constants of the original HH equations by single exponential functions, which lead to activation curves a in explicit form of Boltzmann functions with slope s and half activation voltage V_h with $a = 1/(1 + \exp(-s(V - V_h)))$. The time delays are modeled by a first order differential equation $da/dt = (a_0 - a)/\tau$ with steady state value a_0 and time constant τ . The membrane equation $CdV/dt = \Sigma I$ of our model neurons follows the general form of mechanism-based HH-type equations. Changes of the membrane voltage V over time t across the membrane capacitance C is given by the sum of diverse ion currents I_i which are calculated according to Ohm's law $I_i = g_i a_i (V - V_i)$ with equilibrium voltages V_i and maximum conductances g_i .

In SimNeuron, we use a minimal set of ion currents. Apart from a single term for leak currents there are the voltage dependent sodium and potassium currents for action potential generation. The cold receptor model, our single-neuron pattern generator, is extended by two slow, subthreshold currents, which activate already below the voltage range of spike generation (Braun et al. 2000). The slow repolarizing current is modeled as a simplified version of a voltage- and calcium-dependent potassium current. A noise term can be added to account for a physiologically realistic randomness of spike generation. Temperature effects are implemented by Q10 values of 3.0 for activation time-constant and 1.3 for maximum conductances. Our neural network is organized in a 10x10-matrix of pattern generator neurons in torus-like form. Each neuron is coupled to its 8 direct neighbors via gap-junction currents $I_c(i, j)$ for which $I_c(i, j) = g_c \Sigma (V_{i,j} - V_{l+n, j+m})$ where g_c is the coupling conductance and $V_{i,j}$ the membrane potential of neuron (i, j) , and $V_{l+n, j+m}$ the membrane potential of the neighboring neurons with $\{i, j | 0, \dots, 9\}$ and $\{m, n | -1, 0, 1\}$.

Results

The following sections will present simulation examples ranging from single ion channel activation to voltage and current clamp recordings, including intracellularly recorded action potentials, up to the generation of neuronal impulse pattern and their implications for neuronal network synchronization. Special attention is given to neural stochasticity by emphasizing the functional effects of neuronal heterogeneity and noise.

1) Ion Channels: We start at the functionally lowest level of neural coding, the opening and closing of ion channels. Already at this level a major feature of neuronal mechanism is elucidated, namely the essential contribution of randomness which seem to progress up to the highest levels, eventually even including mental processes like decision-

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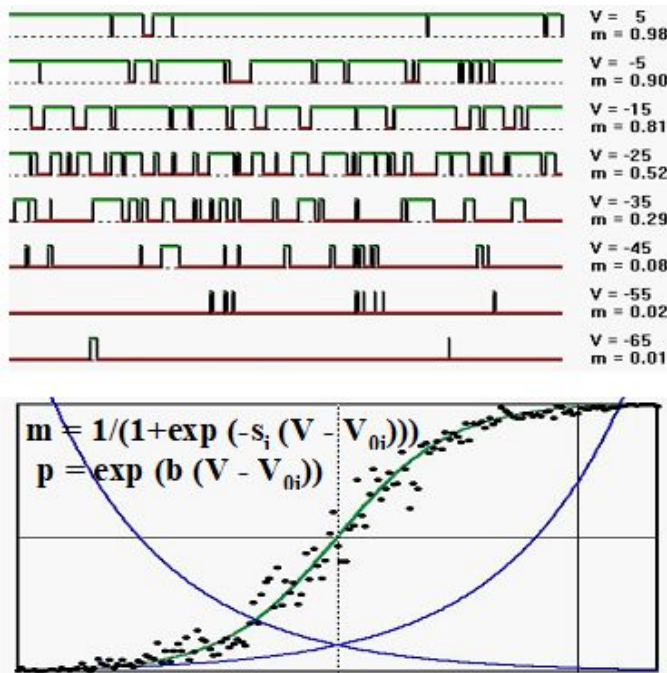


Figure 1: Simulation of voltage-dependent probability changes of ion channel opening and closing. Upper diagrams show the open (green) and closed (red) states. V is the voltage and m is the percentage of open times. Lower diagram: The exponential probability curves p (blue) lead to a sigmoid curve of open states (green). The black dots show the opening probabilities of 100 simulations at different voltages distributed along the sigmoid curve.

making (Braun 2021). Although single ion channels can only attain two states, open or close, their voltage dependencies follow a gradual, approximately sigmoid function, (Boltzmann function, Logistic function). The results from the combination of physiological rules with randomness. Apparently, ion channels randomly fluctuate between open and closed states while the membrane voltage, as well as physiological transmitters or neuromodulators, change the opening and closing probabilities. This is illustrated by the simulation example in Fig. 1 showing in the left part the open and closed states of a single ion channel as a function of the membrane voltage and on the right the exponential transition probabilities together with the resulting Boltzmann function. The black dots show the percentage of open states from 100 simulations runs, which are nicely distributed along the theoretical curve which, of course, will only be hit by chance. The lowest graph on the right shows the noise as it would result from 100 ion channels.

