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A general framework for neurobiological modeling: an application to the vestibular system ☆

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Abstract

The otolith organs in the vestibular system are excellent detectors of linear accelerations. However, any measurement of linear acceleration is ambiguous between a tilt in a gravitational field and an inertial acceleration. Angelaki et al. have put forward a general hypothesis about how inertial accelerations can be computed based on vestibular signals (J. Neurosci. 19 (1999) 316). We have constructed a realistic, detailed model of the relevant systems to test this hypothesis. The model produces useful predictions about what kinds of neurons should be found in the vestibular nucleus if such a computation is actually performed in the vestibular system. The model is constructed using general principles of neurobiological simulation (J. Neurophys. 84 (2000) 2113). © 2002 Published by Elsevier Science B.V.

Keywords: Vestibular system; Neural representation; Large-scale models

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1. Introduction

Given any accelerometer, it is not possible to distinguish inertial acceleration due to translation from gravitational acceleration during tilting. In the mammalian vestibular system, the otoliths serve as an accelerometer. Thus, the otolith signals are ambiguous between these two kinds of acceleration. However, animals can successfully distinguish

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such motions. Precisely, how this distinction is made is a matter of some debate in contemporary neuroscience [2,3,6].

In a series of experiments on macaque monkeys, Angelaki et al. show that inactivation of the semicircular canals results in precisely the kinds of eye movement errors we would expect if only the otolith were used to distinguish inertial from tilt accelerations [2]. Similarly, Merfeld et al. show that reflexive eye movements in humans are most consistent with a combination of canal and otolith information [6]. This kind of evidence strongly supports the contention that there is an integration of sensory signals from both the otolith and semicircular canals used to determine appropriate motor responses (see also [7,8]). Angelaki et al. have suggested a series of transformations that could be used to determine an unambiguous inertial acceleration given the otolith and semicircular canal signals [2]. In this paper, we present a detailed model that implements these transformations using our framework for constructing realistic neurobiological models [5,4,1].

2. A quantitative hypothesis about vestibular function

Angelaki et al. suggest that the following computation is implemented in the brain to determine inertial acceleration, $\mathbf{I}(t)$, provided the otolith signal $\mathbf{A}(t)$ and the canal signal $\mathbf{\Omega}(t)$ [2]:

$$\dot{\mathbf{I}}(t) = \mathbf{\Omega}(t) \times \mathbf{I}(t) + \dot{\mathbf{A}}(t) - \mathbf{\Omega}(t) \times \mathbf{A}(t) \tag{1}$$

$$= \dot{\mathbf{A}}(t) + \mathbf{\Omega}(t) \times [\mathbf{A}(t) - \mathbf{I}(t)]. \tag{2}$$

The well-established assumptions underlying this hypothesis are: (1) that the three semicircular canals generally operate as orthogonal integrating accelerometers which together provide a good estimate of angular velocity, $\mathbf{\Omega}(t)$; and (2) that the two otolith organs encode accelerations in the horizontal and vertical planes, providing a good estimate of linear acceleration $\mathbf{A}(t)$ [8]. In order to characterize the dynamics for a neural population that represents this inertial acceleration, we use a first-order Taylor expansion:

$$\mathbf{I}(t+\tau) \approx \mathbf{I}(t) + \tau\dot{\mathbf{I}}(t), \tag{3}$$

$$= \mathbf{I}(t) + \tau[\dot{\mathbf{A}}(t) + \mathbf{\Omega}(t) \times [\mathbf{A}(t) - \mathbf{I}(t)]]. \tag{4}$$

Eqs. (2) and (4), along with neurophysiological considerations suggest a neural circuit as that shown in Fig. 1. This diagram provides a particular modularization of the computation needed to determine the true inertial acceleration.

We think that this modularization is a reasonable one given that there are strong convergent projections of both the otolith and canal systems within the vestibular nucleus [8, p. 166]. In our model, we use leaky integrate-and-fire neurons tuned to mimic the properties of neurons found in the labyrinth and relevant parts of the vestibular nucleus [8].[1]

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[1] We can replace the LIF neurons with more complex single cell models. But, the computational cost for this increase in detail is unacceptable in this case.

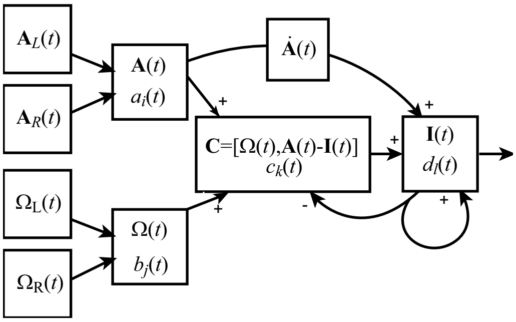

Fig. 1. A high-level modularization of a vestibular circuit that can compute inertial acceleration from the labyrinth inputs.

