Simulation of Different Firing Patterns in Paired Spider Mechanoreceptor Neurons: The Role of Na⁺ Channel Inactivation

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Torkkeli, Päivi H. and Andrew S. French. Simulation of different firing patterns in paired spider mechanoreceptor neurons: the role of Na⁺ channel inactivation. J Neurophysiol 87: 1363–1368, 2002; 10.1152/jn.00440.2001. The spider VS-3 slit-sense organ contains two types of primary mechanoreceptor neurons that are morphologically similar but have different electrical behavior. Type A neurons fire only one or two action potentials in response to a mechanical or electrical step of any amplitude above the threshold, whereas type B neurons fire prolonged bursts of action potentials in response to similar stimuli. Voltage-clamp studies have shown that two voltage-activated ion currents, a noninactivating potassium current and an inactivating sodium current, dominate the firing behavior. We simulated the electrical behavior of the two neuron types, using a simplified form of Hodgkin-Huxley model based on published voltage-clamp and current-clamp recordings. Changing only two parameters of sodium inactivation controls the firing properties of these neurons and indicate that two parameter changes are needed to achieve complete transformation between the two neuron types.

INTRODUCTION

Primary sensory neurons exhibit a wide range of firing patterns that are matched to their functions. Well-known examples among vertebrate mechanoreceptors are Pacinian corpuscles, which adapt rapidly to a constant stimulus but fire readily with vibration (Loewenstein 1959), and Ruffini endings, which can signal a constant mechanical stimulus by prolonged firing of action potentials (Chambers et al. 1972).

There is some evidence that rapid sensory adaptation is a recent feature of vertebrate mechanoreceptors, coinciding with the evolution of morphologically complex receptors that are connected to ectodermal tissues (Malinovsky 1996). Classical descriptions of mechanoreceptors emphasize the significance of mechanical structures in understanding adaptation behavior, such as the sliding lamellae in Pacinian corpuscles. However, a major role for ionic currents in adaptation has also been known for many years (French and Torkkeli 1994; Mendelson and Loewenstein 1964), and a wide range of adaptation patterns are seen in arthropod cuticular mechanoreceptors, even though the morphological structures are relatively uniform (French 1988). It seems probable that most mechanoreceptive neurons possess such ionic adaptation of action potential discharge, and that it modifies their behavior during sensory perception and their regulation of internal body functions. Activity in other receptor neurons, such as chemoreceptors and temperature receptors, could be similarly affected. Discovery of the mechanisms responsible for this dynamic control of firing behavior could have widespread and crucial importance.

The spider VS-3 mechanoreceptor organ (Barth and Libera 1970) contains seven or eight pairs of primary mechanoreceptor neurons. The two neurons of each pair are morphologically similar by light or electron microscopy but have radically different firing patterns. Type A neurons fire only one or two action potentials in response to a step mechanical or electrical stimulus, while identical stimuli produce prolonged bursts of action potentials for hundreds of milliseconds in type B neurons (Seyfarth and French 1994). We have previously used this preparation to explore the ionic basis of rapid sensory adaptation by comparing the properties of the various ionic currents using voltage-clamp measurements. Of the four major currents identified so far (Sekizawa et al. 1999, 2000; Torkkeli et al. 2001), a noninactivating potassium current and an inactivating sodium current seem to dominate the firing properties.

We have now simulated the firing properties of the two types of VS-3 neurons, using published voltage-clamp and current-clamp data. A simplified form of Hodgkin-Huxley model was used to reduce the number of parameters and facilitate fitting current-clamp data. We were able to simulate the behaviors of the two neuron types by changing only two parameters of sodium channel inactivation, the slope factor of the \( h \) curve and the time constant of recovery from inactivation. This supports our earlier finding that differences in sodium inactivation are primarily responsible for the two types of firing behavior in these mechanosensory neurons (Torkkeli et al. 2001).

METHODS

Intracellular recordings

Responses of spider VS-3 neurons to current steps were taken from a large collection of current-clamp recordings that formed part of a study into nonlinear coding by VS-3 neurons (French et al. 2001). Full details of the experimental methods were given in the earlier study.

