

A Minimal Model of a Central Pattern Generator and Motoneurons for Insect Locomotion*

R. M. Ghigliazza[†] and P. Holmes[‡]

Abstract. We adapt the generic three-dimensional bursting neuron model derived in the companion paper [*SIAM J. Appl. Dyn. Syst.*, 3 (2004), pp. 636–670] to model central pattern generator interneurons and slow and fast motoneurons in insect locomotory systems. Focusing on cockroach data, we construct a coupled network that retains sufficient detail to allow investigation and prediction of biophysical parameter changes. We show that the model can encompass stepping frequency, duty cycle, and motoneuron output variations observed in cockroaches, and we reduce it to an analytically tractable symmetric network of coupled phase oscillators from which general principles can be extracted. The model’s modular form allows dynamical analyses of individual components and the addition of other components, so we expect it to be more generally useful.

Key words. central pattern generators, bursting neurons, motoneurons, insect gaits, phase response curves, averaging, bifurcation, stability

AMS subject classifications. 34C15, 34C25, 34C29, 37Gxx, 92B05, 92B20, 92C20

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1. Introduction. Central pattern generators (CPGs) are networks of functionally distinguishable neurons, located in the vertebrate spinal cord or in invertebrate thoracic ganglia, capable of generating and regulating the spatio-temporal activity of motoneurons in the absence of sensory input (e.g., [2, 3, 4]). Over forty years of in vitro and in vivo studies of network architectures, intrinsic membrane properties, and neuromodulators (e.g., [5, 6, 7, 4, 8]) have firmly established their importance in motor behavior. CPG dynamics depends on intracellular, synaptic, and network level phenomena and can display remarkable richness and flexibility.

In this paper, using the reduced bursting neuron ODEs derived and studied in the preceding paper [1], we develop a model of the CPG and associated bursting motoneurons for insect locomotion. We draw on data from the death’s head and American cockroaches *Blaberus discoidalis* and *Periplaneta americana* and focus on rapid running, a regime in which reflexive feedforward control [9, 10] appears to dominate and reflexive feedback plays a less important role [11, 12, 13] than in, e.g., stick insects [14] that use more varied gaits and leg placement strategies. We include enough ionic current and conductance detail to reveal how modulation

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[†]Department of Mechanical and Aerospace Engineering, Princeton University, Princeton, NJ 08544 (rgiglia@princeton.edu). The work of this author was partially supported by a Burroughs-Wellcome Training Grant in Biological Dynamics, 1001782, and a Britt and Eli Harari Fellowship from the Department of Mechanical and Aerospace Engineering, Princeton University.

[‡]Department of Mechanical and Aerospace Engineering and Program in Applied and Computational Mathematics, Princeton University, Princeton, NJ 08544 (pholmes@math.princeton.edu).

of specific biophysical parameters can adjust CPG outputs that determine key locomotive properties, while providing sufficient tractability to enable mathematical analysis. In particular, after reduction of the CPG “core” to phase equations, simple symmetry arguments locate fixed points and describe gaits, and eigenvalue calculations determine their stability properties. We also confirm that these phase reductions correctly represent the dynamics of the full network.

While we emphasize the double-tripod gait employed by *Blaberus* over the speed range 10–60 cm sec^{−1} [15, 16] (and common to many insects), our model also describes other gait patterns, and its modularity will permit the inclusion of additional inter- and motoneurons and reflexive sensing. Indeed, our ultimate goal is to marry it to muscle and body-limb mechanical models of the types developed in [17, 18, 19, 20] and to equip the whole with proprioceptive feedback and goal-oriented direction.

This paper is organized as follows. In section 2 we recall the bursting neuron model of [1] and summarize the effects of bias currents and conductances on its behavior. We then review relevant data on CPG neurons, motoneurons, and network architectures in section 3 and use it to assemble a hexapedal pattern generator in section 4. This comprises six synaptically interconnected CPG bursters, each driving a fast and a slow bursting motoneuron. Finally, to permit elementary analyses of network properties, in section 5 we reduce, via phase response curves (PRCs) and averaging, to phase variables alone. We summarize and outline future work in section 6.

2. A minimal bursting model. The analyses developed in the preceding paper [1] enable us to propose a model sufficiently general to apply to both bursting CPG neurons and motoneurons. It includes a branch of stable equilibria terminating in a saddle-node and one of limit cycles terminating in a global homoclinic bifurcation, separated by a branch of unstable (saddle-type) equilibria. (Index theory [21] implies that the periodic orbits must encircle unstable equilibria, so an upper equilibrium branch also must exist.) A minimal model requires only the saddle-node on the lower equilibrium branch and a Hopf bifurcation to create the periodic orbit on the upper branch. As shown in [1], this can be captured by a fast nonlinear current, e.g., I_{Ca} , a leakage current I_L , a slow potassium current I_K , and an additional very slow current, I_{KS} , giving the system [1, equation (31)]

$$(2.1) \quad \begin{aligned} C\dot{v} &= -[I_{Ca} + I_K + I_L + I_{KS}] + I_{\text{ext}}, \\ \dot{m} &= \frac{\epsilon}{\tau_m(v)} [m_\infty(v) - m], \\ \dot{c} &= \frac{\delta}{\tau_c(v)} [c_\infty(v) - c]. \end{aligned}$$

The currents appearing in (2.1) are

$$(2.2) \quad \begin{aligned} I_{Ca} &= \bar{g}_{Ca} n_\infty(v)(v - E_{Ca}), & I_K &= \bar{g}_K m \cdot (v - E_K), \\ I_L &= \bar{g}_L (v - E_K), & I_{KS} &= \bar{g}_{KSC} \cdot (v - E_K), \end{aligned}$$

where the steady state gating variables $m_\infty(v), n_\infty(v), c_\infty(v)$ and time “constants” $\tau_m(v), \tau_c(v)$

take the forms

$$(2.3) \quad w_{i\infty}(v; k_{i0}, v_{ith}) = \frac{1}{1 + e^{-k_{i0}(v - v_{ith})}},$$

$$(2.4) \quad \tau_i(v; k_{i0}, v_{ith}) = \text{sech}(k_{i0}(v - v_{ith})),$$

with $w_{i\infty} = m_{\infty}(v), n_{\infty}(v), c_{\infty}(v)$. Parameters were generally fixed as specified in Table 1 of section 4; modifications will subsequently be made to accomodate other behaviors. All parameters excepting $C, \bar{g}_K, \epsilon, \delta$ are the same as in Morris and Lécar [22, 23], $\bar{g}_K = 9$ being slightly higher than their value $\bar{g}_K = 8$. The parameters C, ϵ , and δ , which independently determine the time scales of v, m , and c , are set to match typical cockroach data.

As shown in section 4 of [1], variations in three key characteristics of the bursting pattern can be achieved quasi-independently by varying two biophysical parameters for each of the different neuron types to be used in the model. Specifically, in the appropriate regimes, the following hold:

1. The bursting frequency can be adjusted primarily by I_{ext} .
2. The spiking frequency can be adjusted by I_{ext} .
3. The number of action potentials (APs) can be adjusted by I_{ext} and $\bar{g}_{KS}$.
4. The duty cycle can be adjusted by $\bar{g}_{KS}$, although this may also affect frequency.

As we shall see, the bursting frequency and duty cycles of CPG interneurons are primarily responsible for speed adjustment (although leg extension, via stride lengths, is also important at higher speeds [24]), while motoneuron spiking frequencies and AP numbers grade force production. We remark that I_{ext} can be modulated by excitatory and inhibitory synapses from CNS neurons, and $\bar{g}_{KS}$ by suitable neurotransmitters, so both of these are biophysically plausible control parameters in vivo.

3. CPG neurons and motoneurons as bursters. Before proposing specific parameter regimes for cockroach CPG and motoneuron models, we review relevant data on animals and insects in general and cockroaches in particular. We start by briefly commenting on spiking and nonspiking interneurons in CPGs, turn to motoneurons, and then discuss network connectivity.

3.1. CPG neurons: Bursters and nonspikers. Working from direct recordings and deaf-ferented (sensorless) preparations of the American cockroach *Periplaneta americana* [25, 26], Pearson [27, 28] hypothesized a flexor burst generator for each leg that comprises several interneurons, including a bursting interneuron that periodically excites the flexor (levator or swing) motoneurons while inhibiting the extensor (depressor or stance) units. Subsequently, interneurons that do not produce APs were found [29, 28, 30], and their importance in generating motor patterns was stressed. (In the locust they are responsible for coordinating subsets of motoneurons, controlling their spiking frequencies, and altering reflex strengths and movement magnitudes in a continuous and precise manner through graded potentials [31, 32].) Despite this, there is no evidence that nonspiking interneurons exhibit pacemaking capabilities; indeed, their quasi-sinusoidal membrane voltages could simply result from integration of incoming bursts [33]. Thus, while they may be involved in CPG circuits and may contribute in a graded manner to slow motoneuron outputs, we shall omit them from our model.

Plateau potentials, slow voltage oscillations on which the fast spikes ride, do however seem crucial to bursting [34]. These derive from bistability of the type illustrated in the bifurcation diagrams of Figures 9 and 10 of [1] for the fast subsystem, which allows brief inputs to trigger activities that outlast input duration; similarly, brief inhibitory stimuli can terminate plateaus [35]. This nonlinear membrane property plays a pivotal role in structuring bursts and producing cyclic behavior with appropriate time scales for stepping frequencies; it also allows brief proprioceptive inputs to reset and regulate the rhythm. We shall therefore represent each of the six “leg units” of an insect CPG by a single bursting (inter-) neuron of the form (2.1)–(2.2), synapsing directly on motoneurons innervating the dominant depressor muscles. Our model allows for subsequent addition of nonspiking interneurons between CPG and motoneurons.

3.2. Motoneurons. Because of important constraints imposed by their physiology, we discuss motoneurons and muscles in some detail.

The basic functional component of motor pathways is the motor unit, consisting of a motoneuron and the muscle fibers innervated by it. A single AP in the motoneuron causes a contractive twitch in the muscle fibers to which it is attached. Three types of motor units can be distinguished by their motoneuron firing patterns and muscle fiber properties. Slow twitch (S-type) units take about 50 msec to develop peak force and show little decline in force over prolonged periods of repetitive stimulation; they can exert low forces for very long periods. In contrast, fatigue resistant (FR) and fast fatigue (FF) units maximally contract in 5–10 msec. With repetitive stimuli, FR units can sustain moderate forces for ≈ 5 min before steady decline sets in over many minutes. FF motor units can achieve the greatest force of the three types, but with repetitive stimuli the force drops precipitously after 30 seconds or so. Both FR and FF units produce rapid large forces and so are found preferentially in muscles involved in executing fast movements. In the cockroach, slow and fast motoneuron discharges are quite distinct; slow units spike continuously at rates from 100–400 Hz when active [26, 36], while fast units typically produce 1–6 large spikes during a 50–100 msec stance or swing phase [37].

Muscle contraction force is determined by the motor pool in two ways. Small force increases are primarily met by greater motoneuron firing rates, but for larger contractions the number of active motoneurons is increased in a process called recruitment. This occurs in an orderly manner in the sequence S-FR-FF [38], determined jointly by the effect of cell body size [39] on excitatory postsynaptic potentials and on graded inputs to S, FR, and FF units. An incoming (tonic) stimulus sequentially excites the units as it passes their different thresholds [40, 34]. Cockroach coxal depressor motoneurons are innervated by both fast and slow motoneurons [41], and in *Blaberus* fast motoneuron recruitment begins at leg cycle rates of ≈ 6 Hz, corresponding to running speeds of 12 cm sec^{-1} [37]; fast motoneurons dominate at high speeds, but there is a considerable “overlap range” [26].

