Published in final edited form as:

Neurocomputing. 2007 June 1; 70(10-12): 1954–1959. doi:10.1016/j.neucom.2006.10.078.

Resonance tuning of a neuromechanical system with two negative sensory feedback configurations

Carrie A. Williams and Stephen P. DeWeerth

Wallace H. Coulter Department of Biomedical Engineering, Georgia Institute of Technology, Atlanta, GA, USA

Abstract

Resonance tuning in a model of rhythmic movement is compared when the central pattern generator (CPG) consists of two endogenously bursting or two tonically spiking neurons that are connected with reciprocally inhibitory synapses. The CPG receives inhibitory and/or excitatory position feedback from a linear, one-degree-of-freedom mechanical subsystem. As with previously published results [5,15], resonance tuning is limited to frequencies that are greater than the intrinsic CPG frequency with endogenously bursting neurons. In contrast, with tonically spiking neurons, the resonance tuning range is expanded to frequencies that are below the intrinsic CPG frequency.

Keywords

resonance tuning; central pattern generator; sensory feedback; intrinsic membrane dynamics

1. Introduction

The daily activities of many animals are comprised of rhythmic movements, such as walking, swimming, and flying. Intrinsically active neural networks called central pattern generators (CPGs) produce the rhythmic patterns of muscle activation that are necessary for these movements [10]. Even though CPGs have an associated intrinsic frequency, several studies have shown that the preferred frequency of walking in humans [1,7,8] and swimming in jellyfish and scallops [11] is approximately equal to the resonant frequency of the mechanical dynamics. These experimental results suggest that *resonance tuning* occurs, whereby both the mechanical dynamics and the underlying CPG synchronize to the mechanical resonant frequency. Resonance tuning is advantageous in the control of rhythmic movement because it minimizes the energetic cost while maximizing stability [4,6].

Previous resonance tuning studies have shown that sensory feedback is essential to adjust the frequency of the CPG to the mechanical resonant frequency [5,15]. Biological systems have evolved with several strategies to synchronize the mechanical and neural dynamics using sensory feedback. For example, the lamprey swim CPG receives both an inhibitory and an excitatory negative sensory feedback input [14]. Several computational studies have shown that when either the van der Pol or the Matsuoka nonlinear oscillators receive position feedback in the negative, inhibitory configuration, resonance tuning is limited to frequencies that are greater than the intrinsic CPG frequency [5,15].

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

This frequency limit for resonance tuning was determined when the CPGs were configured as half-center oscillators. This configuration is one of the simplest CPGs, consisting of two neurons or groups of neurons that are connected with reciprocally inhibitory synapses. The neurons fire in alternating bursts of activity, with one neuron controlling each phase of movement. There are several intrinsic network properties that can give rise to the characteristic antiphasic bursting of a half-center oscillator [3]. For example, when the excitatory amino acid *N*-methyl-D-aspartate (NMDA) is used to activate the half-center oscillator in the lamprey swim CPG, the individual neurons endogenously burst. Thus, with NMDA activation, the reciprocally inhibitory synapses are only necessary for proper phasing. We will refer to this type of half-center activity as endogenous bursting. Conversely, with kainate/ α -amino-3-hydroxy-5-methyl-4-isoxalone (AMPA) activation, the individual neurons tonically spike, and the antiphasic bursting arises from a combination of synaptic and intrinsic properties [2,12]. We will refer to this type of CPG activity as synaptic bursting.

We hypothesize that the intrinsic membrane dynamics of the CPG affect the ability of a given sensory feedback configuration to tune a closed-loop system to the mechanical resonant frequency. To test this hypothesis, we constructed a computational model of rhythmic movement with CPG and sensory feedback dynamics as found in the lamprey swim CPG. We then compared the resonance tuning of a closed-loop system with synaptic versus endogenous CPG bursting by varying the simulated activation of kainate/AMPA or NMDA receptors. This comparison was repeated when the inhibitory and excitatory negative feedback configurations were individually and concomitantly implemented.