3. The model and results

In order to construct a model of this system, we embed each of the higher-level representations and transformations identified in Fig. 1 into the relevant neural populations. The general principles behind this approach are articulated in [1]. The representations and transformations up to the vestibular nucleus are relatively simple, so we will focus instead on the representation and transformations of the vestibular neurons that perform the cross-product, a highly non-linear operation.

We take the first population of neurons involved in the cross-product transformation, $c_k(t)$, to construct an integrated representation of the input space, $\mathbf{C}$, by encoding $\boldsymbol{\Omega}(t)$ and $\mathbf{A}(t) - \mathbf{I}(t)$ simultaneously using a six-dimensional encoding vector, $\tilde{\phi}^{\mathbf{C}}$:

$$\begin{aligned} c_k(\mathbf{C} = [\boldsymbol{\Omega}, \mathbf{A} - \mathbf{I}]) &= G_k[\alpha_k \langle \tilde{\phi}_k^{\mathbf{C}} \mathbf{C} \rangle + J_k^b] \\ &= G_k[\alpha_k \langle \tilde{\phi}_k^{\boldsymbol{\Omega}} \boldsymbol{\Omega} + \tilde{\phi}_k^{\Delta} (\mathbf{A} - \mathbf{I}) \rangle + J_k^b] \\ &= G_k \left[\sum_k \omega_{kj} b_j + \sum_i \omega_{ki} a_i + \sum_l \omega_{kl} d_l + J_k^b \right], \end{aligned}$$

where $\omega_{kj} = \alpha_k \langle \tilde{\phi}_k^{\boldsymbol{\Omega}} \phi_j^{\boldsymbol{\Omega}} \rangle$, $\omega_{ki} = \alpha_k \langle \tilde{\phi}_k^{\Delta} \phi_i^{\mathbf{A}} \rangle$, and $\omega_{kl} = -\alpha_k \langle \tilde{\phi}_k^{\Delta} \phi_l^{\mathbf{I}} \rangle$.

In order to use this encoding to compute the cross-product of elements in the $\mathbf{C}$ space, we need to determine a decoding for the function $P(\mathbf{C})$, which finds the cross-product of the first and last three elements of any vector $\mathbf{C}$. This decoding will be $P(\mathbf{C}) = \sum_k \phi_k^P c_k$. We find the decoding vectors by solving the appropriate least-squares minimization problem.

This decoding can be used in a description of the dynamics of the subsequent population. Eq. (3) gives an expression for neurons encoding the inertial acceleration: $d_l(t + \tau) = G_l[\alpha_l \langle \tilde{\phi}_l^{\mathbf{I}} \cdot \mathbf{I}(t + \tau) \rangle + J_l^b]$.

Substituting from (4):

$$\begin{aligned} d_l(t+\tau) &= G_l[\alpha_l\langle \tilde{\phi}_l^{\mathbf{I}}(\mathbf{I}(t) + \tau[\dot{\mathbf{A}}(t) + \mathbf{\Omega}(t) \times (\mathbf{A}(t) - \mathbf{I}(t))])\rangle + J_l^b] \\ &= G_l\left[\sum_{l'} \omega_{ll'} d_{l'}(t) + \tau \sum_k \omega_{lk} c_k(t) + \tau \sum_i \omega_{li} a_i(t) + J_l^b\right], \end{aligned}$$

where $\omega_{ll'} = \alpha_l\langle \tilde{\phi}_l^{\mathbf{I}} \phi_{l'}^{\mathbf{I}}\rangle$, and $\omega_{lk} = \alpha_l\langle \tilde{\phi}_l^{\mathbf{I}} \phi_k^{P}\rangle$. $\dot{\mathbf{A}}(t)$ requires separate (though similar) treatment. However, we do not discuss it here.

In order to model these populations with spiking neurons, we use a linear decoding filter on the spike trains to give neuron activity variables, $a_i(t)$, $b_j(t)$, etc. Thus, for each population we write an expression of the form: $a_i(t) = \sum_n h_i(t) * \delta(t - t_n) = \sum_n h_i(t - t_n)$, where the linear filters, $h_i(t)$ are taken to be the postsynaptic currents produced in the postsynaptic neuron given the incoming spike train, $\delta(t - t_n)$. The $h_i(t)$ responses are appropriately weighted by the relevant weights, ω, determined as above.