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Membrane current simulation

The general approach was based on the Hodgkin-Huxley model (Hodgkin and Huxley 1952) using the exponential Euler method for integrating the differential equations (MacGregor 1987) with a step size of 20 μs. The software was constructed as a C++ class library, similar to the Conical simulation system (Strout 1996) but restricted to a single isopotential spherical cell. All simulations were performed on an IBM-compatible personal computer.

In the present case we had reliable data for the current-clamp step responses and the Boltzmann equations describing the infinite values of the activation and inactivation states, n₃, m₅, and h₆, versus voltage

\[ g/g_{\text{max}} = \frac{1}{1 + e^{\frac{V-V_{\text{th}}}{V_{\text{th}}}}}, \]

where \( g \) is membrane conductance, \( V \) is membrane potential, \( V_{\text{th}} \) is the membrane potential at half-maximal activation or inactivation, and \( s \) is the slope factor. We also had measurements of recovery from sodium inactivation under a wide range of conditions. To simplify the fitting problem we assumed that the time constants of activation, inactivation, and recovery from inactivation could be represented by

\[ \tau = \tau_{\text{max}} \frac{e^{\frac{V-V_{\text{th}}}{V_{\text{th}}}}}{1 + e^{\frac{V-V_{\text{th}}}{V_{\text{th}}}}}, \quad 0 < \delta < 1 \]

where \( \tau \) is a time constant of activation, inactivation, or recovery from inactivation, \( \tau_{\text{max}} \) is the maximum value of \( \tau \), and \( \delta \) is a constant (Johnston and Wu 1995). This reduced the number of kinetic parameters for each gate to four (\( V_{\text{th}}, s, \tau_{\text{max}}, \) and \( \delta \)) and simplified the inclusion of a different time constant of recovery from inactivation in the model, which was achieved by changing the value of \( \tau_{\text{max}} \) between the two values, depending on the direction of movement along the Boltzmann curve during each step.

RESULTS

Passive cell parameters

Values for the passive electrical membrane parameters of the two types of VS-3 neurons (Table 1) were taken from Sekizawa et al. (1999). These data were obtained from a large number of measurements using the same procedure, including crushed dendrites and axons. A cell diameter of 54 μm was chosen to reproduce the mean membrane capacitance values with a specific membrane capacitance of 0.01 F/m². This diameter is close to the value of 52 μm calculated before from passive measurements (Juusola and French 1998) and within the range of large VS-3 neurons observed by light microscopy (Seyfarth et al. 1995). The resulting values of membrane time constant agree well with the experimental data.

### Table 1. Passive cell membrane parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type A Neuron</th>
<th>Type B Neuron</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter, μm</td>
<td>54</td>
<td>54</td>
</tr>
<tr>
<td>Specific membrane resistance, Ωm²</td>
<td>1.68</td>
<td>1.0</td>
</tr>
<tr>
<td>Specific membrane capacitance, F/m²</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Leak equilibrium potential, mV</td>
<td>–75</td>
<td>–75</td>
</tr>
<tr>
<td>Membrane resistance, MΩ</td>
<td>183</td>
<td>109</td>
</tr>
<tr>
<td>Membrane capacitance, pF</td>
<td>91.6</td>
<td>91.6</td>
</tr>
<tr>
<td>Membrane time constant, ms</td>
<td>16.8</td>
<td>10.0</td>
</tr>
</tbody>
</table>

The values of the passive membrane parameters used in the simulations. Membrane resistance, capacitance, and time constant were derived from the other values.

Voltage-activated ion currents

Four types of voltage-activated ion currents have been identified previously in the VS-3 neurons: transient and noninactivating potassium currents (Sekizawa et al. 1999), a low-voltage-activated calcium current (Sekizawa et al. 2000), and an inactivating sodium current (Torkkeli et al. 2001). These studies also showed that elimination of either the transient potassium current or the calcium current did not significantly affect the firing properties of the neurons in response to a wide range of step depolarizations. Therefore they were not included in the present simulations. The firing behaviors of the two neuron types could both be simulated well by models containing only a noninactivating potassium current (Iₖ) and an inactivating sodium current (I₉Na).