2) Ion currents and action potentials: For the next step we can go in a comparably easy to overlook voltage-/current clamp lab, SimNeuron (www.virtual-physiology.com), which generally is used for teaching the interdependencies of ion channel activation, ion currents and membrane voltage. This lab only considers, apart from a leak current, the voltage-dependent sodium and potassium currents for action potential generation. Action potentials (APs) arise from the opening and closing of ion channels which, however, additionally request an appropriate timing. This is illustrated in Fig. 2 showing, on the left, in addition to the sigmoid voltage dependencies also the time dependencies of Na⁺ and K⁺-channel activation, including Na⁺-channel inactivation. The voltage- and time-dependencies of ion current activation and inactivation

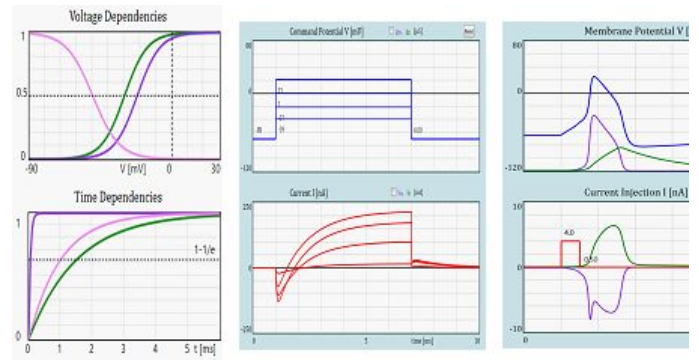


Figure 2: Left: Voltage and time dependencies of Na⁺-current activation (violet) and inactivation (pink) together with K⁺-current activation (normalized). Middle: Whole cell currents (red) of a virtual neuron in response to different voltage-clamp steps (blue). Right: Action potential (blue) of the same neuron in response to an external current pulse (red). Additionally shown are the alterations of ion currents (upper diagram) and conductances (lower diagram) in violet (for Na⁺) and green (for K⁺) Simulations taken for the virtual SimNeuron laboratory (www.virtual-physiology.com).

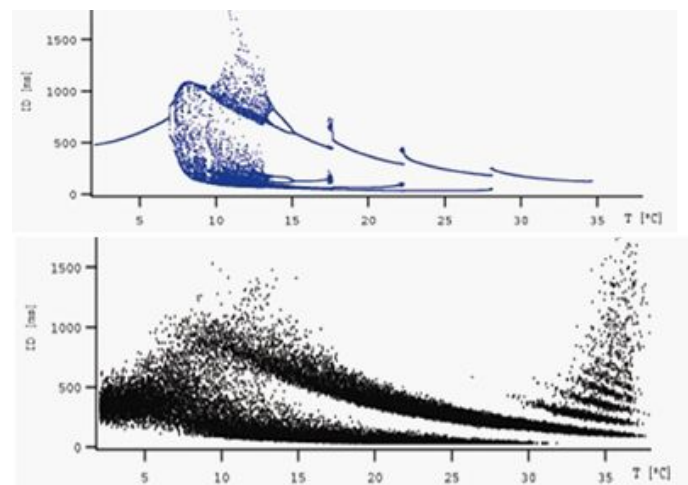


Figure 3: Bifurcation diagram of interspike-intervals of the cold receptor model in a deterministic simulation (upper diagram, blue) and with addition of voltage noise (lower diagram, black).

tion can particularly well be examined in voltage-clamp experiments as illustrated in the middle of Fig. 2. A depolarizing voltage-clamp step activates the voltage dependent Na⁺- as well as K⁺-currents. This leads to an initial transient inward current of Na⁺-ions because of their very short activation time-constant. The Na⁺-channels then inactivate and the K⁺-channels open, both with much longer time constants, which leads to a persistent K⁺-outward current, without inactivation. In this example, the repolarizing K⁺-current activates already at lower potential than the Na⁺-current. It is only due to the much faster opening of the Na⁺-channels which nevertheless allows the generation of action potentials. This is illustrated in right graphs of Fig. 2 which also show the Na⁺- and K⁺-conductances and currents which, by the way, in real experiments cannot be recorded simultaneously. Appropriate simulation approaches can be very useful tools also to connect experimental data (Tchaptchet et al. 2013).