Often only slow motoneurons are modeled. Since these exhibit continuous relationships between firing frequency and force production, compact reductions of the whole neuromotor complex are then possible (e.g., the neuromuscular transforms of [42]). However, since we focus on rapid running, in which fast motoneurons are involved, and there is strong evidence of plateau and bursting capabilities in cockroach motoneurons [35], spiking and bursting

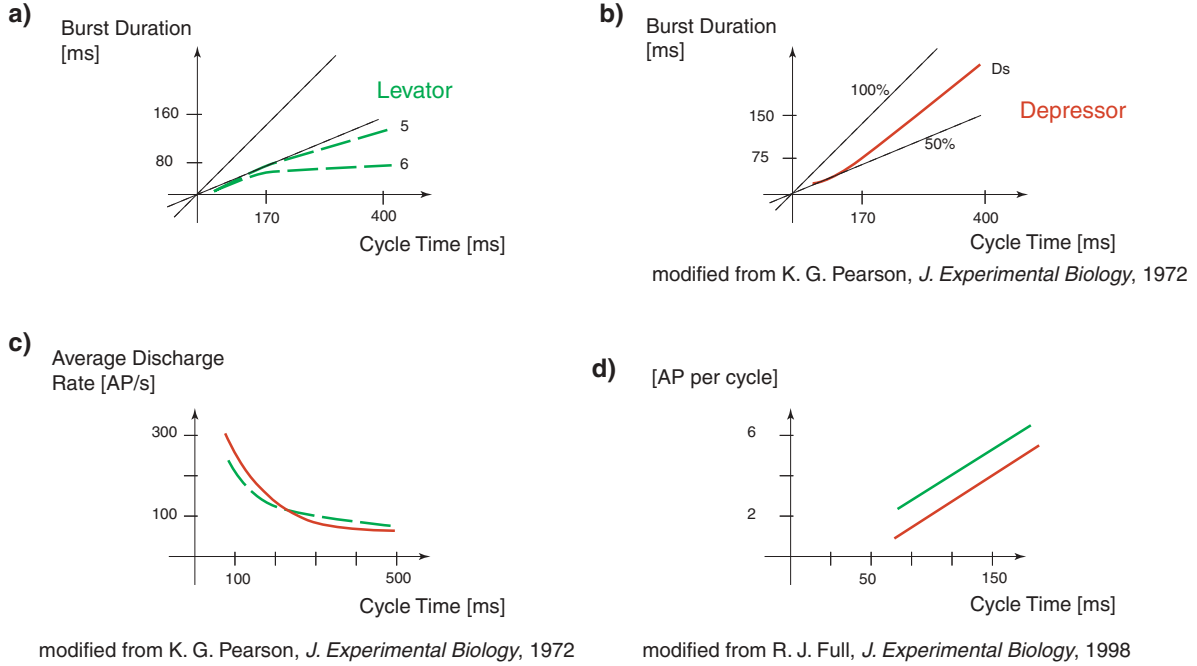
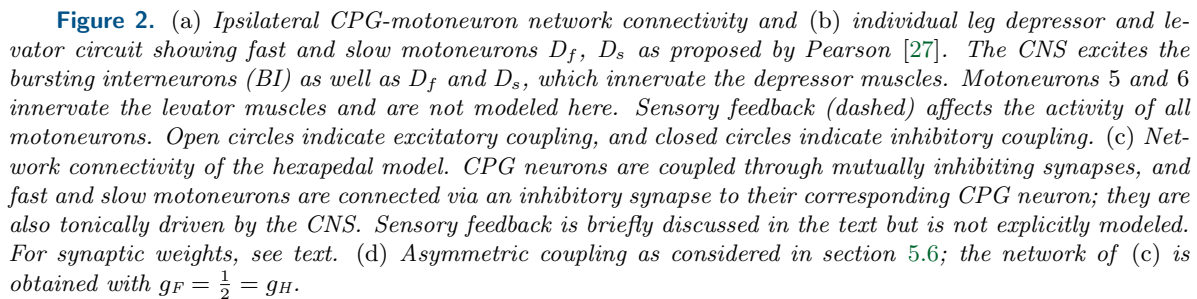


Figure 1. (a) Burst duration in levator (dashed, axons 5 and 6) and (b) slow depressor D_s axon (solid) as a function of cycle time. The two thin dashed lines indicate duty cycles of 100% and 50%. Note that duty cycles of both depressors and levators approach 50% as speed increases (cycle time decreases). Cycle frequency ranges from 2 to 10 Hz. (c) Average spike rate of levator (dashed) and depressor (solid) axons. From Pearson [26]. (d) Approximate numbers of muscle action potentials (MAPs) per cycle in metathoracic (upper curve) and mesothoracic muscle (lower curve): Regression equations $MAPs = 0.051t_{cyc} - 2.5$, $R^2 = 0.52$ for metathoracic and $MAPs = 0.048t_{cyc} - 3.2$, $R^2 = 0.42$ for mesothoracic. From Full et al. [37].

behaviors cannot be ignored. Indeed, given the few large spikes typically seen during rapid running, spike times and interspike intervals may be crucial in determining relative forces in different legs of the stance tripod and in regulating episodes of negative and positive work [37]. These aspects are certainly as important as the analogous role of slow motoneuron spiking frequency in low-speed walking. For this reason, and to allow continuous transition from slow to fast speeds, we shall use the bursting model (2.1), with suitable parameter choices, to represent both fast and slow motoneurons.

In Figures 1(a)–(c) we reproduce *Periplaneta* data from [26, 27] showing burst durations and spiking rates of slow cockroach motoneurons as functions of cycle time or inverse stepping frequency (cf. [43, 36] for analogous and more recent *Blaberus* data). In Figure 1(d) we reproduce data from [37] showing the dependence of number of APs in fast motoneurons as a function of cycle time. Phase relationships among leg muscles (not shown) indicate near constant antiphase between motoneurons associated with the left and right tripods. This data will guide our parameter choices.

3.3. Network configuration. Apart from anatomic identification and the acceptance of some degree of hierarchy [44, 45, 34], the precise division of labor among the higher central nervous system (CNS), the CPG-motoneuron complex, and proprioceptive sensing and



Henceforth we exclude levators, since our mechanical models neglect leg masses and the

swing phase is implicit [17, 12, 20]. In cockroaches there are two slow (177D and 177E) and two fast (178 and 179) coxal depressor muscles, the former being innervated by the slow motoneuron D_s and the latter by D_f ; some fibers in 177D also receive inputs from D_f [41]. With this and the discussion of section 3.2 in mind, we now develop our network model.

4. A hexapedal neuro-motor complex. Pearson did not address contralateral connectivity, but it is natural to extend his model to a network of six mutually inhibiting units, as shown in Figure 2(c) (also cf. the stick-insect pattern generator proposed in [14, Fig. 4]). This architecture promotes contra- and ipsilateral neighbors to burst in antiphase, leading units 1, 2, 3 and 4, 5, 6 to form two groups, internally in phase but mutually in antiphase, thus forming the left and right (depressor) tripods. As noted above, we do not include interneurons, so the output of each CPG neuron inhibits the slow and fast depressor motoneurons directly. By inhibiting motoneuronal activity, the CPG selects both a stepping pattern and sets the leg cycle frequency, but the CPG spiking frequency does not directly affect motoneuron spiking frequencies, which are jointly adjusted by the local proprioceptive feedback and CNS drive; see Figures 2(a), (b). CPG neurons and both slow and fast motoneurons will be modeled by (2.1)–(2.2) with differing parameters as specified in Table 1.

Inhibitory coupling can be achieved via synapses that produce negative postsynaptic currents, or presynaptically by depressing a synapse. Lacking more precise information, we choose the former mechanism. Following [47, p. 15], [48, p. 180], we adopt the first order dynamics

$$(4.1) \quad \dot{s} = \alpha G(v_{\text{pre}}) (1 - s) - \beta s, \quad \text{with} \quad G(v_{\text{pre}}) = \frac{T_{\text{max}}}{1 + e^{-k_{\text{pre}}(v_{\text{pre}} - E_{\text{syn}}^{\text{pre}})}},$$

in which v denotes the potential of the presynaptic neuron and α, β and the parameters $T_{\text{max}}, k_{\text{pre}}$, and $E_{\text{syn}}^{\text{post}}$ defining the concentration of transmitter release $G(v_{\text{pre}})$ set the timescale of the synaptic rise and decay described by the nondimensional variable s . The variable s enters the postsynaptic cell in the first equation of (2.1) as an additional term,

$$(4.2) \quad C\dot{v} = -[I_{\text{Ca}} + I_{\text{K}} + I_{\text{L}} + I_{\text{KS}}] + I_{\text{ext}} - \bar{g}_{\text{syn}} s \cdot (v - E_{\text{syn}}^{\text{post}}),$$

where $\bar{g}_{\text{syn}}$ denotes synaptic strength and the current $I_{\text{syn}} = -\bar{g}_{\text{syn}} s \cdot (v - E_{\text{syn}}^{\text{post}})$ induced in the postsynaptic cell is typically positive and hence depolarizing (resp., negative and hence hyperpolarizing) for excitatory (resp., inhibitory) synapses [33]. A different form of the s -equation (4.1) appears in [49]. We have checked that this produces similar results to those described below.

Table 1 lists parameter values adopted for the CPG and motoneuron models and Table 2 lists those for the synapses (standard inhibitory GABA_A; see [47]). All three types of neurons have equal “fixed” parameter values except for C , ϵ , and E_{syn} . The physiologically adjustable control parameters, $\bar{g}_{\text{KS}}$, I_{ext} , and $\bar{g}_{\text{syn}}$ with nominal “standard” values indicated by asterisks will be varied to match the data summarized in section 3. To obtain equal current injection into all six CPG neurons under stationary conditions, we chose half weights for the synapses from units 1 and 3 to 5, and 4 and 6 to 2 (i.e., $\bar{g}_{\text{syn}} = 0.005$ in place of 0.01 as given in Table 2), since the middle leg units receive input from three others, while other units have inputs from only two (see Figure 2(c)). Contralateral CPG synapses are set at full strength, since in-phase contralateral activity can occur with weak inhibition; cf. [5] and see sections 5.3 and 5.7 below.

Table 1

Parameters for CPG and fast and slow motoneurons D_f , D_s . Maximal conductances are expressed in mS/cm^2 , the reversal and threshold potentials in mV , the slope coefficients in mV/s , and the capacitance C in $\mu\text{F}/\text{cm}^2$.

	C	$\bar{g}_{\text{Ca}}$	$\bar{g}_{\text{K}}$	$\bar{g}_{\text{KS}}$	$\bar{g}_{\text{L}}$	E_{Ca}	E_{K}	E_{L}	v_{thCa}	v_{thK}	v_{thc}
CPG	1.2	4.4	9.0	0.19*	2.0	120	-80	-60	-1.2	2	-27
D_f	1.39	4.4	9.0	0.25*	2.0	120	-80	-60	-1.2	2	-27
D_s	2.4	4.4	9.0	0.50	2.0	120	-80	-60	-1.2	2	-27
	$k_{0\text{Ca}}$	$k_{0\text{K}}$	k_c	ϵ	δ	I_{ext}					
CPG	0.056	0.1	0.8	4.9	0.052	35.6*					
D_f	0.056	0.1	0.8	4.18	0.044	36.3*					
D_s	0.056	0.1	0.8	2.0	0.0002	50*					

Table 2

Synapse parameters. Only the CPG neurons have “outgoing” synapses.

	$E_{\text{syn}}^{\text{pre}}$	$E_{\text{syn}}^{\text{post}}$	k_{syn}	α	β	$\bar{g}_{\text{syn}}$	T_{max}
CPG	2	-70	0.22	5000	0.180	0.01*	$2 \cdot 10^{-3}$
D_f		-70	0.22	5000	0.180	0.2*	
D_s		-70	0.22	5000	0.180	0.9*	

4.1. Pairs of coupled bursting neurons. Before studying the full circuit of Figure 2(c), we consider a pair of CPG neurons with mutually inhibitory and excitatory couplings and a CPG neuron unidirectionally coupled to fast and slow motoneurons.

Depending on their intrinsic bursting frequencies and the strength of the coupling term $\bar{g}_{\text{syn}}$ of (4.2), the units may entrain (frequency lock). Figures 3(a), (b) show pairs of identical CPG neurons mutually coupled by inhibitory synapses (left column) and excitatory synapses (right column). In the first case they antiphase lock within a cycle; in the second the bursts entrain, although individual spikes may not. Unidirectionally driven fast motoneurons entrain to the bursting frequency of CPG neurons in Figures 3(g), (h). Slow motoneurons are essentially continual spikers, but with sufficiently strong inhibitory coupling, they can be made to burst in alternation with the CPG inputs in agreement with animal recordings; see Figure 3(i). With excitatory coupling, spiking persists throughout, but the rate increases during an incoming CPG burst; see Figure 3(j). The intervening panels (c)–(f) show the synaptic variable s and the resulting currents $-I_{\text{syn}}$. The s -dynamics is similar in both inhibitory and excitatory cases; the major difference lies in postsynaptic currents.