We can now test our model by comparing it to experimental results from macaques.[2] In order to show that the vestibular system is able to estimate true inertial acceleration, despite the ambiguity in the otolith signal, the following three experiments were performed. First, the animal was subjected only to translational acceleration. Second, the animal was subjected to a translational acceleration coupled with an angular rotation chosen to double the otolith signal. Third, the animal was subjected to a translation acceleration coupled with an angular rotation chosen to cancel the otolith signal. Any estimate of translational acceleration based solely on otolith information would be severely adversely affected in the last two of these cases. Nevertheless, eye position changes resulting from the translational vestibular ocular reflex (tVOR) are very similar. This suggests that the otolith signal is not solely responsible for the tVOR.

Fig. 2 shows the results of the model compared to both a direct solution of Eq. (2) and the data, for the case when the otolith signal is canceled. Similar agreement between experimental and model results was obtained for the other conditions. Thus, the model verifies that the hypothesis is a reasonable one, and that it can be implemented in a biologically plausible, spiking network.

4. Conclusion

One of the reasons for constructing this model is to determine what the tuning properties of certain populations in the model are expected to look like. Of particular interest are those neurons performing the cross product. On the basis of this model, we expect these neurons to have preferred direction vectors in a six-dimensional space, which are sensitive to both angular velocity and linear acceleration. However, this expectation is based mainly on our chosen means of implementing the equation. It is possible, for instance, to perform the cross product as a series of multiplications

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[2] Thanks are due to Dora Angelaki for providing the experimental data.

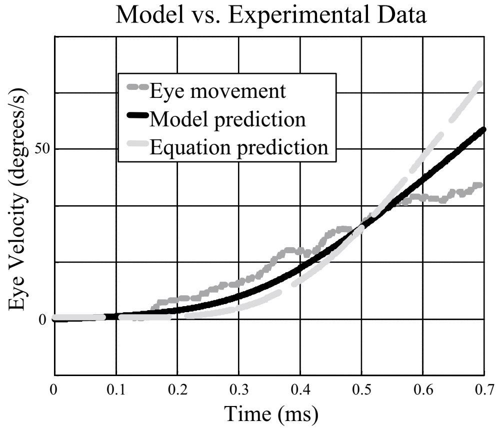

Fig. 2. Experimental data, equation solution, and model solution. The model consists of a total of 5000 LIF neurons. Comparisons incorporate a gain reduction which has been experimentally verified for tVOR, and ranges between about 0.1 and 0.25.

between components of these vectors. In this case, neuron preferred direction vectors would lie in two-dimensional spaces. Nevertheless, however we choose to implement the cross-product, there must be neurons whose preferred direction vectors *mix* the otolith and vestibular signals. Furthermore, given the fact that there is a non-linear transformation being performed, we expect neurons in this population to have fairly low background firing rates and more often than not require significant input to begin firing. This is due to the fact that for non-linear transformations, a greater response range is required as we move away from the resting state compared to linear transformations.

In addition, the dynamics of the network provide predictions regarding the kinds of receptors present on neurons in the various populations. Although we have not discussed the dynamics in detail here, based on this model we expect the neurons participating in the **I** population to have recurrent connections to NMDA-type receptors because the recurrence requires slow synaptic decays to function well. The remaining connections can be fast, AMPA-type receptors. This suggests that some cells in the **I** population will have both kinds of receptors on the same cell. Notably, this is consistent with the general properties of neurons in the vestibular nucleus [9].

References

[1] D.E. Angelaki, J.D. Dickman, Spatiotemporal processing of linear acceleration : primary afferent and central vestibular neuron responses, J. Neurophys. 84 (2000) 2113–2132.

[2] D.E. Angelaki, M.Q. McHenry, J.D. Dickman, S.D. Newlands, B.J.M. Hess, Computation of inertial motion: neural strategies to resolve ambiguous otolith information, J. Neurosci. 19 (1999) 316–327.

[3] L.W. Chen, K.K.L. Yung, Y.S. Chan, Co-localization of NMDA receptors and AMPA receptors in neurons, Brain Res. 884 (2000) 87–97.

[4] C. Eliasmith, C.H. Anderson, Developing and applying a toolkit from a general neurocomputational framework, Neurocomputing 26 (1999) 1013–1018.

[5] C. Eliasmith, C.H. Anderson, Neurobiological simulation: principles and methods, Neurocomputing 38 (2001) 581–586.

[6] C. Eliasmith, C.H. Anderson, Neural Engineering: The Principles of Neurobiological Simulation, MIT Press, Cambridge, in press.

[7] D.M. Merfeld, L. Zupan, R.J. Peterka, Humans use internal models to estimate gravity and linear acceleration, Nature 398 (1999) 615–618.

[8] L. Telford, S.H. Seidman, G.D. Paige, Dynamics of squirrel monkey linear vestibuloocular reflex and interactions with fixation distance, J. Neurophys. 78 (1997) 1775–1790.

[9] V.J. Wilson, G.M. Jones, Mammalian Vestibular Physiology, Plenum Press, New York, 1979.