MODELING THE CONTROL OF FLIGHT LOCOMOTION IN A LOCUST

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ABSTRACT

Can the physiological results of individual neurons in insects, which help experimenters determine proposed interconnections of networks, be used in simulation programs to further develop models to provide insight into the control mechanisms of the networks? Artificial neural networks are being used to provide a general model of a system (such as insect locomotion) which, through weighting structures and learning patterns, can provide the specific functional output. These networks are very powerful and can be modified easily to change their output, but these do not model the actual neural structure of the original system. By using networks of the proposed neural structure, based upon physiological evidence of the interconnections of elements, in neural simulation programs, specific control of portions of the system and control of the developed model can be investigated. These networks may be idiosyncratic and unique in their details of operation, but these may lead to a better understanding of the morphology of the structure, and to new implementations of control for a specific function [1].

INTRODUCTION

Locusts fly with two sets of wings (the forewings and the hindwings) at a frequency of around 20Hz. The hindwings are activated before the forewings by about 5 to 10 milliseconds [2]. The wings themselves are structured to provide the thrust and pitch and provide a certain amount of control during the flying motion, but each wing has about 10 muscles controlling it. These are divided into two sets of synchronous muscles, the elevator and depressor muscles, which alternate their control of the wing to provide motion. There are about 20 motoneurons associated with the muscles of each wing and most of these are identifiable and physically located in the portion of the insect's body attached to that wing. The forewings are associated with the mesothoracic region of ganglia (which is a grouping of neurons and their connections) and hindwings with the metathoracic region. Therefore, homologous sets of motoneurons and sensory neurons (which provide feedback from the stretch receptors and depression receptors) are found in the two thoracic regions.

The neural circuitry which provides the rhythm for flight and controls the activation of the motoneurons is more complicated, because the interneuronal organization is not set up as two distinct controls for forewing and hindwing motor activity. There are three different categories of interneurons; some are members of one of two serially homologous groups controlling either the forewing or the hindwing, some are unique individual interneurons without any homologues, and some are members of a set of serial homologues in the metathoracic and first three abdominal ganglia regions [3]. The fact that similar interneurons which are involved in flight control were found in the abdominal regions added to the complexity of the neural circuitry controlling flight, until it was viewed from an evolutionary perspective [4]. The idea that the locust has evolved from an insect which had movable appendages serially repeated along its body, but has adapted and been selected for flight locomotion could explain the serial repetition of these flight interneurons in the abdominal region for which there is no functional significance.

The pattern generating mechanism for flight is determined by interconnections of the interneurons in the thoracic region. When the insect is deafferented and isolated from all sensory inputs as well as cerebral ganglia inputs, this mechanism can still produce repetitive and alternating control to the elevator and depressor muscles. This is not to say that the sensory inputs have no effect on the rhythm, in fact, they modulate and reset this rhythm, which is necessary for natural flight. The flight rhythm determined by these interneuronal connections drives both pairs of wings. The rhythm seems to be generated by synaptic interactions of the interneurons [5]. These connections are of three types: chemically- mediated excitatory connections, similar inhibitory connections, and delayed excitatory connections. Some subthreshold interactions amongst the interneurons have also been found. Through physiological experimentation many specific interneurons have been described in detail along with their interconnections, but many other interneurons, and especially their interconnections have not fully been mapped out. This network relies on reciprocal inhibitory and excitatory feedback, which in itself would be a trivial problem to generate alternating activity, but the hard part is to coordinate all the oscillators and organize their output timing as well as their inputs received from the sensory systems. Therefore, the locust flight system is a complicated and unique network of many different neuronal interconnections for which a specific model may provide insight in the actual control mechanisms.

A PROPOSED MODEL OF LOCUST FLIGHT SYSTEM

A simplified view of a proposed model of the locust flight system is shown in Fig. 1 below [6].