4.2. A hexapedal CPG. We now move to the full circuit of Figure 2(c). Synaptic currents and other relevant parameters will be distinguished for CPG, fast, and slow motoneurons by adding appropriate subscripts. Each CPG neuron forms three types of synapses: to other CPG neurons through $I_{\text{syn,CPG}}$ and to fast and slow motoneurons through I_{syn,D_f} , I_{syn,D_s} , respectively. Figure 4 shows typical time histories of ipsilateral and contralateral CPG neurons and motoneurons; note the alternating activity of the left (1, 2, 3) and right (4, 5, 6) tripods. Also, burst durations of the slow motoneurons D_s are longer than those of CPG neurons, and

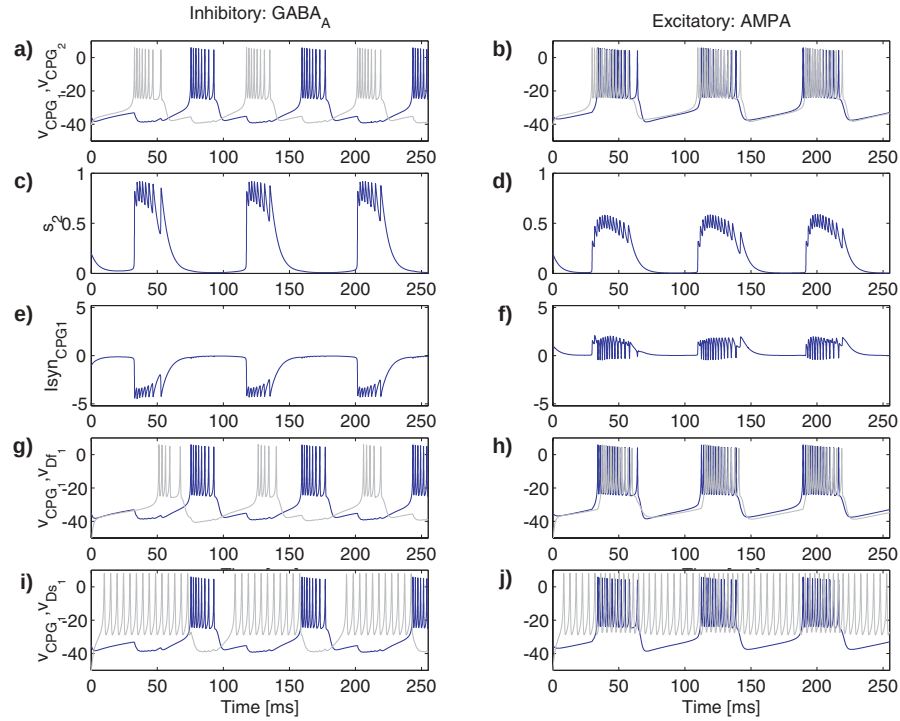


Figure 3. Two coupled bursting neurons, with $GABA_A$ inhibitory synapses (left panels) and AMPA excitatory synapses (right panels). Panels (a)–(b) show membrane voltages of mutually coupled CPG_1 (dark grey) and CPG_2 (light grey) neurons, respectively; inhibitory coupling causes antiphase bursts (a) and excitatory coupling causes in-phase bursts (b). Panels (c)–(d) show synaptic dynamics $s(t)$ and (e)–(f) the resulting synaptic currents $-I_{syn}$: Negative in the inhibitory case and positive in the excitatory case. Panels (g), (h) show the membrane voltage of a CPG neuron (dark grey) superimposed on that of a unidirectionally coupled fast motoneuron D_{f1} (light grey). With inhibitory coupling (g), D_{f1} bursts in antiphase with respect to CPG, but in-phase with excitatory coupling (h). Panels (i)–(j) show the membrane voltage of a CPG neuron (dark grey) superimposed on that of a unidirectionally coupled slow motoneuron D_{s1} (light grey). With inhibitory coupling (i), D_{s1} bursts in antiphase with respect to CPG, but with excitatory coupling, it continues to spike, with increased rate during CPG bursts (j). Parameters are as in Table 1 except for $I_{CPG} = 36.3$, with coupling strengths $\bar{g}_{CPG,CPG} = 0.15$, $\bar{g}_{CPG,D_f} = 0.25$, and $\bar{g}_{CPG,D_s} = 0.4$ for both the inhibitory and excitatory cases. The coefficients for AMPA synapses are as in Table 2, except for $\alpha = 1100$, $\beta = 0.190$, $E_{syn}^{post} = 0$. Some panels show effects of transients, and we note that while bursts are synchronized, individual spikes (and spike numbers) need not be.

their spiking frequency is approximately constant, in agreement with experiments [26].

Via $I_{ext} = I_\alpha$ and $\bar{g}_{KS,\alpha}$, where $\alpha = \{CPG, D_f, D_s\}$, we can adjust the stepping frequency, the number of APs in D_f , the spiking rate of D_s , and their duty cycles, as described in [1] and section 2 above. We note that all three neuron types can have different duty cycles. In the locomotion literature duty cycle normally refers to S-type (slow) muscle fibers or slow motoneuron activity (in fast fibers it is not a relevant measure). In our network, the duty cycle of the slow motoneurons can be indirectly controlled through that of the CPG neurons. Since CPG neurons drive motoneurons through inhibitory synapses, and duty cycles of the former are generally less than 0.5, motoneuron duty cycles typically exceed 0.5. Hence, by suitable

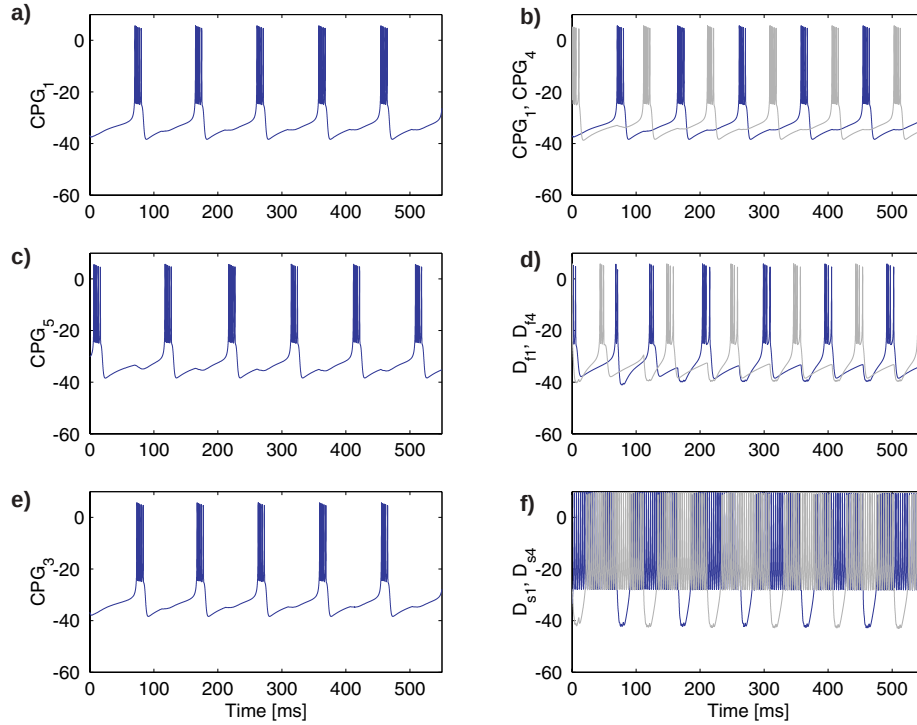


Figure 4. Membrane voltages of CPG neurons and motoneurons in the hexapedal model during fictive locomotion. Neurons are labeled as in Figure 2(c), and parameter values are as in Tables 1 and 2. The left column shows ipsilateral CPG neurons, while the right column shows contralateral CPG neurons and fast and slow motoneurons for units 1 and 4. Bursting frequency is 10.4 Hz, D_f has 4 APs per burst, and D_s spikes at a rate of ≈ 290 Hz with a duty cycle of 0.80; $(f_{\text{Burst}}, n_{\text{AP}}, f_{\text{Spike}}, \Delta_{D_s}) = (10.4 \text{ Hz}, 4, 287 \text{ Hz}, 0.80)$. Parameters are as in Tables 1 and 2.

parameter choices we can reproduce Pearson’s finding that motoneuron burst durations vary from 0.4 to 0.9 of the full cycle period as the latter increases; see Figure 6(d). From now on, unless otherwise stated, by duty cycle we mean the D_s duty cycle.

In the following we show how the network can be adjusted for different locomotive requirements, in comparison with the nominal case of Figures 4(b), (d), (f) in the insect’s preferred speed range, in which the stepping frequency is 10.3 Hz, the D_f have four APs per burst, and the D_s spike at a rate of 279 Hz and have a duty cycle of 0.59. We write these four “outputs” as $(f_{\text{Burst}}, n_{\text{AP}}, f_{\text{Spike}}, \Delta_{D_s})$. Figures 5(a₁)–(a₃) show slow walking (3.66 Hz, 1, 147 Hz, 0.88). Figures 5(b₁)–(b₃) show how it is possible to vary the number of APs in fast motoneurons and the spiking frequency of slow motoneurons independent of stepping frequency: still slow walking but with increased force production, as required, e.g., for hill climbing (3.66 Hz, 7, 353 Hz, 0.95). Figures 5(c₁)–(c₃) show fast stepping, with an intermediate number of APs and spiking rate (17.2 Hz, 4, 287 Hz, 0.44). Note that in this case the D_s duty cycle is slightly less than 0.5 and there is no overlap.

To adjust to rapid external disturbances, flexibility is required in load as well as speed. In the following we show how, in a multiparametric setting, the four main characteristics of

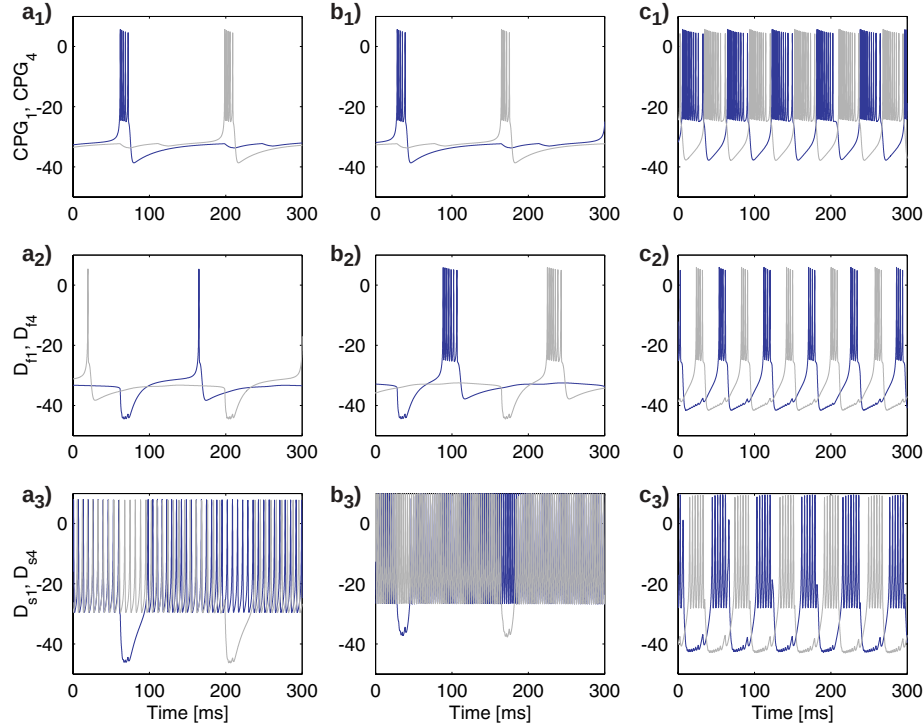


Figure 5. Membrane voltages of CPG neurons and fast and slow motoneurons in the hexapedal model during fictive locomotion, for comparison with “nominal” case of Figure 4 (right column). (a₁)–(a₃) Slow stepping, low force: $(f_{Burst}, n_{AP}, f_{Spike}, \Delta_{D_s}) = (3.66 \text{ Hz}, 1, 147 \text{ Hz}, 0.88)$; parameters are as in Figure 4 except for $I_{CPG} = 35.38$, $I_{D_f} = 35.7$, $I_{D_s} = 44$, $\bar{g}_{KS,D_f} = 0.45$, $\bar{g}_{CPG,D_f} = 0.5$. (b₁)–(b₃) Slow stepping, large force: $(f_{Burst}, n_{AP}, f_{Spike}, \Delta_{D_s}) = (3.66 \text{ Hz}, 7, 353 \text{ Hz}, 0.95)$; parameters are as in Figure 4 except for $I_{CPG} = 35.38$, $I_{D_f} = 35.8$, $I_{D_s} = 64$, $\bar{g}_{KS,D_f} = 0.13$, $\bar{g}_{CPG,D_f} = 0.5$. (c₁)–(c₃) Fast stepping, medium force: $(f_{Burst}, n_{AP}, f_{Spike}, \Delta_{D_s}) = (17.2 \text{ Hz}, 4, 287 \text{ Hz}, 0.44)$. Parameters are as in Figure 4 except for $I_{CPG} = 38.4$, $I_{D_f} = 37.4$, $I_{D_s} = 50$.

the network can be adjusted over a wide range, with sufficient independence. Parameters not explicitly noted are as in Tables 1–2. Figure 6(a) shows the variation of bursting frequency with I_{CPG} parametrized by the maximal conductance $\bar{g}_{KS,CPG}$. Together they span the range 5–26 Hz (although a lower frequency of 3.2 Hz was obtained with $I_{CPG} = 35.38$, $I_{D_f} = 35.7$), encompassing the entire range over which *Blaberus discoidalis* uses the double-tripod gait. Figure 6(c) shows how the duty cycle of slow motoneurons is affected by changes in I_{CPG} and $\bar{g}_{KS}$. Figure 6(b) shows the variation of the spiking frequency of the slow motoneurons with I_{D_s} parametrized by I_{CPG} . Variation of I_{D_s} in the range 38–64 provides frequencies from 124 Hz to 389 Hz. Figure 6(c) shows duty cycle variation with I_{CPG} parametrized by $\bar{g}_{KS,CPG}$, indicating coverage of the range from 0.4 to 0.9, and Figure 6(d) shows this data superimposed on measurements of Pearson [26].

