Investigating the role of low level reinforcement reflex loops in insect locomotion

Clarissa Goldsmith[1], Roger Quinn[2], and Nicholas Szczecinski[1]

[1] Department of Mechanical and Aerospace Engineering, West Virginia University, Morgantown USA [2] Department of Mechanical and Aerospace Engineering, Case Western Reserve University, Cleveland USA

Abstract

This work details the development of a neurorobotic model of the stick insect femur-tibia and coxa-trochanter joint control networks in order to investigate how reinforcement mechanisms (i.e. `positive feedback') may give rise to stepping that can be controlled by simple descending commands from the head ganglia. We describe the development of our model using mappings of the non-spiking interneuron (NSI) joint control network for the extensor muscle of the femur-tibia joint. We extrapolated connectivity for the femur-tibia flexor based on known biological responses, then combined this model with a stepping controller to control a robotic limb. We present experimental data from selectively and asymmetrically inhibiting the network's sensory afferents corresponding to the femoral chordotonal organ (fCO) and observing the reflexes of the simulated joint to a ramp-and-hold-and-release stimulus in mechanically coupled and decoupled scenarios. Inhibiting the network's flexion position and velocity afferents enacts a reflex reversal in the FTi joint from the resistance reflex (RR) to the active reaction (AR) in response to joint flexion.
We also present the results of the network when controlling a robotic limb to describe the effects of this `reflex reversal' and additional descending inputs on stepping. Our findings suggest that descending interneurons, which receive load feedback, can act upon motor neurons, pattern generators, and sensory afferents together to sustain steady locomotion, which can be initiated or halted by brief, transient descending signals from the head ganglia. Using these reinforcement mechanisms, we are able to produce and maintain rhythmic stepping in a robotic limb. Removing portions of this feedback loop or creating a large enough disruption halts stepping. We conclude by discussing why the nervous system might control joint behavior in this manner, as well as how to apply these findings to generalized nervous system understanding and improved robotic control.

Introduction

One feature of the nervous system that makes animals so adaptable is their ability to process sensory information in a task- or context-dependent way. One example of this processing is the apparent "reflex reversal" of insect joint control reflexes. Stretching of the femoral chordotonal organ (fCO) (signifying joint flexion) in an insect's femur-tibia (FTi) joint will result in:

- A **resistance reflex (RR)** that attempts to halt the joint motion while standing.
- An **active reaction (AR)** where the muscles allow the flexion to proceed while actively walking.

Several studies have hypothesized the importance of the groups of non spiking interneurons (NSI) between the sensory neurons and the slow extensor tibiae motorneuron (SETi) for this behavior (Sauer et al. 1997, Driesang et al. 1996). They suggest that the nervous system may switch between the RR in the resting state and the AR in the active state by selectively inhibiting or disinhibiting fCO afferent cells.

What mechanisms in the nervous system could cause modulation of the sensory afferents? Several studies seem to indicate the importance of populations of descending dorsal unpaired median (desDUM) neurons, a major cellular source of octopamine (homolog to noradrenaline) in the ganglial ganglion. Octopamine has previously been found to modulate the activity of sensory organs such as the fCO (Braunig et al. 1998, Ramirez et al. 1993, Matheson et al. 1997), as well as increasing the likelihood of reflex reversal (Büschges et al. 1993, Stolz et al. 2019). It seems reasonable to assume that octopamine from the desDUM neurons contributes, in part, to reflex reversal in the FTi joint. Octopamine has also been observed to play a part in stimulating locomotion (Emanuel et al. 2020). If octopamine increases walking activity in the thoracic ganglion, then the desDUM neurons potentially also activate the CPGs, and perhaps the MNs directly as well.

The exact inputs to the desDUM are unknown. Studies have shown they recieve input when the abdomen, antennae, or parts of the legs were mechanically stimulated (Mentel et al. 2008, Stolz et al. 2019). This behavior points toward the desDUM neurons receiving inputs corresponding to the strain of the leg, likely from campaniform sensilla (CS) load sensors.

To investigate how these specific lower level systems give rise to walking, we present a neural model based on circuits that control insect FTi and CTr joints and used it to control a robotic limb that is dynamically similar to the insect. **Our findings suggest that inhibiting the flexion position and velocity sensory afferents can cause a transition from a resistance reflex (RR) to an active reaction (AR) response to fCO elongation.** We also explored the higher level mechanisms the nervous system might use to transition into the AR in the manner we describe, as well as initiate and sustain walking. We demonstrate that our network can initiate, maintain, and halt stepping in the robotic limb with a combination of sparse descending commands mediated through a neuron representing populations of desDUM in the insect and biologically encoded strain feedback. **These results support our hypothesis that two `positive feedback' mechanisms, i.e. the AR and load feedback reinforcing stepping through desDUM excitation, are each necessary but insufficient to maintain locomotion.**