Values of the parameters for Iₖ (Table 2) were initially based on the same experimental data as the passive parameters (Sekizawa et al. 1999). The only significant differences between the two neuron types in that study were for the values of the \( V_{\text{th}}, s \) parameters of the activation curve, but even these differences were comparatively small. To simplify, we used identical values for both cell types that were between the two sets of experimental values. The voltage-clamp studies did not determine the time constant of Iₖ activation because the current was fast and could not be completely separated from the transient potassium current. Therefore values of \( \tau_{\text{max}} \) and \( \delta \) were chosen to give a good fit to the experimental action potentials, while ensuring that the simulated currents produced by voltage steps were always in good agreement with the experimental currents produced by similar voltage steps (Sekizawa et al. 1999).

Early in the simulation exercise, a problem arose regarding the maximum conductances of both active currents, because the values suggested by voltage-clamp experiments were too small to explain the experimental current-clamp data. The membranes of both neuron types were always less depolarized at the end of a long positive current pulse than they were hyperpolarized by a negative current pulse (Figs. 1 and 2) the typical effect of a delayed rectifier potassium current. It was necessary to increase the maximum conductance of Iₖ by a factor of about 20 times the published value from voltage-
clamp experiments to simulate the correct rectifying voltage responses to long current steps. This new value was then used throughout for simulation of both neuron types.

The voltage-clamp data for $I_{\text{Na}}$ (Torkkeli et al. 2001) presented even greater problems than for $I_{\text{K}}$ because it had been necessary to reduce the concentration of extracellular sodium from the normal value of 223 to 100 mM to obtain reasonable voltage clamp, and it was impossible to measure the sodium current in the complete absence of outward potassium currents. Nevertheless, the voltage-clamp data were used to provide initial values. The equilibrium potential of the sodium channels was obtained by fitting the voltage-clamp activation and inactivation data (normalized currents) with the Boltzmann equation (Eq. 1) and a linear conductance relationship. Fitting either activation or inactivation data in this way gave a reversal potential of 76 mV, corresponding to an internal Na$^+$ concentration of $\sim$5 mM. Then the Nernst equation was used to obtain a reversal potential of 99 mV for the concentration of sodium in normal spider saline (223 mM).

A major finding from the voltage-clamp study was that recovery from sodium inactivation was slower than development of inactivation in both neuron types and differed significantly between the two neuron types. The simulations included these properties by changing the value of $\tau_{\text{max}}$, depending on whether the inactivation function, $h$, was increasing or decreasing in value, which echoed the experimental methods that were used to measure inactivation and recovery from inactivation (Torkkeli et al. 2001).

In summary, we lacked reliable voltage-clamp data for several parameters of $I_{\text{K}}$ and $I_{\text{Na}}$, but we had many recordings of voltage responses to current injections in the two types of neurons. Therefore we chose to minimize the number of variable parameters in the simulations, to fix those parameters that were most reliable (passive parameters, equilibrium potentials and exponents of $m$ and $h$, and time constants for recovery from sodium inactivation), and vary the other parameters in attempts to fit the voltage responses.

**Simulated voltage responses**

Figures 1 and 2 show typical experimental intracellular voltage recordings from type A and type B neurons during current steps, together with the responses predicted by the simulations whose parameters are given in Tables 1–3. Depolarizing stimulus currents are reported as positive throughout. There was good agreement between the simulated and real responses of both types of neurons, with most of the major features of the firing behavior being reproduced. Type A neurons usually have a threshold for step current injections of 0.1–0.5 nA and fire only one action potential once the depolarization exceeds the threshold, regardless of the current amplitude. They also demonstrate an overshooting afterhyperpolarization and a second, small depolarization that sometimes gives rise to a full action potential (Juusola and French 1998; Seyfarth and French 1994). The simulation used here gave a threshold current amplitude of 0.2 nA and a threshold polarization level of about $-55$ mV (20 mV depolarized from rest), which agrees with experimental data (Sekizawa et al. 1999; Seyfarth and French 1994). The time course of the first action potential, the afterhyperpolarization, and the second depolarization were also well reproduced. The simulation never produced a second full action potential with the parameters used here.