Figure 6(e) shows the variation of the number of APs in fast motoneurons with I_{D_f} parametrized by I_{CPG} . In the first case the number of APs per burst changes only from 3 to 4, but this is significant in the 10 Hz frequency range; cf. [37]. A wider range is obtained when $I_{CPG} = 38.4$, corresponding to a stepping frequency of 17.0 Hz; here the number of APs

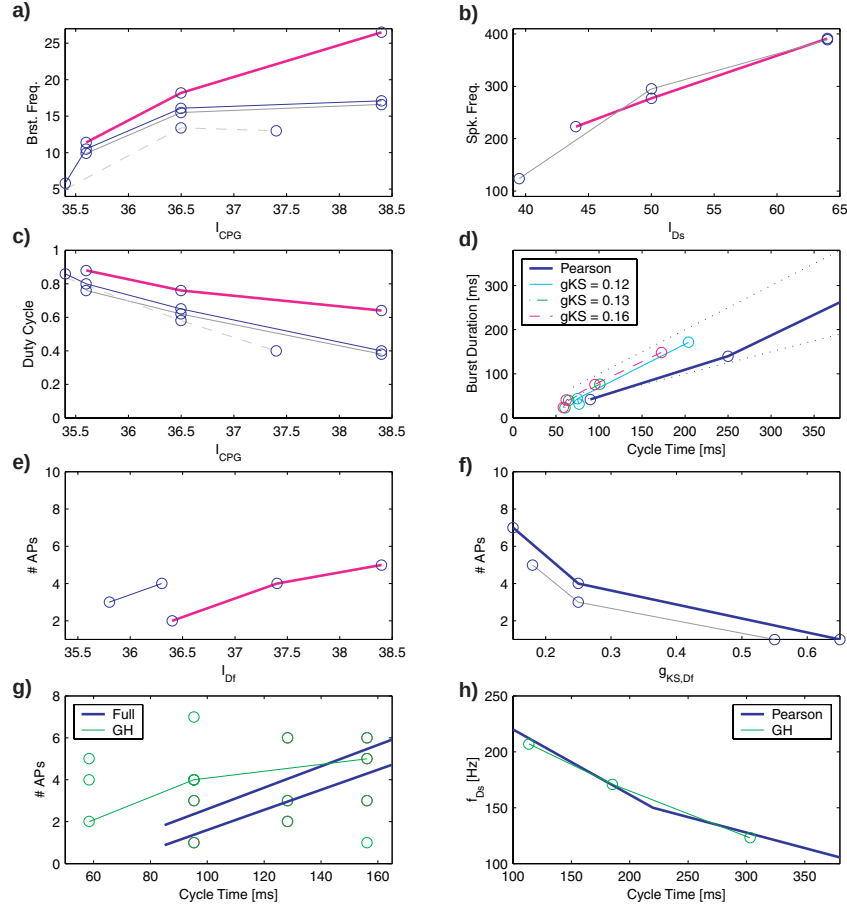


Figure 6. (a) Network bursting frequency vs. I_{CPG} for $\bar{g}_{KS,CPG} = 0.35$ (bold), 0.19 (dark), 0.18 (solid grey), and 0.15 (dashed grey). (b) Slow motoneuron spiking frequency vs. I_{DS} for low $I_{CPG} = 35.6$ (solid, stepping frequency 10.5 Hz), and 38.4 (bold, stepping frequency 17.0 Hz). (c) Duty cycle vs. I_{CPG} , parametrized by $\bar{g}_{KS,CPG}$, parameter values and curves as in panel (a). (d) Duty cycle vs. cycle time: Data from Pearson [26, Fig. 6] (bold) and obtained by varying I_{CPG} from 35.38 to 36.7 and keeping $\bar{g}_{KS}$ fixed at the values indicated (dashed and broken lines); dotted lines correspond to 50% and 100% duty cycle. (e) Number of fast motoneuron APs per burst vs. I_{DF} , for $I_{CPG} = 35.6$ (solid, bursting frequency 10.5 Hz); $I_{CPG} = 38.4$ (bold, bursting frequency 17.0 Hz). (f) Number of fast motoneuron APs per burst vs. $g_{KS,DF}$ for $I_{CPG} = 35.41$, $I_{DF} = 35.7$ (solid, bursting frequency 6.4 Hz); $I_{CPG} = 35.6$, $I_{DF} = 36.5$ (bold, bursting frequency 10.5 Hz). (g) Fast motoneuron APs vs. cycle time: Data from Full [37, Fig. 5] with mesothoracic (upper, bold) and metathoracic (lower, bold) regression lines. Model results shown in circles and broken line; see text for explanation. (h) Slow motoneuron spiking frequency vs. cycle time: Data from Pearson [26, Fig. 7] (bold) and model results on broken line; see text.

ranges from 2 to 5. Figure 6(f) shows variation of the number of APs with $\bar{g}_{KS,DF}$ parametrized by I_{CPG} , indicating that from 1 to 7 APs can be delivered. Recalling that each spike causes a muscle fiber twitch, the model can therefore achieve up to a sevenfold graded increase in force production, covering the entire range described in [37]. Finally, Figures 6(g), (h) replot the fast motoneuron AP numbers and slow motoneuron spike rates achieved by the model in comparison with those measured by Full et al. [37] and Pearson [26], showing that the model

can reproduce the data rather well. To match Full's overall finding that slower stepping (increased cycle time) results in more APs, we adjusted the network bursting frequency via $I_{\text{CPG}} = 35.41, 35.6, 38.4$ and concurrently adjusted the bias current $I_{D_f} = 35.7, 36.3, 36.4$ to produce the broken line (for the rightmost data point $g_{\text{KS},D_f} = 0.18$ was slightly less than for the others $g_{\text{KS},D_f} = 0.19$). The circles in panel (g) show network behaviors obtained for a broader variation of I_{CPG} , I_{D_f} , and $\bar{g}_{\text{KS},D_f}$ that span the wide variability identified in [37, Fig. 5]. The circles in (h) were obtained by concurrently changing I_{CPG} from 35.38 to 35.6 and I_{D_s} from 40 to 44. We also found other cases in which the number of APs decreases with increasing cycle time.

All results shown in this section correspond to tripod gaits, with 1:1 entrainment of CPG and fast and slow motoneurons, as in Figure 5. The impossibility of extending some curves beyond the ranges shown (e.g., Figure 6(d), solid line) is due to failure of “normal” network properties: e.g., CPG neurons, fast and/or slow motoneurons cease to fire at all (typically at low values of the current), or fire tonically (high bias currents); or 1:1 phase locking of fast motoneurons and CPG neurons is lost. Nonetheless, these simulations show that the tripod gait can be maintained over a wide range of speeds and duty cycles, and that in a multiparameter setting, bursting frequency, spiking frequency, duty cycle, and the number of APs can be almost independently changed.

5. Reduction to phase oscillators. In this section we review the phase reduction and averaging methods and apply them to coupled bursting CPG neurons of the type (2.1) with synaptic dynamics (4.1). We derive reduced sets of ODEs describing mutually coupled pairs of neurons and the CPG network of Figure 2(c) in terms of relative phases, and analyze them to find phase locked solutions and their stability properties.

5.1. The phase response curve. We write the ODE for a single cell in the compact form

$$(5.1) \quad \dot{\mathbf{x}} = \mathbf{f}(\mathbf{x}) + \alpha \mathbf{p}(\mathbf{x}, t, \dots); \quad \mathbf{x} \in \mathbb{R}^n,$$

where $\mathbf{p}$ denotes the coupling function (of strength α) and $(\dots)$ in the argument of $\mathbf{p}$ contains state variables of all cells that synapse onto the one in question, as well as external inputs. The phase reduction method originated in work of Malkin and Winfree [50, 51]; more details can be found in [52, Chap. 9] and [53], and an application to the “standard” Hodgkin–Huxley equations in [54].

We assume that, for $\alpha = 0$, (5.1) has an attracting hyperbolic limit cycle Γ_0 , with period T_0 and frequency $\omega_0 = \frac{2\pi}{T_0}$ (the bursting cycle). We define a scalar phase variable $\phi(\mathbf{x}) \in [0, 2\pi)$ for all $\mathbf{x}$ in some neighborhood U of Γ_0 (within its domain of attraction), such that the phase evolution has the simple form $\dot{\phi} = \omega_0$ for all $\mathbf{x} \in U$. The cycle Γ_0 persists for small $\alpha \neq 0$ [21], and, from the chain rule, we deduce that

$$(5.2) \quad \dot{\phi} = \frac{d\phi}{dt} = \frac{\partial \phi}{\partial \mathbf{x}} \cdot [\mathbf{f}(\mathbf{x}) + \alpha \mathbf{p}(\mathbf{x}, t, \dots)] = \omega_0 + \alpha \frac{\partial \phi}{\partial \mathbf{x}} \cdot \mathbf{p}(\mathbf{x}, t, \dots).$$

Equation (5.2) defines a first order PDE that the scalar field $\phi(\mathbf{x})$ and its inverse $\mathbf{x} = \mathbf{x}(\phi)$ must satisfy; $\phi(\mathbf{x})$ is unique up to a translational constant which may be fixed by setting $\phi(\mathbf{x}) = 0$ at a distinguished point of Γ_0 . For periodically spiking neurons, this is often the

voltage peak; in the present application it will be the upward crossing of -30 mV preceding the first spike in the burst. The theory of isochrons [55] implies that the phase space $\mathbb{R}^n$ near Γ_0 is foliated by $(n-1)$ -dimensional manifolds

$$M_{\bar{\phi}} = \left\{ \mathbf{x} \in \mathcal{B} : \lim_{t \rightarrow \infty} \mathbf{x}(t) \sim \phi(t) = \omega_0 t + \bar{\phi} \right\},$$

from which solutions approach Γ_0 with the same asymptotic phase.

Introducing the relative phase $\psi = \phi - \omega_0 t$ and approximating the derivative in (5.2) by its value on the uncoupled limit cycle $\mathbf{Z}(\phi) \stackrel{\text{def}}{=} \frac{\partial \phi}{\partial \mathbf{x}}|_{\Gamma_0(\phi)}$, (5.2) becomes

$$(5.3) \quad \dot{\psi} = \alpha \mathbf{Z}(\phi) \cdot \mathbf{p}(\phi).$$

For mutual coupling among N identical units, defining the phase variables $\mathbf{x}_i = \mathbf{x}_i(\phi_i)$ and $\psi_i = \phi_i - \omega_0 t$, this generalizes to

$$(5.4) \quad \dot{\psi}_i = \sum_{j \neq i}^N \alpha_{ji} \mathbf{Z}(\phi_i) \cdot \mathbf{p}_{ji}(\phi_i, \phi_j).$$

For weak coupling ($|\alpha| \ll 1$), the phases ϕ_i evolve on a much faster time scale than ψ_i , so we may appeal to averaging theory [21] (cf. [52, p. 259, Malkin's theorem]) to integrate over the unperturbed period and obtain

$$(5.5) \quad \dot{\psi}_i = \sum_{j \neq i}^N \alpha_{ji} H_{ji}(\psi_i - \psi_j),$$

where

$$(5.6) \quad H_{ji}(\psi_i - \psi_j) = \frac{1}{T_0} \int_0^{T_0} \mathbf{Z}(\omega_0 t + \psi_i) \cdot \mathbf{p}_{ji}(\Gamma_0(\omega_0 t + \psi_i), \Gamma_0(\omega_0 t + \psi_j)) dt.$$

Note that only phase *differences* appear in the averaged coupling functions H_{ji} due to periodicity of the integrand in (5.6).

In the case that the perturbation or coupling functions $\mathbf{p}_{ji}$ only enter through the first component of $\mathbf{x}$, as in $\dot{v}$ via I_{syn} in (4.2), we have

$$(5.7) \quad \alpha_{ji} \mathbf{p}_{ji}(\mathbf{x}_i, \mathbf{x}_j, t) = \begin{bmatrix} \bar{g}_{\text{syn},ji} s_j \cdot (v_i - E_i) \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

where s_j denotes the synaptic variable associated with the j th cell and $\bar{g}_{\text{syn},ji}$ the synaptic strength from the j th to the i th cell. Here only the first component $Z_1(\phi)$ of the 2π -periodic function $\mathbf{Z}(\phi)$ appears in (5.6); this is called the *phase response curve* (PRC), and it may be approximated numerically by perturbing from the limit cycle at each phase ϕ with a voltage

increment $v \mapsto v + \Delta v$ and allowing the solution to recover to its new asymptotic phase $\phi \mapsto \phi + Z_1(\phi)$. The resulting *infinitesimal PRC* is valid in the combined limit

$$(5.8) \quad Z_1(\phi) = \lim_{\substack{\Delta v \rightarrow 0 \\ t \rightarrow \infty}} \frac{\Delta \phi}{\Delta v}.$$

$Z_1(\phi)$ may also be computed by use of adjoint theory [52], e.g., as implemented in the software XPP [56]. For $Z_1(\phi) > 0$ (resp., < 0), positive voltage perturbations advance (resp., retard) the phase.