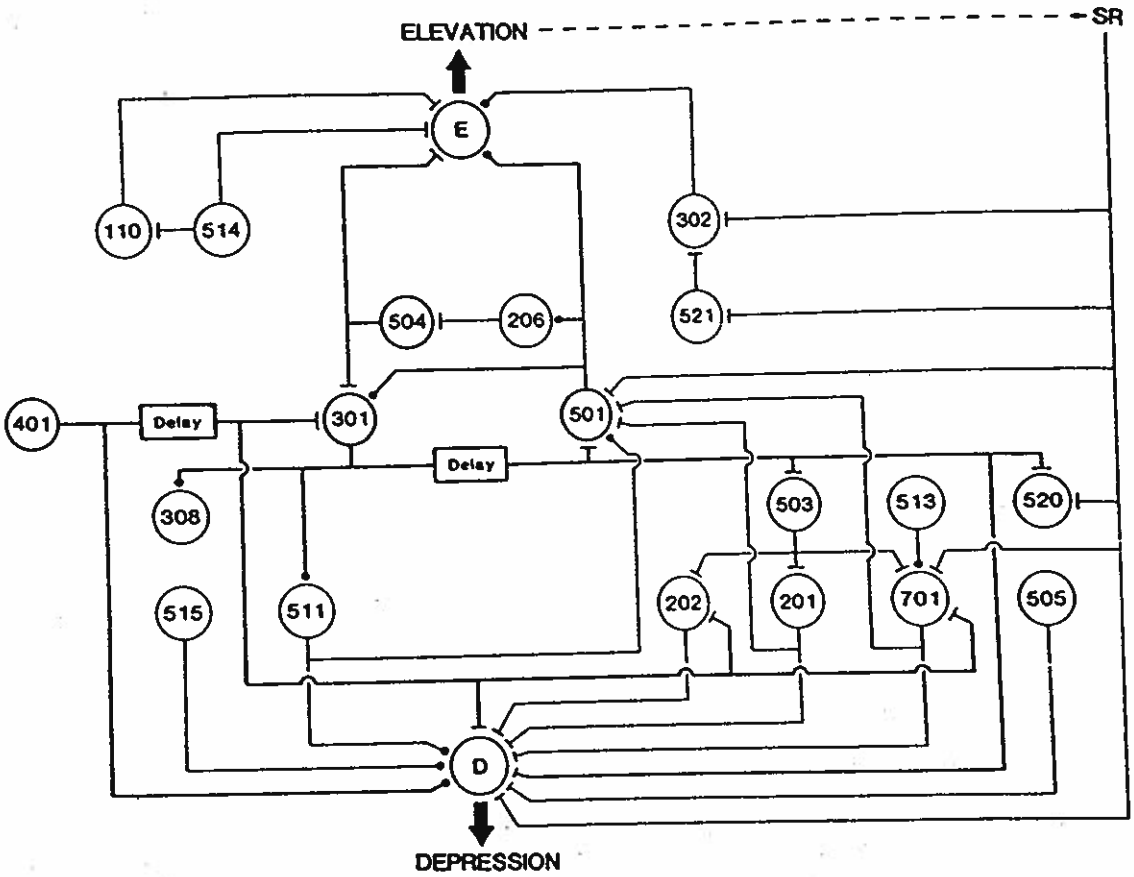

Figure 1 : Diagrammatic representation of the connections found between flight neurons.

The model is based on physiological results from experiments determining monosynaptic connections between one labelled interneuron and another. There were found to be at least 50 interneurons phasically active with flight rhythm, and the generation of this rhythm depends upon the interaction of a group of central and peripheral cells. Interneurons 301 and 501 are involved in the central pattern generator and can reproduce the flight rhythm without sensory input at a lower frequency than normal, but can maintain alternate activity between the elevator (E) muscles and depressor (D) muscles. Different portions of the model need to be developed independently and simulated with realistic test inputs. The system can be broken down into at least three main control networks. One network consists of the interneuron interactions with the motoneurons which control the flight muscles, another network consists of the different pattern generators and their interconnections to provide the alternate synchronous rhythm of flight, and the last network involves the sensory neuron feedback control of the generators. Building these smaller portions of the control system at the cellular level points out questions about the proposed interconnections of the interneurons, as the simulations may not corroborate the experimental results. This problem arises for many reasons. The model of the cell may not be accurate enough , or the constraints on the parameters in the equations of the cellular functions are not adjusted correctly, or the interconnections from one neuron to the next are not modeled correctly,or inputs or outputs may be missing from the original proposed model. These questions can be posed to the neurobiologists to determine new experiments to provide more details on a specific function to redefine the parameters associated with a particular part of a model element or to redefine the equation or the model itself. The program for modeling must be reprogrammable at the element level and provide an easy way to change the initial parameters or constraints on the model.