**Table 3. Na$^+$ channel parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type A Neuron</th>
<th>Type B Neuron</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum conductance, nS</td>
<td>400.0</td>
<td>400.0</td>
</tr>
<tr>
<td>Equilibrium potential, mV</td>
<td>99.0</td>
<td>99.0</td>
</tr>
<tr>
<td>Activation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exponent of $m$</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>$V_{\text{h}}$, mV</td>
<td>$-45.0$</td>
<td>$-45.0$</td>
</tr>
<tr>
<td>$s$, mV</td>
<td>5.0</td>
<td>5.0</td>
</tr>
<tr>
<td>$\tau_{\text{max}}$, ms</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>$\delta$</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Inactivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exponent of $h$</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>$V_{\text{h}}$, mV</td>
<td>$-60.0$</td>
<td>$-60.0$</td>
</tr>
<tr>
<td>$s$, mV</td>
<td>5.0</td>
<td>9.0</td>
</tr>
<tr>
<td>$\tau_{\text{max}}$, ms</td>
<td>20.0</td>
<td>20.0</td>
</tr>
<tr>
<td>$\delta$</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Recovery from inactivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\tau_{\text{max}}$, ms</td>
<td>120.0</td>
<td>40.0</td>
</tr>
</tbody>
</table>

Parameters used in Eqs. 1 and 2 for simulation of the sodium current in type A and type B neurons. The only differences between the 2 simulations were in the $s$ parameter and the time constant of recovery from inactivation, $\tau_{\text{max}}$. 

FIG. 1. Type A neuron firing properties. Current-clamp recordings of a type A neuron (left) in response to current steps of $-0.5$, 0.5, and 1.0 nA are shown together with simulated responses (right) using the parameters in Tables 1–3. Type A neurons usually fire only 1 action potential, regardless of the step amplitude. The resting potential in this and all other simulations was $-75$ mV.

FIG. 2. Type B neuron firing properties. Current-clamp recordings of a type B neuron (left) in response to current steps of $-0.5$, 0.5, and 1.0 nA are shown together with simulated responses (right) using the parameters in Tables 1–3. Type B neurons fire prolonged bursts of action potentials when depolarized.
Voltage-clamp data suggested that the major difference between type A and type B neurons was the rate of recovery from sodium inactivation (Torkkeli et al. 2001). However, there was also a significant difference in the inactivation $V_{50}$ parameter between the two cell types and a substantial numerical (but not statistically significant) difference in the inactivation $s$ parameters. After testing the effects of varying these three parameters, we found that the time constant of recovery from inactivation and the inactivation $s$ values had the greatest effect on firing behavior. The simulated type B cells (Fig. 2) were obtained by changing only these two parameters. Activation and inactivation functions of $I_K$ and $I_{Na}$ for both neuron types are shown in Fig. 3, and time constant of recovery from inactivation functions for type A and type B neurons are shown in Fig. 4.

Type B simulations reproduced the repetitive firing properties of the neurons, which contrasts strongly with type A neurons. The decrease in action potential amplitude after the first action potential was also reproduced, but the gradual slowing of action potentials with time in type B neurons did not occur during the period of 100 ms that was used for simulation. The voltage threshold of type B simulations was also about $-55$ mV (20 mV depolarized from rest), which is similar to the threshold difference reported experimentally (Sekizawa et al. 1999), but this required a larger current pulse of 0.26-nA amplitude. Current thresholds for the two neuron types have not been studied systematically, but the lower input resistance (Table 1) would predict that type B neurons require more current to produce a threshold depolarization.