5.2. PRCs and averaged coupling functions. Applying the theory sketched above to (2.1), we obtain the infinitesimal PRC of Figure 7(a). Here $Z_1(\phi)$ was computed numerically by taking successively smaller voltage perturbations Δv starting at $\Delta v = 31.6 \text{ mV}$ and reducing to 0.1 mV ; the PRC stabilized in the form shown for $|\Delta v| < 1 \text{ mV}$. If the linearity assumption inherent in (5.8) holds, positive and negative perturbations should yield the same result, since the infinitesimal PRC is a derivative. We verified that this is the case using perturbations $\Delta v = \pm 0.08$ and finding good convergence over the whole range $\theta \in [0, 2\pi)$; see the solid and dashed curves in Figure 7(a).

In Figure 7(b) we show how the estimate of Z at $\theta = 30.6^\circ$ changes as perturbation size increases. This indicates how weak the coupling should be for the theory to hold, i.e., the maximum size of α allowed in (5.2)–(5.3). As $|\Delta v|$ increases, linearity is lost in three different respects: (i) (for a given θ) the phase difference $\Delta \phi$ is no longer proportional to $|\Delta v|$; (ii) positive and negative perturbations give different contributions; (iii) strong nonlinear effects appear in the perturbed limit cycle; spikes can be deleted, as in Figure 7(c) for $\Delta v = -7.5 \text{ mV}$, and spikes or entire bursts can be added, as in Figure 7(d) for $\Delta v = +7.5 \text{ mV}$. The threshold for “small” perturbations may depend on θ and on specific parameters (see comment in section 5.4). In Figure 7(e) we show the estimate of Z at $\theta = 168.8^\circ$; for negative perturbations, linearity is maintained up to $h = -31.6 \text{ mV}$ but is lost at around $h = +7 \text{ mV}$ for positive perturbations. This could imply that antiphase solutions are more robust than in-phase solutions; see the discussion in section 5.4.

We observe three distinct regions in the PRC. During the burst, sensitivity to each spike is evident, with maximal sensitivity to the final one. After this, there is a period of relative insensitivity, followed by a region dominated by a large smooth phase advance. This third region is largely unaffected by changes in bursting frequency, duty cycle, or number of APs in the burst, since the end of the cycle remains very similar; see also Figures 10(b₁)–(b₂) and 11(a₁)–(a₂), below. A PRC of similar form was derived experimentally by Delcomyn [57] for the locust flight CPG.

The inability of the infinitesimal PRC to represent the loss or addition of spikes and bursts follows from the tight coiling of the limit cycle of (2.1) in phase space. This implies that the isochronic manifolds $M_{\tilde{\phi}}$ are globally convoluted, and (moderate) perturbations exist that can skip or repeat spikes by taking the perturbed voltage $v + \Delta v$ from one spike to a point near another. Hence phase reduction must be used with care, although if the number of spikes within a burst is large compared to the burst period, skipping or adding spikes will not greatly affect the averaged coupling functions H_{ji} of (5.5)–(5.6). In what follows synaptic conductances are small enough to remain within the range of infinitesimal PRC validity.

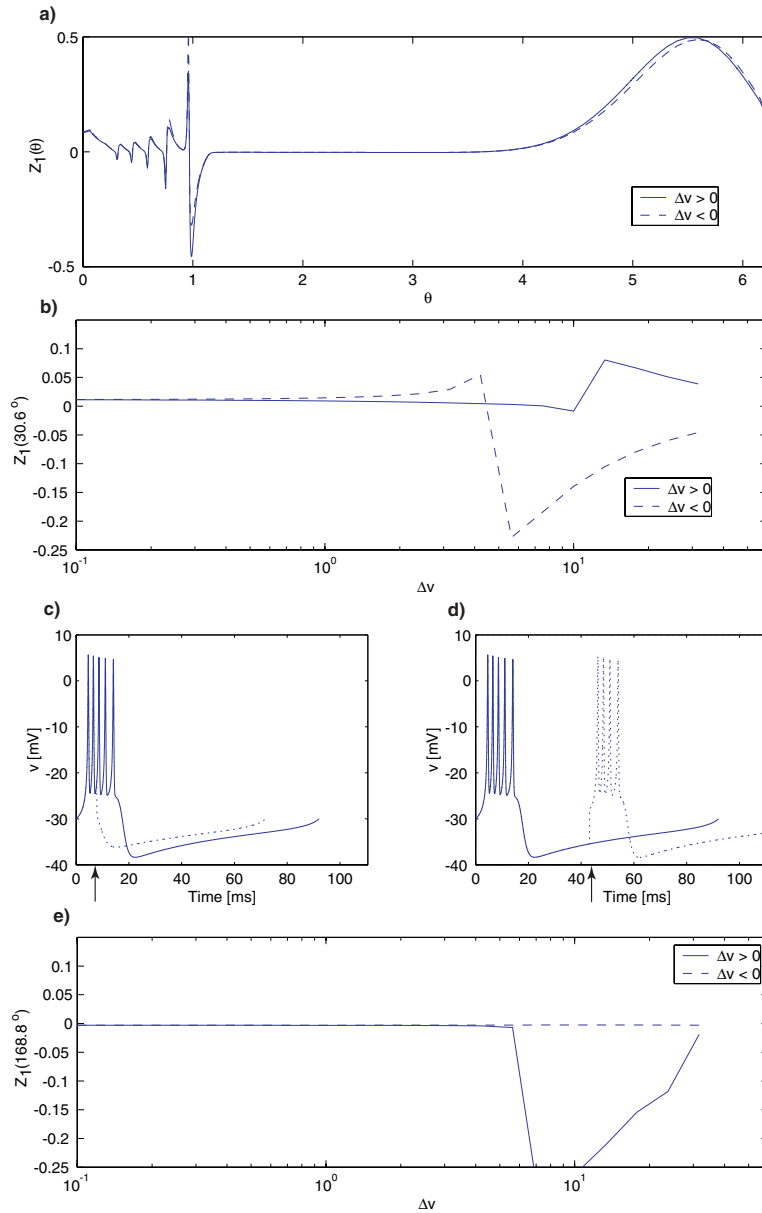


Figure 7. The infinitesimal PRC of (2.1) for the standard parameter set of Tables 1–2. (a) $Z_1(\phi)$ computed with a perturbation of $\Delta v = \pm 0.08$ mV (solid and dashed, respectively). (b) $Z_1(30.6^\circ)$ as a function of perturbation size and sign: $\Delta v > 0$ (solid), $\Delta v < 0$ (dashed). (c) The unperturbed (solid) and perturbed (dashed) cycles with $\Delta v = -7.5$ mV applied at $\theta = 30.6^\circ$ (arrow): Spikes can be removed. (d) The unperturbed (solid) and perturbed (dashed) cycles for $\Delta v = +7.5$ mV applied at $\theta = 168.8^\circ$ (arrow): Spikes or entire bursts, as here, can be added. (e) $Z_1(168.8^\circ)$ as a function of perturbation size and sign: $\Delta v > 0$ (solid), $\Delta v < 0$ (dashed).

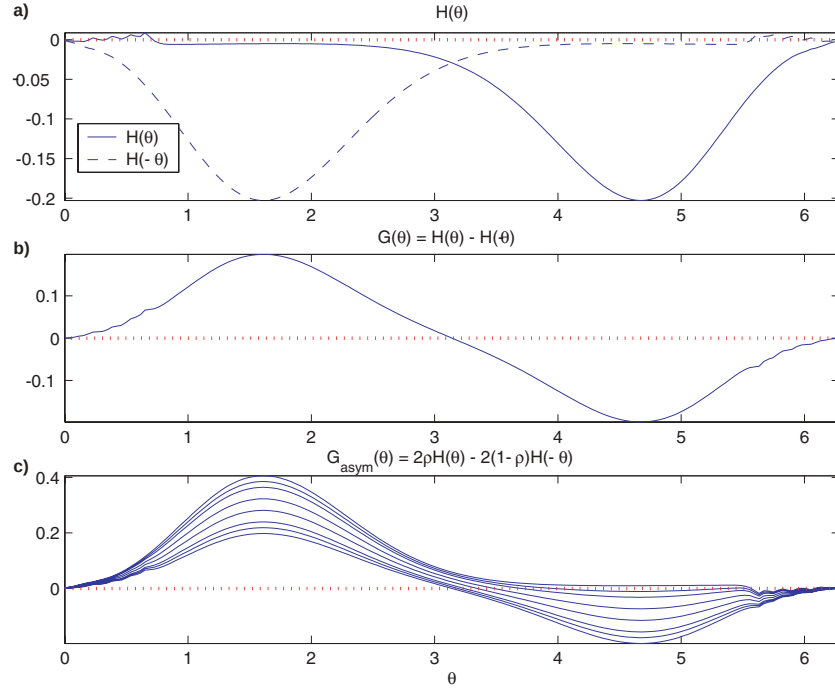


Figure 8. (a) The averaged coupling function $H(\theta)$ (solid) for an inhibitory synapse; $H(-\theta)$ also shown (dash-dotted). (b) The phase difference coupling function $G(\theta) = H(\theta) - H(-\theta)$. Note that $G'(0) > 0 > G'(\pi)$. (c) Asymmetric coupling: $G(\theta) = 2\rho H(\theta) - 2(1-\rho)H(-\theta)$; ρ varies from 0 to 0.5 from top to bottom curve.

5.3. Coupled bursting neurons. We now return to study coupled bursters in a phase-reduced setting. From Figures 2(c), (d), two types of coupling appear: motoneurons are unilaterally driven by the CPG neurons, whereas CPG neurons are bi- or trilaterally coupled.

Fast motoneurons, unilateral coupling. Allowing for different intrinsic frequencies $\omega_0 + \epsilon_j$, the PRC and averaging theory of section 5.1 lead to phase-reduced dynamics for a CPG neuron ψ_1 and a fast motoneuron ψ_2 of the form

$$(5.9) \quad \dot{\psi}_1 = \epsilon_1, \quad \dot{\psi}_2 = \epsilon_2 + \alpha H(\psi_2 - \psi_1).$$

Phase locking occurs when $\dot{\theta} = \dot{\psi}_1 - \dot{\psi}_2 = 0$:

$$(5.10) \quad \epsilon_1 - \epsilon_2 = \alpha H(\psi_2 - \psi_1).$$

As shown in Figure 8(a) H is almost always negative for the nominal case. Phase reduction therefore predicts that locking can only occur if $\epsilon_1 < \epsilon_2$, i.e., when the intrinsic CPG bursting frequency is lower than that of the motoneurons. This is illustrated in Figures 14(a), (b) below, where $f_{\text{CPG}} = 10.5 \text{ Hz} < f_{\text{D}_f} = 15.4 \text{ Hz}$. On the contrary, phase locking does not occur when $f_{\text{CPG}} = 18.1 \text{ Hz} > f_{\text{D}_f} = 15.4 \text{ Hz}$; see Figures 14(c), (d). Indeed, inhibitory coupling delays the bursts; therefore, it is natural to expect that for unilateral coupling the driving neuron should be slower than the follower. If that were not the case, the already slower “follower” would be further slowed, preventing 1:1 phase locking. In fact, for the simulations

reported here, 2:1 locking occurs; see Figure 14(d). This could explain the phenomenon of “double bursting” described in [58].

Thus, provided their bursting frequencies are chosen appropriately, fast motoneurons follow CPG neurons and so need not be explicitly included in the reduced analysis of locomotion rhythms to follow. Similarly, slow motoneurons need not be included in a reduced phase description, since they also phase lock via synaptic depression and their duty cycles are determined by those of the CPG neurons (cf. Figures 3(i), 6(c)).

CPG neurons, mutual coupling. For mutual coupling between two identical CPG neurons the reduced phase equations (5.5) become

$$(5.11) \quad \dot{\psi}_1 = \alpha H(\psi_1 - \psi_2), \quad \dot{\psi}_2 = \alpha H(\psi_2 - \psi_1)$$

(since $\alpha_{12}H_{12} = \alpha_{21}H_{21}$, here we may drop the subscripts), and subtracting these we may further reduce to a single scalar ODE for the phase difference $\theta = \psi_1 - \psi_2$:

$$(5.12) \quad \dot{\theta} = \alpha[H(\theta) - H(-\theta)] \stackrel{\text{def}}{=} G(\theta).$$

Fixed points of (5.12) occur at $H(\theta) = H(-\theta)$, and since H is 2π -periodic, we have $G(\pi) = \alpha[H(\pi) - H(-\pi)] = \alpha[H(\pi) - H(\pi)] = 0$ as well as $G(0) = 0$, implying that, *regardless of the form of H* , (exact) in-phase and antiphase solutions exist; see Figure 8(b). Note that, for $\bar{\theta} = 0$ and π , the equations in (5.11) become $\dot{\psi}_1 = \dot{\psi}_2 = \alpha H(\bar{\theta})$, so that, unless $H(0) = 0$ and/or $H(\pi) = 0$, coupling does change the common frequency $\dot{\phi} = \omega_0 + \dot{\psi}_i$ of the units, even when phase locking occurs.