2. Computational Models

To study the effects of intrinsic CPG properties on resonance tuning, we used a computational model of rhythmic movement with neural and feedback dynamics similar to those in the lamprey swim CPG. The lamprey CPG was chosen because the membrane dynamics are well defined and have been shown to produce half-center oscillator activity with two distinct mechanisms that we define as endogenous and synaptic bursting. By then coupling the CPG to a simple, one-degree-of-freedom mechanical subsystem, the effects of the CPG dynamics on resonance tuning are emphasized. Fig. 1 shows a diagram of the CPG and the mechanical subsystem along with the feedforward and feedback couplings.

2.1. CPG

Our model CPG is a half-center oscillator, consisting of two neurons with reciprocally inhibitory synapses. The neurons are modeled with Hodgkin-Huxley dynamics as previously used to describe the interneurons in the lamprey swim CPG. Each neuron consists of five compartments: an initial segment, a soma, and three dendritic compartments. The model equations and parameters are taken directly from Kozlov et al. [9] and are reproduced in the Appendix.

Activating the CPG with simulated bath application of the excitatory amino acids kainate/AMPA or NMDA results in synaptic or endogenous bursting, respectively. Exclusive activation of kainate/AMPA receptors provides a generalized increased excitability, and as a result, the individual neurons tonically spike. When the two neurons are coupled with reciprocally inhibitory synapses, the synaptic dynamics along with summation of postspike afterhyperpolarizations cause the neurons to fire in alternating bursts of activity. Therefore, with kainate/AMPA activation, the reciprocally inhibitory synapses are essential for burst generation. This type of CPG activity is referred to as synaptic bursting. In contrast, because of the slow NMDA receptor dynamics, sufficient NMDA activation causes the individual neurons to endogenously burst. Consequently, when the neurons are connected in a half-center oscillator configuration, the reciprocally inhibitory synapses are mainly responsible for anti-

synchronous phasing rather than burst generation. This type of CPG activity is, therefore, referred to as endogenous bursting. The open-loop activity of the CPG with both types of bursting is illustrated in Fig. 2.

We compared resonance tuning with endogenous and synaptic bursting by activating the CPG with exclusively kainate/AMPA or NMDA. As with previous studies of this model, the CPG has a higher intrinsic frequency with kainate/AMPA than with NMDA activation. Different activation parameters and, consequently, different intrinsic frequencies show similar resonance tuning trends to those shown here; with the chosen values, however, the closed-loop system oscillates over similar ranges of resonant frequencies with both kainate/AMPA and NMDA activation.

2.2. Mechanical Subsystem

In the closed-loop system shown in Fig. 1, the output of the CPG is the input to the mechanical subsystem. The mechanical subsystem was modeled as a one-degree-of freedom system, such as a joint, with the dynamics of a mass-spring-damper system that are governed by

$$\frac{X(s)}{F(s)} = \frac{1/m}{s^2 + \frac{\omega_r}{Q}s + \omega_r^2} \quad (1)$$

where m is the mass, X is the position, F is the output of the CPG, Q is the quality factor, and ω_r is the *undamped mechanical resonant frequency*. For all simulations, $m = 10$ kg, $Q = 8$, and ω_r was varied from 0.2 to 5.7 Hz in increments of 0.5 Hz.

2.3. Sensory Feedback Connections

The position of the mechanical subsystem was used to calculate the feedback signals. As found in the lamprey swim CPG [14], there are two possible negative feedback configurations that were individually or concomitantly implemented: ipsilateral inhibition (II) and contralateral excitation (CE). These feedback connections are illustrated in Fig. 1. As shown, N_1 drives the mechanics in the positive direction, and it receives inhibitory feedback from positive position (x_1) and/or excitatory feedback from negative position (x_2). Since N_2 drives the mechanics in the opposite direction, the feedback connections to this neuron are reversed. The inhibitory and excitatory feedback currents were calculated by

$$I_{1,2} = g_{syn} \tanh(S x_{1,2})(V - E) \quad (2)$$

where g_{syn} is the maximal conductance, $x_1 = \max(x, 0)$, $x_2 = -\min(x, 0)$, $S = 5$ is the slope of the activation function, V is the voltage of the postsynaptic cell, and E is the reversal potential. For inhibitory synapses $E = -85$ mV and for excitatory synapses $E = 0$ mV. When the II and CE connections were individually implemented, $g_{syn} = 0.02 \mu\text{S}$ and when combined, $g_{syn} = 0.01 \mu\text{S}$.