Methods

Most neurons were modeled as non-spiking leaky integrators:

$$C_m \frac{dV_m}{dt} = I_{leak} + I_{syn} + I_{app} \quad (1)$$

where:
I_{app} = external stimulus V_m = membrane voltage
G_m = membrane conductance C_m = membrane capacitance

$$I_{leak} = \sum_{i=1}^{n} G_{m_i} \cdot (E_i - V) \qquad I_{syn} = \sum_{j=1}^{n} G_{ij} \cdot (E_{ij} - V)$$

$$G_{ij} = G_{max_{ij}} \cdot \begin{cases} 1 & \text{if } V_{pre} > E_{ij} \\ \frac{V_{pre} - E_{ij}}{E_{ij} - E_{ij}} & \text{if } E_{ij} \le V_{pre} \le E_{ij} \\ 0 & \text{if } V_{pre} < E_{ij} \end{cases}$$

$G_{max_{ij}}$ = max synaptic conductance V_{pre} = membrane voltage
E_{ij} = synaptic threshold E_{ij} = synaptic saturation

Sensory neurons and motorneurons utilize integrate-and-fire model for spiking neurons:
Follows Eq. (1) with condition: if $V = \theta$ then $V(t) \rightarrow E_r$
where: θ = threshold value

When a neuron spikes, G_s set to G_{max} then decays according to:

$$\tau_s \frac{dG_s}{dt} = -G_s$$

where: τ_s = synapse time constant

Joint mechanics governed by equation of motion:

$$J \cdot \ddot{\theta} = \tau_{ext} + \tau_{flex} - k_{spring} \cdot \theta$$

where:
J = limb moment of inertia k_{spring} = joint parallel elasticity
$\tau_{ext, flex}$ = torques determined by software to drive joint at speeds and to positions indicated by adapters

Methods

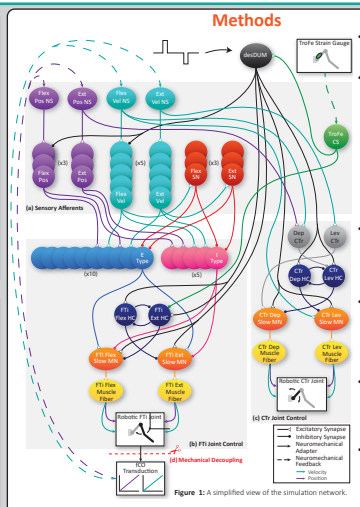

Figure 1: A simplified view of the simulation network.

- Simulation constructed in Animatlab 2 (Cofer et al. 2010).
- Higher level stimulus excites the **desDUM neuron**, which initiates walking by:
  - Exciting the pattern generating networks and motor-neurons (MN) in each joint
  - Inhibiting sensory afferents to create reflex reversal
- Network receives:
  - Position and velocity feedback from the FTi
  - Strain feedback from the trochantofemur
- E type and I type NSI excited by position and velocity sensory neurons and delayed inhibition from spiking interneurons.
- NSI synapse onto the MNs for the FTi, controlling the mechanical joint via non-spiking slow muscle fiber neurons .
- CTr functions similarly to the FTi, with own set of dual interneurons

- Constructed a mechatronic robot leg for the network to control
  - Includes strain into the network without complex simulation and computation
- Strain gauge is located at the proximal end of the trochanterofemur (**Fig. 2(c)**).
- Strain signal is filtered to mimic the discharge dynamics of campaniform sensilla.

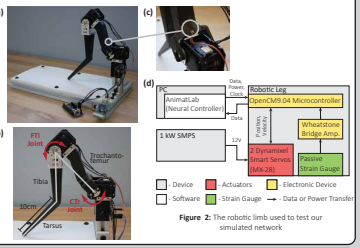

Figure 2: The robotic limb used to test our simulated network

Results

Baseline Behaviors

- Mechanically decoupled case: limb rapidly extends when it receives stimulus corresponding to fCO elongation (flexion).
- Mechanically coupled case: limb oscillates between small angles of flexion and extension throughout the applied stimulus.

Both of these behaviors resemble the RR.

Joint motion either affects the fCO afferents (mechanically coupled) or does not (mechanically decoupled).

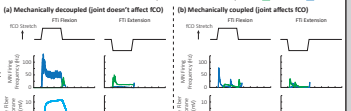

Figure 3: Joint response to ramp stimuli corresponding to joint flexion (stretching of the fCO) and joint extension (relaxation of the fCO).

Results

Effect of Sensory Neurons on Motion

Most drastic change to joint motion occurs when inhibiting the flexion position neurons.

- When combined with some degree of velocity inhibition, effect becomes more pronounced
- Cases involving high inhibition of flexion position and velocity resulting in no reaction to joint flexion.

This mechanically decoupled response resembles motion characterizing the active reaction (AR).

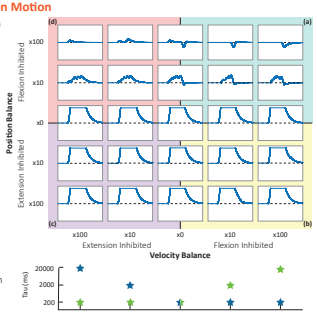

Figure 4: the mechanically decoupled joint motion of the system in response to fCO stretching for different combinations of inhibited sensory neurons.