DETERMINATION OF A MODELING AND SIMULATION PROGRAM

One of the key elements of all modeling and simulation research, during present times, is the computer program written to implement the model and provide the simulation results. Biological neural network programs provide a different aspect to the research of physiological functions than artificial neural network programs. The former will provide information about the control of the actual networks in the biological system. Designing a biological neural network program relies upon detailed models of the individual elements within the system, the type of computations involved to describe the action of each element, the model of different types of element connections to inputs and outputs. The biological neural network program could be designed to be very detailed about the individual elements and therefore try to mimic the actual biological elements themselves, but there is a limitation on this as many of the biological elements are not truly defined. Therefore, these networks will be working with a proposed model with a limited degree of detail and equations to describe their interactions.

The most important individual element which the biological neural networks are based upon is the neuron cell, and in particular, its membrane, which is the key to all interactions. Hodgkin and Huxley in 1952 came up with a model of a cell's membrane based upon its conductivity to certain ions and related this to the voltage potential across the membrane [7]. They explained the classical phenomena of electrical excitation due to sodium and potassium influx and efflux. Using hand calculations, they came up with a model based mainly on developed equations which fit the curves of experimental observations. This in turn provided insight into the mechanisms of changes in Na+ (sodium ion) and K+ (potassium ion) permeabilities. The HH Model of the cell membrane became the backbone of biological research of systems [8]. Even researchers at the cellular level used the model and then tried to research how the mechanisms at the atomic level verified this model. Hille describes the model workings by the opening and closing of specific ionic channels created by proteins oriented within the membrane structure according to the voltage potential that the atomic structures of the protein sees [9].

Many biological neural network programs have developed their models based upon the HH model kinetics. Usually these programs were modeled around the specific neuronal properties of the researcher's

interest. To develop a large network model and simulation of neural systems in general, certain factors become important in choosing the neural network program. First, a modular format for the program makes a much more efficient way to define similar elements and even similar networks and reprogramming and deleting is much easier. Next, a graphical interface to the program, which is interactive with the simulation, provides a means to change simulation parameters as well as neuronal properties and see their results while a simulation is taking place. Lastly, as the networks grow, so does the computation time and the type of computer used for the program becomes important, in fact, the program should eventually be able to port to parallel processing machines as the number of elements increase.

NEURONAL MODELING USING THE GENESIS PROGRAM

Genesis, a general modeling and simulation program, runs on a Sun or Sparc workstation and uses XODUS, a graphical interface built upon MIT's X11 library. This program provides a modular, interactive, graphical means to build a network of biological cells and gives researchers the opportunity to probe at the elements of the network to study their control functions.

This program has been utilized by many researchers working on different systems. The program can be tailored to provide the idiosyncracies of the individual elements and connections that each system needs. This structural modeling provides a means to study the functional properties f complex networks which are constrained by the detailed experimental biological data. As a network is broken down into subsystems, the different complexities of the definition become important. The model and simulation of the neural system for control of flight of the locust is still under development, but portions of the system have been input and analyzed. The specific interconnections between the interneurons 301 and 501 and others involved in the flight central pattern generation may require less specification of the models of the interconnections at first, and more detail about the individual cell model [10]. Whereas, the interconnections from the pattern generators to the motoneurons, muscle activating neurons, require more specification of the details of the interconnections, specifically the dendritic tree branching and length of connection to provide proper time response for that system [11].