2.4. Simulations

Simulations of the closed-loop system were run in MATLAB/Simulink (Mathworks, VA) for 40.5s with a variable-step stiff solver. The closed-loop frequency of the outputs from both the CPG and the mechanical subsystem (F and x) were determined by averaging the last four consecutive cycles.

3. Results

Previous resonance tuning studies with position feedback have shown that the closed-loop system only tunes to resonant frequencies that are above the intrinsic frequency of the CPG (ω_{CPG}). To determine if the underlying CPG dynamics affect this limitation, we compared the resonance tuning of a closed-loop system with endogenous versus synaptic CPG bursting. Results are shown in Figs. 3 and 4 when the inhibitory and excitatory feedback configurations were individually and symmetrically implemented.

3.1. Endogenous Bursting

With NMDA activation, resonance tuning is limited to frequencies that are above ω_{CPG} (Fig. 3). This is similar to the previously published results in which either the van der Pol or the Matsuoka oscillators were used as CPGs [5,15]. Fig. 3 also illustrates that the CPG and the mechanical subsystem do not always converge to the same frequency. For higher resonant frequencies, the mechanical subsystem oscillates at the mechanical resonant frequency and the CPG oscillates at its intrinsic frequency. There is a large deviation in the cycle-to-cycle amplitude of x when this disparity occurs. By increasing the feedback conductance, the CPG and mechanical dynamics converge to a larger range of resonant frequencies. Even with large increases in the feedback conductance, the system will not tune to resonant frequencies that are below ω_{CPG} .

Resonance tuning is also affected by the connectivity of sensory feedback. With NMDA activation, the closed-loop system tunes to the greatest range of resonant frequencies with the CE connectivity and only tunes to a subset of these frequencies with the II connectivity (Fig. 3). Combining the two feedback configurations changes the maximum resonant frequency to which the system will tune, while maintaining the same overall conductance.

3.2. Synaptic Bursting

In contrast to NMDA activation, Fig. 4 shows that with exclusively kainate/AMPA activation, the closed-loop system tunes to resonant frequencies that are both above and below ω_{CPG} . The CPG and the mechanical subsystem also synchronize to a greater range of resonant frequencies with kainate/AMPA activation. Therefore, the same resonance tuning range can be achieved with smaller feedback conductances when the CPG is activated with kainate/AMPA versus NMDA. This is consistent with other studies that have found a decreased range of neural entrainment with increased amounts of NMDA activation [12].

The connectivity of sensory feedback also dramatically affects resonance tuning with kainate/AMPA activation. The closed-loop system tunes to a larger range of frequencies that are below ω_{CPG} with the II connectivity. Although only the symmetric weighting of feedback is shown in Fig. 4, by more heavily weighting one type of connectivity, the tuning curves can be shifted between the CE and II trends. In addition, increasing the feedback conductance will extend the resonance tuning range both above and below ω_{CPG} .

4. Summary

Using a biologically inspired closed-loop system, we have shown that the intrinsic properties of the CPG modulate the range of resonant frequencies to which a closed-loop system tunes. When the CPG consists of intrinsically bursting neurons (NMDA activation), resonance tuning is limited to frequencies that are above ω_{CPG} . This limit is true either when the II and CE feedback configurations are individually or concomitantly implemented. Conversely, when the isolated CPG neurons tonically spike (kainate/AMPA activation), resonance tuning occurs both

above and below ω_{CPG} . In this case, the propensity of the closed-loop system to tune to resonant frequencies that are below ω_{CPG} is greater with the II than with the CE feedback connectivity.

Although resonance tuning both above and below ω_{CPG} has previously been shown with proportional-integral-derivative (PID) feedback, our results are the first to show that the CPG dynamics can also be used to expand the resonance tuning range below ω_{CPG} . Our results emphasize that the intrinsic dynamics of the CPG are as important as the type of feedback in determining the closed-loop frequency and that explicitly biological dynamics can be used to modify the resonance tuning properties.