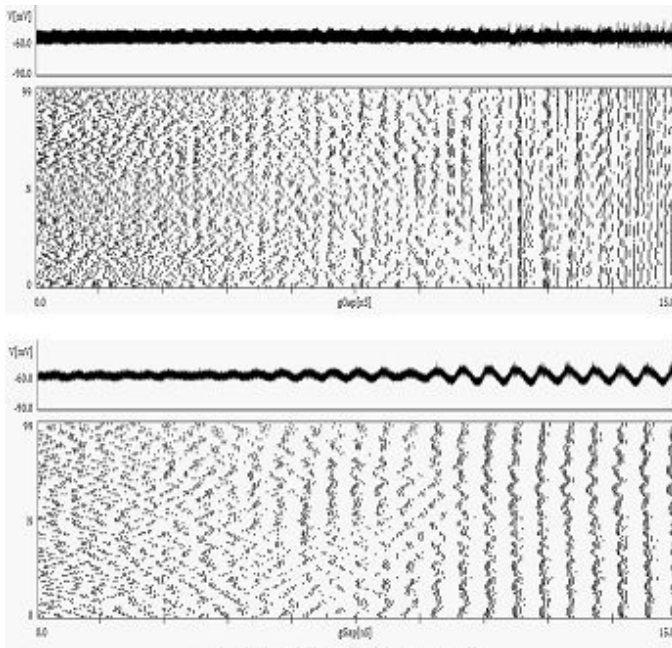


Figure 4: Synchronization simulations of two networks of 100 gap-junction coupled neurons in a torus-like map during increasing coupling from 0 to 15 nS. The graphs show the raster plot of spike times of all 100 neurons together with their averaged membrane potential (upper traces). The networks are composed of a heterogeneous set of noisy neurons randomly taken out of a temperature range between 6°C and 12°C (upper diagrams) and between 12°C and 18°C (lower diagrams).

3) Impulse Pattern: We are going one step further to neurons with additional ion currents with the special feature that they are already activated in the regular operating range of the neurons. Here we refer to a well-examined model neuron, which originally has been developed to simulate the temperature dependencies of spontaneous cold receptors discharges but in between has become a widely used model as a generic neural pattern generator. The equations and parameter values have repeatedly been described (Braun et al. 2000). Fig. 3 shows the deterministic and noisy bifurcation diagrams of the original cold receptor model. By the way, the most relevant control parameters for temperature scaling are the activation time-constant of the subthreshold currents. Noise is essential to account for a certain activity pattern arising from subthreshold oscillation with random spike generation, which is an often observed pattern also in the central nervous system. It does not appear in purely deterministic simulations. Deterministic simulations, in turn, clearly discover a broad range of deterministic chaos at the transition from single spike activity (tonic firing) to impulse groups (burst discharges).

4) Network synchronization: Transitions from single spike activity to burst discharges are not only carrying information of sensory receptors but also are considered a most relevant feature to facilitate neuronal network synchronisation, e.g. during epileptic seizures, in Parkinson disease, and especially well known, at the transitions from wake to sleep states (Postnova et al. 2010). We illustrate this with simulation examples of network synchronization when the neurons are taken out of the tonic firing and chaotic regime compared with a network which also include bursting neurons. To consider the general randomness of neuronal activity we have added noisy. Additionally, to account for

the physiological heterogeneity of neurons (Tchaptchet 2018), we have randomly taken the neurons out of broader temperature ranges of the bifurcation diagram in Fig. 2. One range spreads from 6 to 12 °C and the other one from 12 to 18°C. The synchronization results in Fig. 4 are obtained with a torus-like networks of 10 to 10 nearest neighbour, gap-junction coupled neurons during increasing coupling strengths. At the beginning the spikes, as indicated in the raster plots, appears at rather random time, due to noise and also because of the heterogeneity of the neurons. With increasing coupling strength the neurons begin to synchronize but only the network with bursting neurons exhibit a clearly structured pattern of compound spike generation. This is also reflected in the middle trace of Fig. 4 representing kind of a field potential in which slow wave oscillations develop, but only when bursting neurons are included.

Discussion

We have been going from single ion channels to ion currents and action potentials up to neuronal network synchronisation and thereby have specifically focused on basic functions in neural coding. This is, first, the sigmoid activation curve, which is the result of an essential contribution of noise while its nonlinear form is again particular susceptible to further stochastic influences in whatever form. The second point concerns the time delays, recognizable in any physiological functions, already at the lowest level of ion channel activation. These characteristics apparently proceed to all higher levels of neural functions up to neural networks.

Conclusion

The network simulations clearly demonstrate that neuronal network synchronization is not only a question of the coupling strength but also of the intrinsic dynamics of the neurons. The neurons in the one network differ from the neurons in the other network mainly by slight differences in the activation time-constants of subthreshold currents. Similar transitions between tonic firing and bursting activity, presumably with similar synchronization effects, can likewise be achieved with alteration of activation curves of other ion channels.

Citation

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Disclosures

Hans A. Braun is co-owner of the *Engineering Office for Biomedical Technologies* (BM&T), producer and distributor of the Virtual Physiology series, which includes the above-mentioned SimNeuron laboratory, programmed by Aubin Tchaptchet.

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