The input/output relationship for each neuron is defined by specifying the passive or active membrane properties and repetitive firing characteristics associated with each particular interneuron. The time course and strength of each synaptic action is specified. The axon and dendritic tree is compartmentalized and provided with parameters to simulate linear cable theory. The network is developed and provided with realistic parameters from experimental data which provides the constraints for the simulation runs. The single compartment model for an individual neuron incorporates passive and active membrane properties shown with the equivalent circuit, Fig.2. This shows the passive properties to include a resting potential (E_{leak}) and membrane resistance ($1/ g_{leak}$)and cell capacitance ($\hat{}c_m$) whose parameters fit those measured for the particular neuron from the membrane potential exponential.

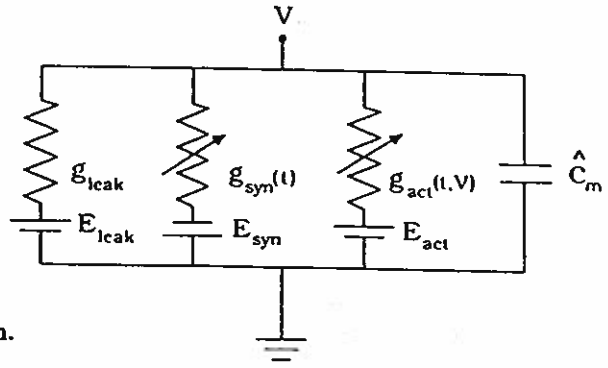

Figure 2 : Circuit model of neuron.

The active properties are based on an integrate and fire model. The neuron sums inputs and fires when the membrane potential reaches a threshold level (Θ (t)). After each spike the level increases and is allowed to decay towards an asymptotic level. There are three contributions of current in the cellular model, the leakage current due to the passive ionic channels, the voltage-dependent currents which correspond to the active ionic channels present in the cell membrane, and the synaptic currents which correspond to the chemically-gated inputs. The postsynaptic potentials are proposed to be due to the binding of transmitter substance and supposed opening of channels, which is modeled by a time-generated conductance increase in association with a reversal potential. An externally applied current (I_{stim}) can be applied at any time with any waveform, this provides a way to control the cell or provide test inputs. The currents and their equations in the circuit model show their different relationships due to their conductances as described below. The membrane potential is described in relation to these currents [12].

For each cell, the membrane potential is described by

$$\hat{c}_m \frac{dV_m(t)}{dt} = -I_{leak}(V_m) - I_{syn}(V_m, t) - I_{act}(V_m, t) - I_{stim}(t)$$

$V_m =$ membrane potential(mV)

$\hat{c}_m =$ membrane capacitance(nF)

$I_{leak} =$ leak current(nA)

$I_{syn} =$ synaptic current(nA)

$I_{act} =$ intrinsic voltage-dependent current(nA)

$I_{stim} =$ externally applied currents(nA)

Leakage Current

$$I_{leak} = (V_m - E_{\text{leak}})g_{leak}$$

$E_{\text{leak}} =$ resting potential(mV)

$g_{leak} =$ input conductance(μS)

$R_{\text{m}} =$ input resistance$(M\Omega) = \frac{1}{g_{leak}}$

Synaptic Current

$$I_{syn} = \bar{g}_{syn} \cdot g_{syn}(t)(V_m - E_{syn})$$

$\bar{g}_{syn} =$ maximum conductance for a given synapse (μS)

$E_{syn} =$ synaptic reversal potential(mV)

$g_{syn}(t) =$ synaptic conductance time course

$$g_{syn}(t) = \frac{\tau_d}{\tau_d - \tau_o} \sum_{i=1}^{N} (e^{(t_i - t)/\tau_d} - e^{(t_i - t)/\tau_o})$$

τ_o = onset time constant ($msec$)

τ_d = decay time constant ($msec$)

N = number of presynaptic spikes before time t

t_i = time of ith presynaptic spike ($msec$)

Voltage-Dependent Current

$$I_{act} = \bar{g}_{act} m^x h (V_m - E_{act})$$

$\bar{g}_{act}$ = maximum conductance (μS)

m = activation parameter

x = exponent on activation parameter

h = inactivation parameter

E_{act} = reversal potential of active process (mV)

$$\frac{dm}{dt} = \frac{m_\infty - m}{\tau_m}$$

$$m_\infty = \frac{1}{[1 + e^{(V_m + B)/C}]} = \text{steady state activation}$$

B = shift parameter (mV)