Overall, our results with Hodgkin-Huxley neural dynamics further indicate that resonance tuning is a general property of nonlinear oscillators that are coupled to a resonant mechanical subsystem, as first suggested by Hatsopoulos [5]. However, we have shown that the specific tuning characteristics depend on the dynamics of the oscillator and the connectivity of sensory feedback. The inclusion of biological CPG and sensory feedback dynamics would, therefore, improve current robotic designs by increasing the control and flexibility of resonance tuning.

Acknowledgments

This work was supported by a Whitaker graduate fellowship to CAW and by NIH NS43098 to SDW

References

1. Abe MO, Yamada N. Modulation of elbow joint stiffness in a vertical plane during cyclic movement at lower or higher frequencies than natural frequency. *Exp Brain Res* 2003;153:394–399. [PubMed: 14513302]
2. Brodin L, Traven HGC, Lansner A, Wallen P, Ekeberg O, Grillner S. Computer simulations of *N*-methyl-D-aspartate receptor-induced membrane properties in a neuron model. *J Neurophysiol* 1991;66:473–484. [PubMed: 1723094]
3. Friesen WO. Reciprocal inhibition: a mechanism underlying oscillatory animal movements. *Neurosci Biobehav Rev* 1994;18:547–553. [PubMed: 7708368]
4. Goodman L, Riley MA, Mitra S, Turvey MT. Advantages of rhythmic movements at resonance: minimal active degrees of freedom, minimal noise, and maximal predictability. *J Motor Behav* 2000;32:2–8.
5. Hatsopoulos NG. Coupling the neural and physical dynamics in rhythmic movements. *Neural Comp* 1996;8:567–581.
6. Holt KG, Jeng S-F, Ratcliffe R. Energetic cost and stability during human walking at the preferred stride frequency. *J Motor Behav* 1995;27:164–178.
7. Holt KG, Hamill J, Andres RO. The force-driven harmonic oscillator as a model for human locomotion. *Hum Mov Sci* 1990;9:55–68.
8. Jeng S-F, Liao H-F, Lai J-S, Hou J-W. Optimization of walking in children. *Med Sci Sports Exerc* 1997;29:370–376. [PubMed: 9139176]
9. Kozlov AK, Ullen F, Fagerstedt P, Aurell E, Lansner A, Grillner S. Mechanisms for lateral turns in lamprey in response to descending unilateral commands: a modeling study. *Biol Cybern* 2002;86:1–14. [PubMed: 11918208]
10. Marder E, Bucher D. Central pattern generators and the control of rhythmic movements. *Curr Biol* 2001;11:R986–996. [PubMed: 11728329]
11. Pabst DA. Springs in swimming animals. *Amer Zool* 1996;36:723–735.
12. Traven HGC, Brodin L, Lansner A, Ekeberg O, Wallen P, Grillner S. Computer simulations of NMDA and non-NMDA receptor-mediated synaptic drive: sensory and supraspinal modulation of neurons and small networks. *J Neurophysiol* 1993;70:695–709. [PubMed: 8105036]
13. Verdaasdonk BW, Koopman HFJM, Van Der Helm FCT. Energy efficient and robust rhythmic limb movement by central pattern generators. *Neural Networks* 2006;19:388–400. [PubMed: 16352419]

14. Viana Di Prisco G, Wallen P, Griller S. Synaptic effects of intraspinal stretch receptor neurons mediating movement-related feedback during locomotion. *Brain Res* 1990;530:161–166. [PubMed: 1980227]
15. Williamson M. Neural control of rhythmic arm movement. *Neural Networks* 1998;11:1379–1394. [PubMed: 12662756]

Appendix

Appendix

The model neuron consists of an initial segment, a soma, as well as proximal, medial, and distal dendritic compartments. The segmental voltages are given by

$$CV'_{\text{soma}} = I_{\text{leak}} + I_{\text{Na}} + I_{\text{K}} + I_{\text{KCa}} + I_{\text{AMPA}}^{\text{bath}} + I_{\text{NMDA}}^{\text{bath}} + \sum_j I_a^j \quad (\text{A1})$$

$$CV'_{\text{initial segment}} = I_{\text{leak}} + I_{\text{Na}} + I_{\text{K}} + I_{\text{AMPA}}^{\text{bath}} + \sum_j I_a^j \quad (\text{A2})$$