Converting type A to type B neurons

The two models had significant differences in passive parameters (Table 1) as well as the differences in sodium inactivation. However, it was possible to convert the type A model to type B behavior by changing only the two sodium inactivation parameters ($s$ and $\tau_{max}$ for recovery). The separate and combined effects of these two modifications of sodium inactivation are illustrated in Fig. 5. Increasing the $s$ parameter of inactivation, which broadens the $h_s$ and $\tau_i$ functions, caused the model to behave in a mixed mode, with single action potentials above a current threshold of 0.2 nA and bursts of action potentials above a threshold of 0.3 nA. In contrast, decreasing $\tau_{max}$ for recovery from inactivation caused oscillatory behavior to appear at low thresholds but never produced multiple action potentials (Fig. 5). When the two changes in inactivation were combined, robust type B behavior was observed.

Although the simulations in Fig. 5 used type A passive parameters, it was also possible to perform the conversion in the other direction, from type B to type A behavior, using the type B passive parameters (data not shown). This supports the

 FIG. 3. Activation and inactivation parameters for type A and type B neuron simulations. The Hodgkin-Huxley $n$, $m$, $h$, $\tau_i$, $\tau_m$, and $\tau_r$ functions for potassium and sodium (Tables 2 and 3) plotted vs. membrane potential. The same potassium activation (top traces) and sodium activation (bottom traces) functions were used for simulating both type A and type B neurons. The $h_s$ and $\tau_i$ functions for sodium inactivation (bottom traces) are shown for both type A and type B simulations (solid and dashed lines, respectively).

 FIG. 4. Time constant of recovery from sodium inactivation, $\tau_{h,r}$, for type A and B simulations (solid and dashed lines, respectively).

 FIG. 5. Converting a type A neuron to a type B neuron. Simulated responses to a depolarizing current step of 0.5 nA are shown. All simulations used type A passive parameters (Table 1). A type A neuron simulation (left) had the 2 inactivation parameters changed separately (top and bottom middle) and then together (right) to give type B firing properties. Both changes led to repetitive activity, but the detailed firing patterns were quite different. Changing the $h_s$ slope caused prolonged firing but a higher threshold, smaller action potentials and slower firing than a type B neuron. Reducing the time constant of recovery from inactivation produced only oscillations in membrane potential without true action potentials. Combining the 2 changes led to robust type B behavior.
idea that differences in passive parameters are not crucially important in deciding the dynamic behavior of the VS-3 neurons.

Parameter sensitivity

An exhaustive test of the sensitivity of the simulations to all parameters is beyond the scope of the present work, but a simple sensitivity analysis was conducted of the two maximum conductance parameters (Fig. 6), since the simulated values were both larger than observed experimentally. Decreasing the maximum potassium conductance caused excessive steady-state depolarization in both neuron types, causing sodium inactivation and making it difficult to produce long bursts in type B simulations. Increasing potassium conductance reversed this effect but made it more difficult to start a burst in a type B neuron. Changing the maximum sodium conductance had a significant effect on action potential amplitude, particularly in the second and subsequent action potentials of type B bursts.

Discussion

Validity of the simulations

The simulations reproduced the current-clamp voltage responses to step stimuli well. We tried to reduce the number of parameters as much as possible by simplifying the model, but with any complex nonlinear model, it can always be argued that other combinations of parameters might produce equally good, or better simulations. Attempts to reduce the number of parameters further by using simpler exponential functions of voltage for the activation and inactivation time constants were not successful. The use of different time constants for development of inactivation and recovery from inactivation required a method to decide which process was occurring. We used the slope of $h$

![Diagram](image)

**FIG. 6.** Sensitivity analysis for maximum potassium and sodium conductances. Voltage responses to a 100-ns, 1-nA depolarizing pulse are shown for type A and type B neurons with maximum conductance values of 50, 100, and 200% of the parameters used in the normal simulations (Tables 2 and 3).