The stability of fixed points $\bar{\theta}$ of (5.12) is determined by $\frac{\partial G}{\partial \theta}|_{\bar{\theta}} = 2\alpha H'(\bar{\theta})$. As expected, for inhibitory coupling $\alpha H'(0) > 0 > \alpha H'(\pi)$ (Figure 8(b)), so the in-phase solution $\bar{\theta} = 0$ is unstable and the antiphase solution $\bar{\theta} = \pi$ is stable. Stability of the “full” two-phase system (5.11) is determined by the eigenvalues of the 2×2 matrix obtained by linearizing at $\psi_1 - \psi_2 = \bar{\theta}$:

$$(5.13) \quad \alpha \begin{bmatrix} H'(\bar{\theta}) & -H'(\bar{\theta}) \\ -H'(\bar{\theta}) & H'(\bar{\theta}) \end{bmatrix};$$

these are 0 and $2\alpha H'(\bar{\theta})$ with eigenvectors $(1, 1)^T$ and $(1, -1)^T$, respectively. Hence the dynamics is only neutrally stable to perturbations that advance or retard the phases of both units equally, but the antiphase solution is asymptotically stable to perturbations that disrupt the relative phase $\psi_1 - \psi_2$, as indicated by Figure 3.

Finally we consider asymmetric coupling of the type shown in Figure 2(d), which will be discussed further in the context of phase lags. The phase equations for two identical neurons become

$$(5.14) \quad \begin{aligned} \dot{\psi}_1 &= 2\rho\alpha H(\psi_1 - \psi_2), \\ \dot{\psi}_2 &= 2(1 - \rho)\alpha H(\psi_2 - \psi_1), \end{aligned}$$

where ρ measures the degree of asymmetry, $\rho = \frac{1}{2}$ being the symmetric case (5.11). The phase difference is then governed by

$$(5.15) \quad \dot{\theta} = \alpha[2\rho H(\theta) - 2(1 - \rho)H(-\theta)] \stackrel{\text{def}}{=} G_{\text{asym}}(\theta).$$

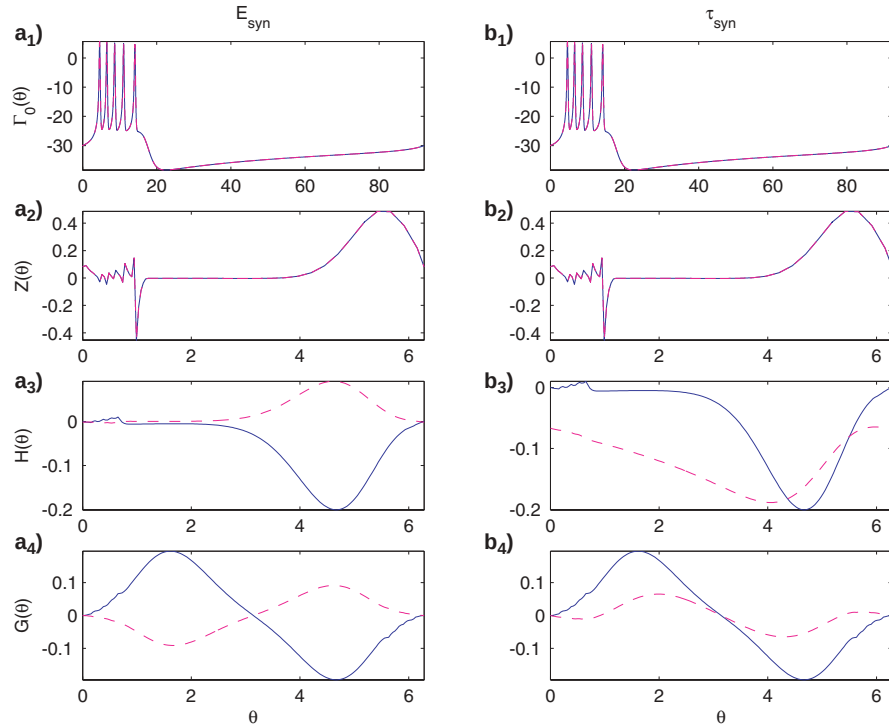


Figure 9. Dependence of the PRC $Z(\theta)$, the averaged coupling function $H(\theta)$, and the “difference” function $G(\theta)$ on synaptic parameters. Panels (a₁)–(a₄) show dependence on synapse type: Inhibitory $GABA_A$ (solid) and excitatory AMPA (dashed). Panels (b₁)–(b₄) show dependence on synapse timescale: Regular inhibitory $GABA_A$ (solid) and slower inhibitory $GABA_A$ (dashed) with $\alpha = 500$, $\beta = 0.018$.

Plots of $G_{\text{asym}}(\theta)$ for different values of $\rho \in [0, 0.5]$ are shown in Figure 8(c). Note that the interior zero occurs at increasing values of $\theta > \pi$, representative of a shifted “antiphase” solution. In this case the in-phase solution remains at $\theta = 0$, but this is a special property of the averaged functions H , which vanish at $\theta = 0$.

5.4. Parameter dependence of the PRC and averaged coupling functions. We will now investigate the effect of certain parameters on the shapes of the functions Z , H , and G . From (5.6), we see that the only parameter that can be factored out is the coupling strength $\alpha_{ji} = \bar{g}_{\text{syn},ji}$, which scales Z , H , and G ; the other parameters change their forms more generally.

Changing from inhibitory $GABA_A$ to excitatory AMPA synapses implies changing α , β , and $E_{\text{syn}}^{\text{pre}}$ in (4.1) (cf. caption to Figure 3). As a result, H becomes almost always positive (Figure 9(a₃)), but more importantly $G'(\pi)$ becomes positive and $G'(0)$ negative, making the antiphase solution unstable and the in-phase solution stable; see Figure 9(a₄). Note that $Z(\theta)$ is unaffected by this change. Even more interesting is the effect of a slower time scale on *inhibitory* synapses. Under a ten-fold increase ($\alpha = 500$, $\beta = 0.018$) the in-phase solution becomes *stable* and two new unstable solutions appear in a pitchfork bifurcation; see Figure 9(b₄).

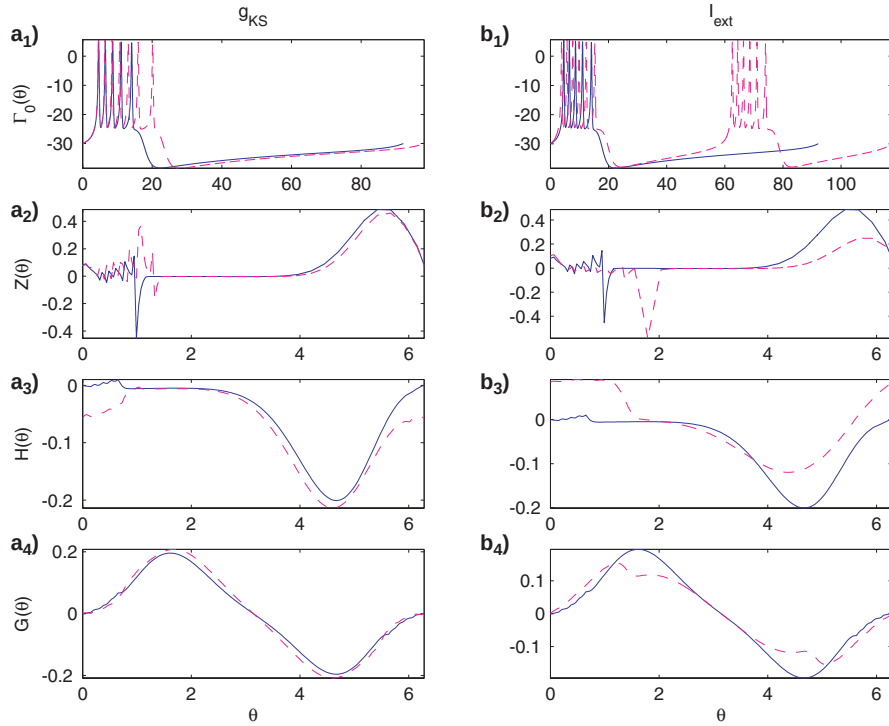


Figure 10. Dependence of the PRC $Z(\theta)$, the averaged coupling function $H(\theta)$, and the function $G(\theta)$ on $\bar{g}_{KS}$ and I_{ext} . Panels (a₁)–(a₄) show dependence on the maximal conductance $\bar{g}_{KS}$: Nominal case of Tables 1 and 2 (solid) and decreased value: $\bar{g}_{KS} = 0.16$ (dashed). Panels (b₁)–(b₄) show dependence on the external current: Nominal case (solid) and increased current $I_{ext} = 36.5$ (dashed).

Changing the maximal conductance $\bar{g}_{KS}$ can affect the bursting frequency, the duty cycle, and the number of APs per burst. In this case, even though bursting frequency and AP numbers are significantly modified (Figure 10(a₁)), and accordingly $Z(\theta)$ and $H(\theta)$ (Figures 10(a₂)–(a₃)), the net effects on the function $G(\theta)$ largely average out. Results not shown indicate that increasing the maximal conductance to $\bar{g}_{KS} = 0.25$ can introduce extra spikes, violating the infinitesimal PRC assumption; this case is discussed further as a finite perturbation below. Changing I_{ext} yields analogous results; e.g., in spite of a substantial change in bursting frequency (Figure 10(b₁)), the final form of $G(\theta)$ is not greatly modified (Figure 10(b₄)).

Finally we show that, even in cases in which spikes are lost or added, the infinitesimal PRC undergoes sharp transitions (Figures 10(b₁)–(b₂); cf. Figure 7(b)), and the averaged coupling function also changes significantly; both it and the difference function $G(\theta)$ retain similar forms near $\theta = \pi$ (Figures 11(b₃), (b₄)). Hence we may still deduce stability information regarding antiphase solutions from the reduced description. Indeed, Figure 7(e) shows that $Z_1(\phi \approx \pi)$ is insensitive to the negative perturbation magnitude up to $\Delta v \approx 30$ mV.

5.5. A phase-reduced model of the CPG. Extension of the above reduction of a mutually coupled pair to the network of six CPG neurons in Figure 2(c) is immediate. We again assume identical units, but as noted in section 4 below Table 1, employ different synaptic strengths

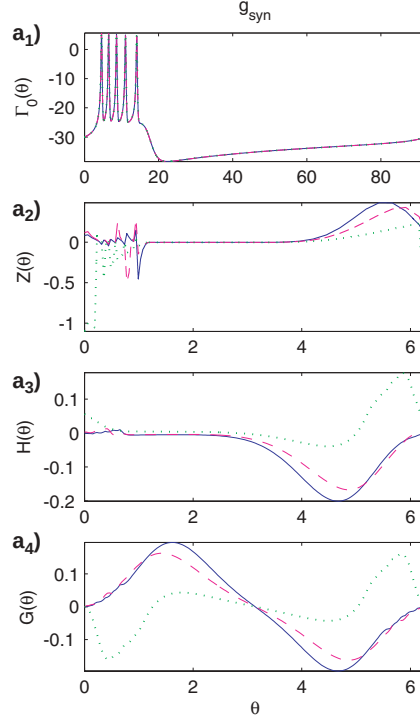


Figure 11. Dependence of the (infinitesimal) PRC $Z(\theta)$, the coupling function $H(\theta)$, and the function $G(\theta)$ on the size of the perturbation Δv . (a₁)–(a₄): Nominal case $\Delta v = -0.08$ (solid), medium size $\Delta v = -1$ (dashed), and large $\Delta v = -5$ (dotted). Note that form of Z , H , and G near $\theta = \pi$ remains similar.