$$CV'_{\text{dendrite}} = I_{\text{leak}} + I_{\text{AMPA}}^{\text{bath}} + \sum_j I_a^j + \sum_{\text{syn}} I_{\text{syn}} \quad (\text{A3})$$

where V is the membrane potential, C is the membrane capacitance, I_{leak} is the leak current, I_{Na} is the sodium current, I_{K} is the fast potassium current, I_{KCa} is the calcium-dependent potassium current, $I_{\text{AMPA}}^{\text{bath}}$ is the bath-activated AMPA current, $I_{\text{NMDA}}^{\text{bath}}$ is the bath-activated NMDA current, I_a^j are the axial currents from neighboring compartments, and I_{syn} are the synaptic currents. Only the proximal dendritic compartment contains the synaptic currents. The membrane capacitance (F) is equal to 0.01 multiplied by the membrane area (m^2), where the areas of the soma, dendritic compartments, and initial segment are 0.006, 0.024, and 0.0006 mm^2 .

Each ionic current is described by

$$I = \bar{g} m^{k_1} h^{k_2} (V - E_{\text{rev}}) \quad (\text{A4})$$

where $\bar{g}$ is the maximal conductance, E_{rev} is the reversal potential, m and h indicate activation and inactivation of the channels, and k_1 and k_2 are the exponents determining the shape of the (in)activation dynamics. E_{rev} and the specific conductance are given in Table A1. $\bar{g}$ is calculated by multiplying the specific conductance by the area of the compartment. The level of kainate/AMPA and NMDA activation are given by k_a and k_n . The gate variables m and h are described by

$$\dot{x} = \alpha_x(V)(1 - x) - \beta_x(V)x \quad (\text{A5})$$

where α_x and β_x are the opening and closing rate coefficients. These coefficients are described by one of three equations,

$$y_1(V) = A \exp\left(\frac{V - V_0}{B}\right) \quad (\text{A6})$$

$$y_2(V) = A \left(\exp\left(\frac{V - V_0}{B}\right) + 1 \right)^{-1} \quad (\text{A7})$$

$$y_3(V) = A(V - V_0) \left(\exp\left(\frac{V - V_0}{B}\right) - 1 \right)^{-1} \quad (\text{A8})$$

The specific equation and the values for the constants A , B , and V_0 that were used for each current are indicated in Table A1. I_{KCa} depends on the concentration of calcium ($[\text{Ca}]$) that enters after an action potential (I_{CaAP}) and from the activation of NMDA receptors (I_{CaNMDA}). The rate change of calcium is given by

$$[\text{Ca}]' = B_{\text{Ca}} I_{\text{Ca}} - \frac{[\text{Ca}]}{\tau_{\text{Ca}}} \quad (\text{A9})$$

where τ_{Ca} is the calcium decay rate and B_{Ca} is the calcium influx rate. The values for these rate constants depend on the calcium current: for I_{CaAP} , $\frac{B_{\text{Ca}}}{\text{area}} = 0.67 \text{ ms}^{-1} \text{ mV}^{-1} \text{ mm}^{-2}$ and $\tau_{\text{Ca}} = 0.05$ s; for I_{CaNMDA} , $\frac{B_{\text{Ca}}}{\text{area}} = (0.001107 * 75 * \text{kn}) \text{ ms}^{-1} \text{ mV}^{-1} \text{ mm}^{-2}$ and $\tau_{\text{Ca}} = 0.5$ s. The axial currents are given by

$$I_a^{(j)} = \bar{g}_a (V - V^{(j)}) \quad (\text{A10})$$

where $V^{(j)}$ are the voltages of the neighboring compartments. The reciprocally inhibitory synapses are described by

$$I_{\text{syn}} = \bar{g}_{\text{syn}} s(t) (V - E_{\text{syn}}) \quad (\text{A11})$$

with the synaptic activation $s(t)$ modeled by

$$s' = \begin{cases} \rho(1 - s), & 0 < t - (t_{\text{spike}} + \tau_{\text{del}}) < T_{\text{dur}} \\ -\sigma s, & \text{otherwise} \end{cases} \quad (\text{A12})$$

where $\rho = 0.1$ ms and $\sigma = 20$ ms are growth and decay time constants, $T_{\text{dur}} = 0.5$ ms is the duration of transmitter release, $\tau_{\text{del}} = 1$ ms is the synaptic delay, and t_{spike} is the time of a spike. For these synapses, $E_{\text{syn}} = -85$ mV and $\bar{g}_{\text{syn}} = 0.35 \mu\text{S}$