because this corresponded closely to the methods that were used to measure the time constants and it was easy to add to the simulation. However, we realize that this complicates any attempt to base the simulations on a simple physical model of channel gating. An alternative approach is to change the time constant for value of $h$ above and below 0.5 (Destexhe and Huguenard 2000). Simulations using this method were also able to produce type A and type B behavior, but required a larger difference between the $s$ parameters of the two models. The greatest differences between the simulation parameters and experimental data were in the maximum conductances of $I_K$ and $I_{Na}$, and the sensitivity analysis (Fig. 6) shows that the simulation values could not easily be reduced. However, the values measured by voltage-clamp experiments were already known to be significantly smaller than would be expected from large neurons using sodium-dependent action potentials, where tens, or even hundreds of nanoamps are common (Torkkeli et al. 2001). The peak sodium current that could be obtained in the simulated type A neurons was about 30 nA with steps from −90 mV to about −30 mV. This can be compared with dissociated cockroach dorsal unpaired median (DUM) neurons, which also have somatic action potentials driven by an inactivating sodium current (Grolleau and Lapied 2000). DUM neurons have a similar size (40–60 μm diam) and an initial peak sodium current of −10 nA (Lapied et al. 1990), which grows to −25 nA after 72 h in culture (Tribut et al. 1991).

It is not clear why the voltage-clamp experiments would underestimate the ion conductances so significantly, but single-electrode voltage clamp of voltage-activated currents through high resistance electrodes in large neurons has limitations. The intracellular milieu cannot be changed, and the potassium currents are partially resistant to blocking agents such as tetraethylammonium (TEA) and 4-aminopyridine (4-AP), so it is difficult to block potassium currents and impossible to record sodium currents separately over a range of depolarized potentials. Similar arguments apply to the interference of calcium currents with potassium current measurements. In practice, it was impossible to make reliable recordings above −10 mV, which probably contributed to the low estimates of maximum conductance. Finally, the sodium current measurements could only be made with reduced extracellular sodium concentration.

The activation and inactivation parameters used in the simulation agree well with the data from DUM neurons (Lapied et al. 1990). The $m_s$ functions used in the simulations (Fig. 3) had similar properties to that in DUM neurons, and the DUM neuron $h_m$ curve was closer to the $h_n$ curve used for type B rather than type A simulations (Fig. 4), which is interesting because DUM neurons can also fire continuous trains of action potentials (Grolleau and Lapied 2000).

Basis of rapid sensory adaptation

Mechanoreceptive neurons are widely distributed throughout the bodies of both vertebrates and invertebrates. In addition to their well-known functions in sensory perception, mechanoreceptors are crucial to the regulation of many internal organ systems, so control of their action potential firing properties is an important component of normal physiological function and a potential site for pharmacological intervention. It has been known for many years that membrane currents make a major contribution to rapid adaptation of action potential discharge in...
mechanoreceptors, but experimental difficulties have limited investigation of the ionic basis of adaptation to a small number of preparations.

In the cockroach tactile spine neuron, blockade of a calcium-activated potassium current markedly reduced the adaptation (Torkkeli and French 1995), although there was also evidence for a slow component of sodium inactivation (French and Torkkeli 1994). The spider VS-3 neurons both have significant calcium currents (Sekizawa et al. 2000), but we could find no evidence for calcium-activated potassium currents in the somata of these neurons. We found no role for calcium currents in the firing behavior previously (Sekizawa et al. 2000), and the simulations also worked well without including a calcium component. In crayfish stretch receptor neurons, a differential distribution of sodium channels between rapidly and slowly adapting neurons was suggested to cause differences in both adaptation rate and action potential size (Lin and Rydqvist 1999). However, there is no evidence for differences in sodium channel distribution or action potential amplitude in the VS-3 neurons (Torkkeli et al. 2001).

The previous voltage-clamp studies found that the most prominent differences between type A and type B neurons were in the inactivation properties of their sodium channels, particularly recovery from inactivation. Different time courses of recovery from sodium inactivation were also found in two populations of sodium channels in mammalian sensory neurons (Elliott and Elliott 1993), and the faster time course of recovery caused by a shift in the relative proportions of these sodium channel types has been linked to increased excitability (Cummins and Waxman 1997). The present results echo this suggestion, with the decrease in time constant of recovery from inactivation in type B neurons being associated with decreased voltage threshold and repetitive firing. We expect that future work will explore the basis of the different sodium current properties in these two types of sensory neurons.

S. Sekizawa made the sample current-clamp recordings.

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REFERENCES