Figure 5 shows the reaction of the joint to the stimuli used previously, but with high inhibition of flexion position and velocity afferents. Both the mechanically decoupled and coupled responses resemble motion characterizing the active reaction (AR).

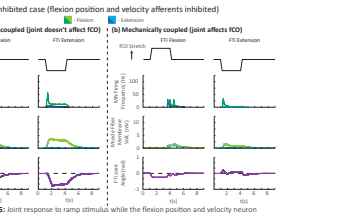

Figure 5: Joint response to ramp stimulus while the flexion position and velocity neuron groups have been inhibited.

Positive feedback in initiating, sustaining locomotion

In each test, the only stimulus entering the network is to the desDUM neuron.

Figure 6 shows the activity from baseline stepping.

- Externally applied stimulus at two seconds excites the desDUM neuron, beginning stepping
- Each time the tarsus impacts the ground, the resulting strain excites the desDUM
  - Keeps the membrane voltage within a range of excitation to continue stepping

Figure 7 shows the effects of both joint strain and desDUM modulation on stepping.

- Without strain excitation the leg will only step until the desDUM neuron voltage decays past a certain threshold.
- With strain providing positive feedback as intended, an inhibitory stimulus to the desDUM neuron is necessary to halt walking
  - Applied at 16 seconds in our baseline case
  - Once applied, the joint passively returns to equilibrium position over time.
- Without this inhibition, the strain will continuously maintain the desDUM neuron voltage, allowing the leg to continue stepping.

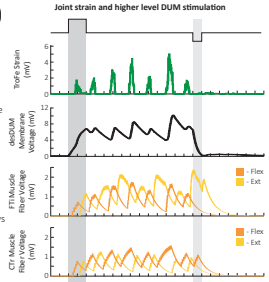

Figure 6: Through a combination of strain feedback and higher level desDUM neuron stimulation, our network can generate biologically plausible walking.

Results

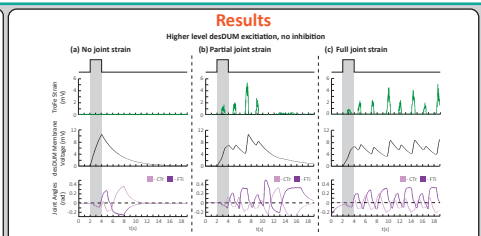

Figure 7: Strain feedback from the CS is integral to initiating and maintaining stepping.

In cases where the network only receives strain feedback without higher level desDUM stimulation, the resulting limb motion can vary (**Fig. 8**).

- Small amounts of applied strain result in only slight perturbation of the limb.
  - The MNs are fully activated, but the voltage is not enough to begin rhythmic oscillation.
- Larger strains can force stepping in the limb without higher level desDUM excitation

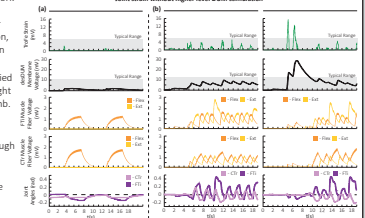

Figure 8: Depending on the magnitude, applied strain with no other stimulation of the desDUM neuron can produce varying joint motions.

Reflex reversal effects during stepping

Figure 9 shows the effects of stepping without sensory afferent inhibition.

- In the RR, the limb is unable to complete viable stepping.

This data supports our hypothesis that reflex reversal assists stepping by changing the allowable motions of the joint.

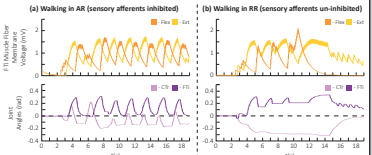

Figure 9: Inhibition of sensory afferents to initiate reflex reversal ensures proper stepping of the leg.

Discussion

Our data seems to imply:

- **The nervous system may actively modulate the lowest level sensory afferents in order to alter motion and control**. Sparse descending commands could reduce latency through the distributed nervous system, which could ensure stable and capable walking for rapid insect walking.
- **The nervous system seems to use reflex reversal to define constraint forces instead of active motion forces.** These constraints allow the joint to resist undesired movements from external forces while allowing assistive external forces to move the joint. Insects may adopt this counter-intuitive control scheme to reduce energy expenditure, which is critical for a living organism.
- **Sensory feedback driven reinforcement mechanisms (positive feedback in biological literature) may reduce control complexity by allowing for distributed, low level management of behaviors.** Brief descending input signals can initiate a behavior, then the inputs added by lower level feedback will attempt to maintain the behavior automatically. If the system seems to have `failed` (i.e. feedback no longer present), the lack of further inputs will automatically halt the behavior. Similarly, strong enough feedback can rapidly initiate helpful behaviors without any higher level inputs.

In insects, the mechanisms of communication between the `higher level command centers' (HLCC, e.g. the cerebral and gnathal ganglia) and the `low level motor centers' (LLMC) in the ventral nerve cord (VNC) are largely unknown. Specific questions include: What types of information are shared between the brain and VNC by descending neurons and ascending neurons? What do these pathways encode? What are their neuronal downstream targets? The present study begins to address these questions. Our model may improve understanding the nervous system in general and lead to new robotic controllers.