Biographies

Carrie A. Williams is a Ph.D. student in the Interdisciplinary Bioengineering Program at Georgia Tech. She received a B.A. in Physics from the College of Wooster and a B.S. in Bioengineering from Washington University in 2000 through a dual-degree engineering

program. Her research interests include using computational models to understand the control of rhythmic movement.

Stephen P. DeWeerth is a Professor in the Wallace H. Coulter Department of Biomedical Engineering and in the School of Electrical and Computer Engineering at the Georgia Institute of Technology and at the Emory University School of Medicine. He received the M.S. degree in Computer Science and the Ph.D. degree in Computation and Neural Systems from the California Institute of Technology in 1987 and 1991, respectively. His research focuses on the implementation of neuromorphic electronic and robotic systems, on the development of neural interfacing technologies, and on the study of the biological control of movement.

Fig. 1.

The closed-loop neuromechanical system with inhibitory (black circles) and excitatory (white circles) synapses. The neural activity is scaled, low-pass filtered, and then subtracted to generate the CPG output (F). The gain (0.25) of the filter was chosen to generate an open-loop CPG output with an amplitude approximately equal to one. The CPG output is used as the input to the mechanical subsystem. The position of the mechanical subsystem (x) is half-wave rectified and used to generate both ipsilateral inhibitory (II) and contralateral excitatory (CE) feedback inputs to the CPG.

Fig. 2. Open-loop CPG activity with synaptic bursting AMPA=3.35 (top) and endogenous bursting NMDA=1 (bottom). The intrinsic CPG frequencies are 3.28 Hz, and 1.33 Hz, respectively.

Fig. 3. Resonance tuning with endogenous bursting. Frequencies of the CPG (F) and the mechanics (x) as the mechanical resonant frequency varies. Results are shown with the II and CE feedback connections individually implemented and combined (IICE). The solid $x=y$ line indicates ideal resonance tuning while the intrinsic CPG frequency (ω_{CPG}) is shown with the vertical dashed line.

Fig. 4. Resonance tuning with synaptic bursting. Frequencies of the CPG (F) and the mechanics (x) as the mechanical resonant frequency varies.

TABLE A1

Parameters for current equations.

Current	Specific conductance (S m^{-2})	E_{rev} (mV)	k_1	k_2	Coeff.	Eqn.	A ($\text{mV}^{-1}\text{ms}^{-1}$)	B (mV)	V_0 (mV)
I_{Na}	835 (initial segment)	50	3	1	α_m	y_3	-0.2	-1	-45
	50.1 (soma)				β_m	y_3	0.06	20	-54
I_{K}					α_h	y_3	0.08	1	-45
					β_h	y_2	0.4 ms^{-1}	-2	-41
	415 (initial segment)	-80	1	0	α_m	y_3	-0.02	-0.8	-45
	83 (soma)				β_m	y_3	0.005	0.4	-35
$I_{\text{Ca}}^{\text{bath}}$ I_{NMDA}	75 * kn	0	1	0	α_m	y_1	0.7 ms^{-1}	17	0
I_{CaAP}					β_m	y_1	10.08 s^{-1}	-17	0
	1 μS	150	5	1	α_m	y_3	-0.08	-11	15
I_{CaNMDA}					β_m	y_3	0.001	0.5	15
	1 μS	20	1	0	α_m	y_1	0.7 ms^{-1}	17	0
I_{KCa}					β_m	y_1	10.08 s^{-1}	-17	0
	$0.04 \mu\text{S} * 4[\text{Ca}_{\text{AP}}] + [\text{Ca}_{\text{NMDA}}]$	-80	0	0					
$I_{\text{AMPA}}^{\text{bath}}$	1 * ka	0	0	0					
I_{leak}	0.83 (initial segment, dendrites)	-70	0	0					
	1.67 (soma)								
I_{a}	300 (initial segment)								
	4.2 (dendrites, soma)		0	0					