so that all units receive the same net input at steady state. Thus all contralateral connections and ipsilateral connections from units 2 and 5 to 1, 3, 4, and 6 are set at $\bar{g}_{\text{syn}}$, and ipsilateral connections from 1, 3, 4, and 6 to 2 and 5 are set at $\bar{g}_{\text{syn}}/2$. This leads to the following set of six phase equations:

$$\begin{aligned}
 \dot{\psi}_1 &= \bar{g}_{\text{syn}} H(\psi_1 - \psi_4) + \bar{g}_{\text{syn}} H(\psi_1 - \psi_5), \\
 \dot{\psi}_2 &= \frac{\bar{g}_{\text{syn}}}{2} H(\psi_2 - \psi_4) + \bar{g}_{\text{syn}} H(\psi_2 - \psi_5) + \frac{\bar{g}_{\text{syn}}}{2} H(\psi_2 - \psi_6), \\
 \dot{\psi}_3 &= \bar{g}_{\text{syn}} H(\psi_3 - \psi_5) + \bar{g}_{\text{syn}} H(\psi_3 - \psi_6), \\
 \dot{\psi}_4 &= \bar{g}_{\text{syn}} H(\psi_4 - \psi_1) + \bar{g}_{\text{syn}} H(\psi_4 - \psi_2), \\
 \dot{\psi}_5 &= \frac{\bar{g}_{\text{syn}}}{2} H(\psi_5 - \psi_1) + \bar{g}_{\text{syn}} H(\psi_5 - \psi_2) + \frac{\bar{g}_{\text{syn}}}{2} H(\psi_5 - \psi_3), \\
 \dot{\psi}_6 &= \bar{g}_{\text{syn}} H(\psi_6 - \psi_2) + \bar{g}_{\text{syn}} H(\psi_6 - \psi_3).
 \end{aligned}
 \tag{5.16}$$

We first observe that there exist solutions in which the tripods 1, 2, 3 and 4, 5, 6 remain internally in-phase. Indeed, seeking (possibly time-dependent) solutions of the form $\psi_1 = \psi_2 = \psi_3 \equiv \psi_L(t)$, $\psi_4 = \psi_5 = \psi_6 \equiv \psi_R(t)$, (5.16) collapses to the pair of equations

$$\dot{\psi}_L = 2\bar{g}_{\text{syn}} H(\psi_L - \psi_R) \quad \text{and} \quad \dot{\psi}_R = 2\bar{g}_{\text{syn}} H(\psi_R - \psi_L),
 \tag{5.17}$$

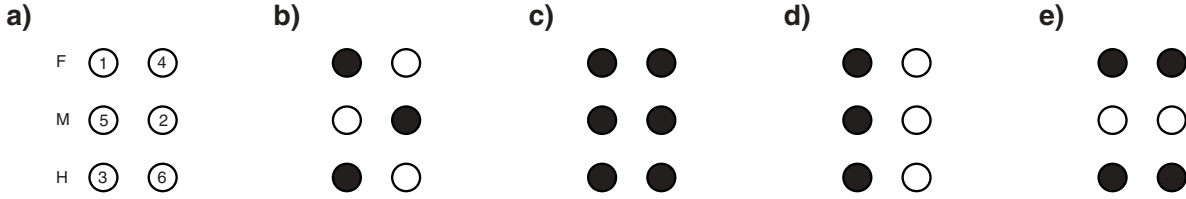


Figure 12. Some gaits produced by the hexapedal network of Figure 2(c). (a) Numbering convention for CPG neurons; (b) tripod; (c) pronk; (d) pace; (e) gallop.

and the arguments used above may be applied to conclude that $\psi_R = \psi_L + \pi$ and $\psi_R = \psi_L$ are fixed points of the $\psi_L - \psi_R$ tripod phase difference equation, again regardless of the form of H . Stability in the full six-dimensional (reduced) phase space is obtained from the 6×6 matrix obtained by linearizing (5.16),

$$(5.18) \quad \bar{g}_{\text{syn}} \begin{bmatrix} 2H' & 0 & 0 & -H' & -H' & 0 \\ 0 & 2H' & 0 & -H'/2 & -H' & -H'/2 \\ 0 & 0 & 2H' & 0 & -H' & -H' \\ -H' & -H' & 0 & 2H' & 0 & 0 \\ -H'/2 & -H' & -H'/2 & 0 & 2H' & 0 \\ 0 & -H' & -H' & 0 & 0 & 2H' \end{bmatrix},$$

where the derivatives H' are evaluated at the appropriate (constant) phase differences. The antiphase tripod $\psi_L - \psi_R = \pi$ gives one zero eigenvalue with “equal phase” eigenvector $(1, 1, 1, 1, 1, 1)^T$, and the remaining eigenvalues and eigenvectors are as follows:

$$(5.19) \quad \begin{aligned} \lambda &= \bar{g}_{\text{syn}} H' : (1, 0, -1, 1, 0, -1)^T, \\ \lambda &= 2\bar{g}_{\text{syn}} H', m = 2 : (1, -1, 1, 0, 0, 0)^T, \text{ and } (0, 0, 0, -1, 1, -1)^T, \\ \lambda &= 3\bar{g}_{\text{syn}} H' : (1, 0, -1, -1, 0, 1)^T, \\ \lambda &= 4\bar{g}_{\text{syn}} H' : (1, 1, 1, -1, -1, -1)^T. \end{aligned}$$

Since $\bar{g}_{\text{syn}} H'(\pi) < 0$ for the nominal parameters, this again indicates asymptotic stability with respect to perturbations that disrupt the tripod phase relationships; moreover, the system recovers fastest from perturbations that disrupt the relative phasing of the tripods ($\lambda = 4\bar{g}_{\text{syn}} H'$: last entry of (5.19)). Since $\bar{g}_{\text{syn}} H'(0) > 0$ (Figure 8(a)), the in-phase “pronking” gait with all legs in phase is unstable.

Other gaits may be found by appealing to discrete symmetries of the network, as in extensive work by Golubitsky and colleagues (see, e.g., [59]). Although they are not directly relevant to cockroach running, we give two examples, shown schematically in Figure 12 along with the antiphase tripod and pronking gaits.

Pace. An appeal to bilateral symmetry $\psi_1 = \psi_5 = \psi_3 = \psi_L$ and $\psi_4 = \psi_2 = \psi_6 = \psi_R$ also yields two equations of the type (5.11), but with an additional term $H(0)$ in each. Since this cancels in subtracting the equations, in- and antiphase solutions again exist.

Gallop. A slightly more complicated gait is obtained by a subgroup of the symmetry group $\mathcal{D}_6$. Setting $\psi_1 = \psi_4 = \psi_F$, $\psi_5 = \psi_2 = \psi_M$, and $\psi_3 = \psi_6 = \psi_H$, (5.16) collapses to the

three differential equations

$$\begin{aligned}
 \dot{\psi}_F &= \bar{g}_{\text{syn}} [H(0) + H(\psi_F - \psi_M)], \\
 \dot{\psi}_M &= \bar{g}_{\text{syn}} \left[H(0) + \frac{1}{2}H(\psi_M - \psi_F) + \frac{1}{2}H(\psi_M - \psi_H) \right], \\
 \dot{\psi}_H &= \bar{g}_{\text{syn}} [H(0) + H(\psi_H - \psi_M)].
 \end{aligned}
 \tag{5.20}$$

This can be further simplified by seeking solutions $\psi_F = \psi_{FH} = \psi_H$ to obtain

$$\dot{\psi}_{FH} = \bar{g}_{\text{syn}} [H(0) + H(\psi_{FH} - \psi_M)] \quad \text{and} \quad \dot{\psi}_M = \bar{g}_{\text{syn}} [H(0) + H(\psi_M - \psi_{FH})],
 \tag{5.21}$$

which is again an instance of (5.11), admitting in-phase and antiphase solutions. The former is just the pronk noted above, but the antiphase “gallop” is new.

5.6. Phase lags and asymmetric coupling. Nominally identical neural oscillators can display differing cycle periods when isolated [60]. Indeed, phase relationships among coupled oscillators can arise from differences in periods as well as from intersegmental coupling characteristics such as strength, projection span, and degree of symmetry [61, 62]. Sensory inputs, moreover, can alter oscillation periods and coordinate mechanical coupling between limbs or segments; they may even form sensory-central oscillatory loops [60]. For example, experimental evidence indicates that the activation of the depressor muscles in *Blaberus discoidalis* does not occur simultaneously even within the tripod gait regime [37]; activation lags are distributed in a range of 0–60% of the cycle. In section 5.3, Figure 8(c), we saw how asymmetric coupling can induce a phase lag, within an antiphase (stable) solution. Here, we extend the two-oscillator analysis to the hexapedal network.

Keeping the left-right symmetry unbroken, we introduce asymmetric ipsilateral coupling $(1 - \bar{g}_F)$ and $2\bar{g}_F$ between front and middle legs and $(1 - \bar{g}_H)$ and $2\bar{g}_H$ between hind and middle legs, as shown in Figure 2(d). This leads to modified phase equations

$$\begin{aligned}
 \dot{\psi}_1 &= H(\psi_1 - \psi_4) + 2\bar{g}_F H(\psi_1 - \psi_5), \\
 \dot{\psi}_2 &= (1 - \bar{g}_F)H(\psi_2 - \psi_4) + H(\psi_2 - \psi_5) + (1 - \bar{g}_H)H(\psi_2 - \psi_6), \\
 \dot{\psi}_3 &= 2\bar{g}_H H(\psi_3 - \psi_5) + H(\psi_3 - \psi_6), \\
 \dot{\psi}_4 &= H(\psi_4 - \psi_1) + 2\bar{g}_F H(\psi_4 - \psi_2), \\
 \dot{\psi}_5 &= (1 - \bar{g}_F)H(\psi_5 - \psi_1) + H(\psi_5 - \psi_2) + (1 - \bar{g}_H)H(\psi_5 - \psi_3), \\
 \dot{\psi}_6 &= 2\bar{g}_H H(\psi_6 - \psi_2) + H(\psi_6 - \psi_3),
 \end{aligned}
 \tag{5.22}$$

where we have included the overall scaling factor $\bar{g}_{\text{syn}}$ in H . We seek solutions which preserve the alternating tripod gait but exhibit phase lags within it:

$$\begin{aligned}
 \psi_4 &= \psi_1 + \pi, & \psi_5 &= \psi_2 + \pi, & \psi_6 &= \psi_3 + \pi, \\
 \psi_2 &= \psi_1 + \Delta_F, & \psi_5 &= \psi_4 + \Delta_F, & \psi_3 &= \psi_2 + \Delta_H.
 \end{aligned}
 \tag{5.23}$$

(Note that this implies that $\psi_6 = \psi_5 + \Delta_H$, and it automatically ensures phase locking between the tripods: $\dot{\psi}_1 - \dot{\psi}_4 = \dot{\psi}_5 - \dot{\psi}_2 = \dot{\psi}_3 - \dot{\psi}_6 = 0$.) Substituting (5.23) into (5.22) and setting

all time derivatives to zero, we obtain two expressions relating the lags (Δ_F, Δ_H) to the asymmetry parameters $(\bar{g}_F, \bar{g}_H)$:

$$(5.24) \quad \begin{aligned} 2\bar{g}_F H_{\pi-\Delta_F} - (1 - \bar{g}_F) H_{\pi+\Delta_F} - (1 - \bar{g}_H) H_{\pi-\Delta_H} &= 0, \\ \bar{g}_F H_{\pi-\Delta_F} - \bar{g}_H H_{\pi+\Delta_H} &= 0. \end{aligned}$$

In deriving these, we use 2π -periodicity of H , implying that $H(-\pi + \Delta) = H(\pi + \Delta)$, and we adopt the abbreviated notation $H(\pi \pm \Delta) = H_{\pi \pm \Delta}$. Lacking explicit formulae for H , we cannot solve (5.24) analytically, but rearranging the equations to extract $\bar{g}_F, \bar{g}_H$,

$$(5.25) \quad \bar{g}_F = \frac{H_{\pi+\Delta_F} + H_{\pi-\Delta_H}}{\left(2 + \frac{H_{\pi-\Delta_H}}{H_{\pi+\Delta_H}}\right) H_{\pi-\Delta_F} + H_{\pi+\Delta_F}}, \quad \bar{g}_H = \bar{g}_F \frac{H_{\pi-\Delta_F}}{H_{\pi+\Delta_H}},$$

we can find semiexplicit solutions numerically. Typical slices of the functions $\bar{g}_F(\Delta_F, \Delta_H)$ and $\bar{g}_H(\Delta_F, \Delta_H)$ are shown in Figures 13(a), (b). Solutions of (5.25) yield the relative synaptic strengths required to achieve given phase lags: for example, Figure 13(a) indicates that setting $\bar{g}_F \approx 0.6865$, $\bar{g}_H \approx 0.1255$ will give $\Delta_F \approx 15^\circ$, $\Delta_H \approx 35^\circ$. Figures 13(c), (d) show that for these coupling strengths the equations in (5.22) indeed lock into a tripod gait with $\Delta_F = 14.96^\circ$ and $\Delta_H = 35.06^\circ$. Finally, Figures 13(e), (f) show that the lags predicted by the phase-reduced theory agree extremely well with those obtained from direct numerical simulations of the full network of (2.1) and (4.1)–(4.2), over a range of biophysically relevant coupling strengths.

5.7. Comparison of phase-reduced and full CPG models. We have already noted (Figures 13(e), (f)) that the phase-reduced model (5.22) and the phase lag/coupling strength relations (5.25) derived from it can predict lags observed in the full network model (2.1), (4.1)–(4.2). We also noted that the symmetric phase-reduction (5.16) correctly captures the stability of the antiphase and instability of the in-phase solutions for the standard parameter set.