C = shape parameter (mV)

τ_m = activation time constant ($msec$)

$$\frac{dh}{dt} = \frac{h_\infty - h}{\tau_h}$$

$$h_\infty = \frac{1}{[1 + e^{(V_m + B)/C}]} = \text{steady state activation}$$

B = shift parameter (mV)

C = shape parameter (mV)

τ_h = inactivation time constant ($msec$)

Threshold

$$\theta(t) = \theta_{\infty} + (\theta_0 - \theta_{\infty})e^{(t_x - t)/\tau_{\theta}}$$

θ_{∞} = steady-state threshold (mV)

θ_0 = threshold value immediately after a spike (mV)

t_x = time of last spike in cell ($msec$)

τ_{θ} = threshold time constant ($msec$)

The single compartment is the basic element of the modeling program, but it is built upon components which can be changed easily and the parameters constraining these components can be changed even during simulation run time. Each interneuron has its own compartment specifications representing its particular characteristics which were extrapolated from experimental data for that interneuron. The model of the system is built upon the interconnection of many of these elements, therefore the simulation results are only as good as the individual parameter specifications because it is developed upon structural and physiological results. The simulation can be run using a script as input, or it can be run interactively through a graphical interface and the changes can be stored as a new script. An example of the types of interaction with the simulator is shown in Fig. 3 [13].

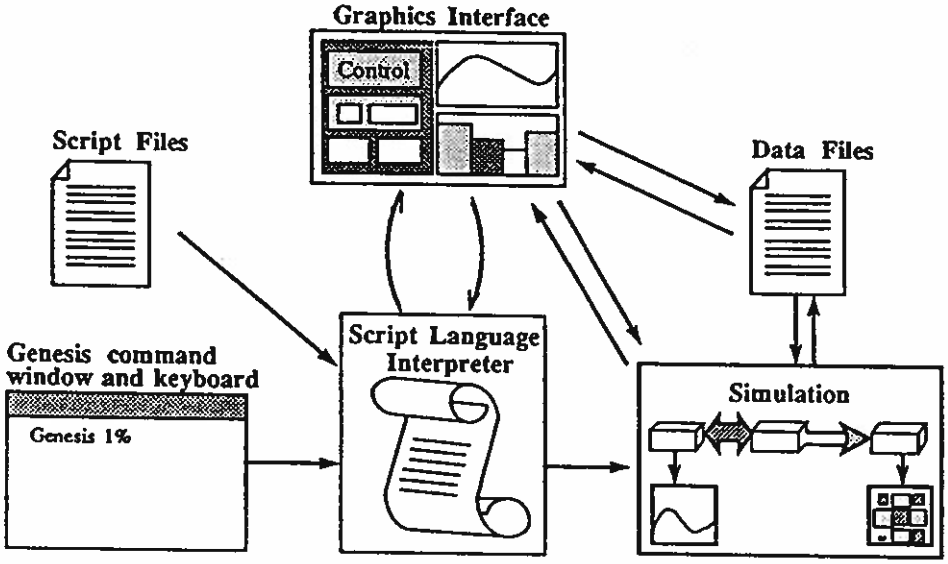

Figure 3 : Levels of interaction with the simulator.

CONCLUSIONS

The results from models and simulations from the biological neural network program have provided respecification of parameters for the individual elements. This has lead to better definition of the model which provides closer similarities between simulation results and physiological results. In the redefinition of the model interconnections are better understood and this provides more insight into the control mechanisms of the system.

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MODELING AND SIMULATION
VOLUME 23

Part 5
Networks, Communications, Biomedical, General

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William G. Vogt
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Proceedings of the Twenty-Third Annual Pittsburgh Conference
Held April 30 - May 1, 1992
School of Engineering - University of Pittsburgh

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School of Engineering - University of Pittsburgh