We may go further and observe that the phase-reduced model predicts a timescale for antiphase locking to occur between pairs of oscillators (or, indeed, between left and right tripods). Specifically, linearizing (5.12) at $\theta = \pi$ and using the slope $G'(\pi) \approx -0.15/ms$ from Figure 9(a₄) (solid line), we expect locking to be accomplished within one bursting cycle, and the data of Figures 3, 4, and 5 indicates that this is indeed correct. We also recall that the simple two-oscillator analysis of section 5.3 ((5.9)–(5.10)) predict that pairs of unilaterally coupled CPG and motoneurons should phase lock more readily when the uncoupled frequencies of the former are lower than those of the latter. Figures 14(a)–(d) confirm this with direct simulations of the full network (2.1), (4.1)–(4.2).

The averaged coupling functions obtained in phase reduction also suggest considerable robustness of the (stable) antiphase solution. The multiparametric analyses of section 5.4, illustrated in Figures 9 and 11, show that the slope of G near $\theta = \pi$ remains essentially unchanged even when burst properties are significantly modified (existence of the antiphase fixed point is ensured for any H for the symmetric network of Figure 2(c), as noted in section 5.3). This robustness is implicit in Figure 6, which shows that antiphase tripod solutions of (2.1), (4.1)–(4.2) were found over a substantial domain of a four-dimensional parameter space.

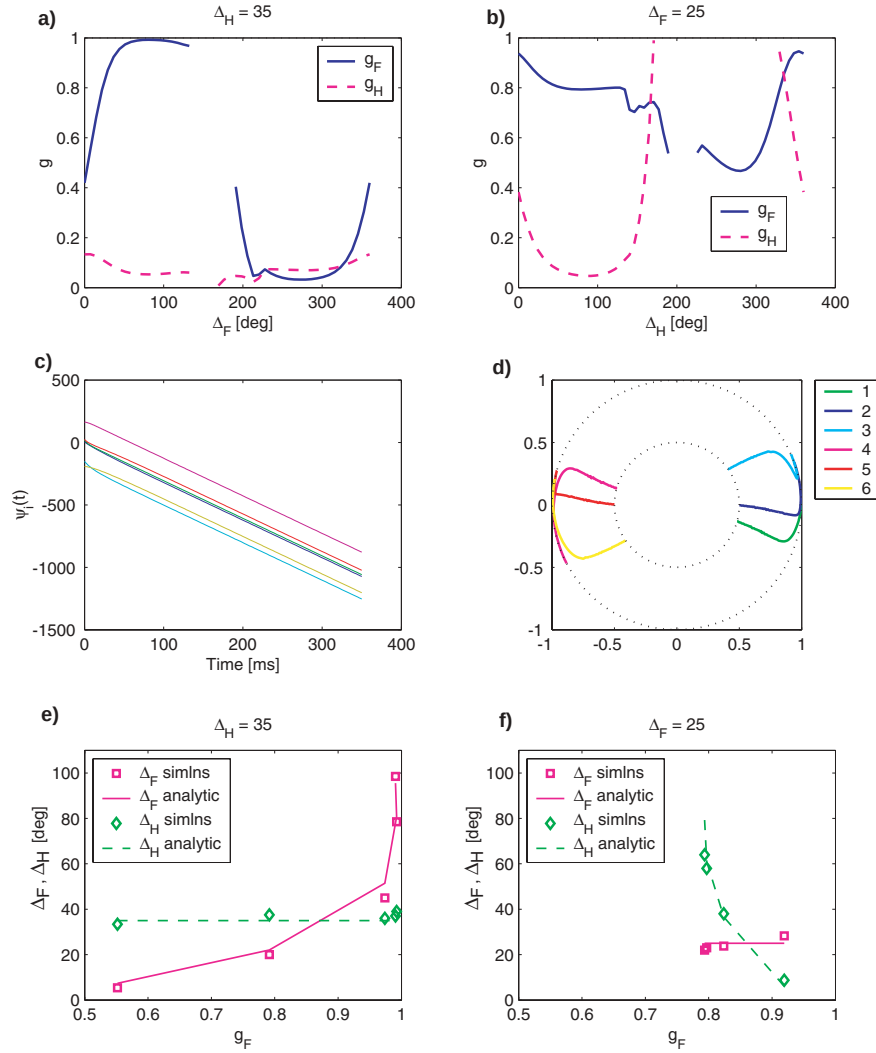


Figure 13. Phase lagged tripod solutions for the hexapedal network of Figure 2(d). Top panels show $\bar{g}_F$ (solid) and $\bar{g}_H$ (dashed) as functions of (a) Δ_F for fixed $\Delta_H = 35^\circ$, and (b) Δ_H for fixed $\Delta_F = 25^\circ$, computed from (5.25). Values $\bar{g} > 1$ invert the synapse’s “sign” and are invalid, causing broken curves. Panels (c), (d) show time histories and polar plots of the six phases with $\bar{g}_F = 0.6865$, $\bar{g}_H = 0.1255$, computed from phase-reduced model (5.22). Phases start at $t = 0$ on the outer circle and end at $t = 350$ msec on the inner circle, having attained the desired lags. Panels (e), (f) compare predictions of phase-reduced theory with direct network simulations using (2.1), (4.1)–(4.2): Phase lags Δ_F and Δ_H from (5.25) are shown as solid and dashed lines, and from (2.1), (4.1)–(4.2) as squares and diamonds, respectively. In panel (a), $\bar{g}_F, \bar{g}_H$ are chosen to keep Δ_H fixed, and in (f), to keep Δ_F fixed.

Predictions of in-phase solutions with *inhibitory* synapses are even more interesting and potentially delicate. Recalling Figures 9(a₄) and (b₄), we expect stable in-phase solutions for excitatory synapses but *also* for slow inhibitory synapses. That this indeed occurs in the full network is shown in Figures 14(e)–(h): (e) and (f) show anti- and in-phase solutions with excitatory coupling, (g) an in-phase solution which coexists with an antiphase solution, and

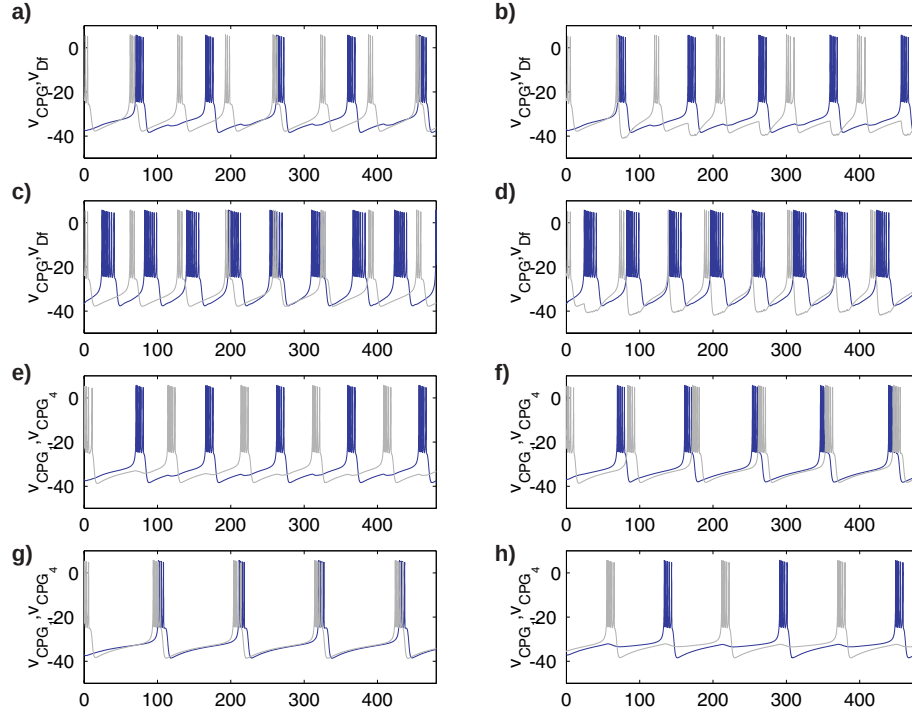


Figure 14. Membrane voltages of CPG neurons in the hexapedal network of Figure 2(c). Panels (a), (c) show uncoupled CPG (dark) and fast motoneurons D_f (grey) with $f_{CPG} = 10.5 \text{ Hz} < f_{D_f} = 15.4 \text{ Hz}$ and $f_{CPG} = 18.1 \text{ Hz} > f_{D_f} = 15.4 \text{ Hz}$, respectively, and (b), (d) show that unidirectional coupling causes 1:1 phase locking in the first case, but not the second, which yields 1:2 locking. Panels (e)–(h) show mutually coupled contralateral CPG neurons 1 and 4, indicating antiphase locking with inhibitory coupling (e), in-phase locking with excitatory coupling (f), and coexistence of in-phase (g) and antiphase (h) locking with slow inhibitory coupling, as in Figure 9(b₄); (g) and (h) are obtained for the same parameter values but different initial conditions. Some panels show the effects of transients, and (in the case of (f) and (g)) the relatively slow approach to in-phase solutions.

(h) for slow inhibitory coupling, the latter two solutions being found for identical parameter values but different initial conditions. For such a network both the tripod and the pronk gaits are stable.

6. Conclusions. This paper develops a minimal model for the CPG and representative motoneurons responsible for insect locomotion. We incorporate sufficient biophysical detail to permit appropriate parameter choices and variations to reproduce experimental data, focusing on the cockroaches *Blaberus discoidalis* and *Periplaneta americana*, but we strive for generality and (relative) simplicity. Much current research concerns subcellular details of ionic currents and channels and molecular messengers [63, 64, 65], but despite the ability of “detailed” models to reproduce experimental data (e.g., [66, 7, 67]), their complexity and sensitivity to parameter variations renders them effectively unanalyzable. We believe that massive simulations or experiments alone do not provide global understanding, which profits more from the identification of a few key mechanisms. Thus, our aim is to extract “principles for locomotion” by judicious *selection*, rather than inclusion, of biological data, and in

doing so to provide a flexible and tractable mathematical framework within which biological hypotheses can be investigated and novel experiments suggested.

The bursting model (2.1) developed in the preceding paper [1], along with a single equation (4.1) describing synaptic dynamics, is used as the basic subunit to describe the neural architecture of cockroach locomotion. The overall model, which is a “cartoon” representing only a single power stroke (depressor) output per leg, comprises six coupled CPG (inter)neurons, six fast motoneurons, and six slow motoneurons. With appropriate parameter choices, all 18 neurons can be described by the same “minimal” ODE (2.1), and we show how a variety of behaviors, encompassing the range observed in the animals, can be achieved by varying two control parameters separately in the CPG and motoneurons. Since motoneurons are entrained, external currents to CPG interneurons (presumably deriving from higher brain areas and proprioceptive feedback) set the stepping frequency, and a CPG conductance primarily determines the duty cycle. Numbers of APs of fast motoneurons and spike rates of slow motoneurons can be separately adjusted by their external currents and conductances, thereby determining muscle forces in coarse and fine manners.

Finally we show how to prove existence, and investigate stability and phase relationships, of gait patterns through an additional reduction using PRCs and averaging theory. This collapses some 60 ODEs of the hexapedal model to six equations for “leg phases” (5.16) and shows that a single network architecture produces a variety of gaits, whose stability properties are primarily determined by the magnitudes and signs of synaptic conductances. We show that the phase-reduced models reproduce the behaviors of the full hexapedal model remarkably well; in particular, Figures 13(e), (f) show phase lags predicted to better than 5% accuracy over a substantial parameter range. It also suggests that further questions regarding how gaits and their stability depend upon neuronal and synaptic parameters will be accessible via the coupling functions H_{ji} of (5.5)–(5.6).

This study, which builds upon earlier work on conservative mechanical and simple actuated models [17, 18, 12, 19, 20], is another step toward integrated neuromechanical models for legged locomotion. In future work we will couple the CPG model developed here to models of muscles and body-limb mechanics and introduce reflexive feedback. In addition to questions on the dynamics and stability of natural gaits such as the double tripod employed by *Blaberus*, and the roles of intrinsic neural parameters and reflexive and reflexive feedback in the CPG, this will allow investigation of questions such as how *Periplaneta* switches to high speed bipedal locomotion [16] and how animals adjust their gaits to quadrupedal patterns within few steps following middle leg amputation [68, pp. 95–99]. Our framework is sufficiently flexible to allow for different numbers of legs and/or motoneurons, for proprioceptive reflexes and CNS feedforward control, as well as for more detailed models of CPG circuitry, and we anticipate that reduced-phase models, with appropriate modifications to PRCs and coupling functions, will continue to provide analytical understanding of such generalized models. Indeed, they hold promise that a CPG model can be coupled to a simple mechanical model to form an integrated neuromechanical system, all in less than 10–15 ODEs.

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