HUMAN-COGNITION- IN-THE-LOOP:
A FRAMEWORK TO INCLUDE DECISION PROCESS IN CLOSED LOOP CONTROL

by

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This thesis proposes a framework to include human cognitive process in making decision into control loop. Our approach in solving this problem is to add a complementary control which includes a model of human decision to the controller, capable of predicting human command accuracy in the near future. The outcome of decision unit can either be presented to the operator as a directive or may adjust the issued command toward better results. In order to construct human decision model, we combined tree approaches of bio-physical connectionist, mathematical abstraction and behavioral cognitive models in the adaptive gain theory framework. We first extended the classic DDM to include a layer that represent the change of strategy from weighted additive to heuristics and proved that such model is mathematically sound and well defined. Then by the help of adaptive gain theory, we showed that by collecting feedback signals from operator, we are able to predict her decision quality.

We also presented a prototype for a supervisory controller that includes the dynamics of making decisions by humans in the control loop. A two-stage model for strategy selection and decision making was utilized to cover a range of situations, in which the operators are required to make proper decisions. A controller was designed to dispatch the tasks between the system and the operator to keep the operator close to the best performance region. A case study for a simple one-attribute task was

simulated to show the effectiveness of the proposed controller.

One major assumption in designing complementary control unit was that the feedback physiological signals can be mapped onto NE-LC gamma plane, hence the quality of decision at each time is known to controller. In the experiment section, we relaxed this assumption and showed that by using commercially available technologies, it is possible to infer the decision strategy and accuracy.

DEDICATION

To Poupack and the one we are expecting...

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Chapter 1

Introduction

On September 8, 2011, the largest power outrage in California's history occurred because a technician erroneously shut down a power line. None of the automated control and protection systems could prevent this incident because the human operator had the authority to override autonomy. While fool-proofing complex cyber-physical networks with unprecedented emergency scenarios is a common approach, the important lesson learned from such an incident is that a comprehensive solution to system control should include human behavior in the loop. In the current "human-in-the-loop" perspective, researchers provide only a rough caricature of actual human cognition and decision making. Without cognitively realistic models of human decision making, one cannot appropriately model these systems and develop decision tools to help human operators cope with abrupt changes and unprecedented situations. Thus, a fundamental need exists to bridge this critical gap in understanding of human operator decision making in CPS.

The *ultimate goal* of this project is to improve the expected performance of CPS, considering human factors in the control loop. This includes robustness and resilience of CPS against catastrophic failures. The physical system, human operators, and the control system together form a so-called human-in-the-loop supervisory control

system. The proposed research aims to optimize this human-in-the-loop supervisory control system by mainly controlling the parts that affect human performance. In other words, the project aims to achieve the goal by affecting human operators status instead of only controlling physical systems parameters. For this purpose, the first step is to develop appropriate models for human roles in CPS by integrating cognitive science perspectives on human decision making. Then, one can focus on searching reasonable decision-making strategies for the human-in-the-loop supervisory control system. The proposed research can refine these two steps into the following objectives:

Objective 1: Develop and simulate models for human decision making process, interacting with cyber-physical network specially under internal and external stress factors such as emergencies.

Objective 2: Propose a control strategy to maximize the average success rate of human decision making for CPS.

Furthermore, in order to model actual human decision making, an experiment is design to empirically validate the theoretical results. That is, in addition to investigate theoretically, which classes of models perform well, this study aims to test whether these models capture the decisions that people actually make. The main feature of the experiment is using commercialized technology to establish the closed loop control scheme, including the data required from physical and emotional state of decision maker. In this way, minimal set of effecting factors, i.e. stress and fatigue were chosen and their effect on decision models investigated.

In Chapters 2, we first review approaches in modeling human decision making process known as biophysics-based models. Spiking neural network (SNN) models is neuron-level approach to mimic the process of decision making in the brain by simulating the dynamics that govern a single neuron activity as well as the dynamics of synaptic connections. Spiking neural networks mostly use the *leaky integrate-*

and-fire (LIF) model for each single neuron, which is essentially a one-dimensional model without the capability of producing the diverse and intricate range of neuronal spiking behavior. For that reason, we decided to choose *simple model* which is two dimensional dynamical model and by help of mathematical tools, specifically bifurcation theory, can produce a diverse range of neuronal spiking regimes. Later in Chapter 5, we simulated the behavior of population of neurons, each modeled with simple model te get a measure of human decision performance for a sample task.

In Chapter 3, mathematical abstraction methods in describing decision process are investigated. This approach is built on drift diffusion model (DDM). A DDM is a mathematical abstraction in which the continuum limit of the sequential probability ratio test is employed in the sense that it captures the optimal decision time, assuming a fixed accuracy rate and initial conditions. It can account for how behavioral performance improves over time as a result of the accumulation of information, and has been successfully applied to predict human accuracy and reaction time. Two separate approaches in deriving continuous and discrete DDM processes are introduced and a complete model for multi-cue multi-choice task with strategy selection layer is proposed. Through mathematically rigorous process, we proved that the proposed model is well defined and converge in first and second statistical moments.

Chapter 4 deals with the notation of strategy selection which is a common term in behavioral science. In general, strategy selection is a tool to cope with situation that flow of information and/or the decision process itself undergo changes that deviate form optimal practice. One good example is the level of stress in decision maker that affect her performance in a great deal. Here we specifically focus on locus coeruleus-norepinephrine (LC-NE) framework, a process in human brain that plays a complex and specific role in judgment and decision. Combining our proposed DDM model with LC-NE process and strategy selection practice, we construct a closed loop feedback

model to mimic the human decision process in an enterprise level control platform.

Chapter 5 gives an example of supervisory and control system that utilize the proposed decision model. Our purpose in building such example is to show that constructed model can be used in conjunction with modern AI technologies to enhance the performance of the system in a great deal. Though some assumptions are made, specially in terms of biological feed back data (excitatory-inhibitory gain level) which are not collectible with today's commercial technologies. To address this shortcoming, in Chapter 6 we design an experiment to show that with more accessible data such as hear-beat variability we can estimate the state of decision maker in terms of using different strategies in stressful condition. Therefore, even with today's commercialized technologies it is viable to use the human cognition in the loop framework for enhanced system control.

The innovation of the current study stems from the integration of three different approaches in modeling human process, namely bio-physically inspired models, mathematical abstract models and behavioral models into one comprehensive framework which is capable to pair with system autonomy and improve the overall performance specifically under stress condition. Having such model in hand and validate it through experiment, the proposed research may be able to inspire the development of a network supervisory control scheme that facilitates better operator decision making by helping reduce operator's error specially during emergency situations.

Chapter 2

Neuro-Physiological models

Finding a complete description of nervous system dynamics is still a far fetching goal, however considerable advancements have happened in recent years to construct a mathematical model that can emulate the physiological properties of a single neuron. Neurons produce action potential or *spike* to communicate with each other. Spike is an abrupt and transient change of membrane voltage. Inputs which are received through dendritic tree or synapses cause trans-membrane currents and consequently post-synaptic potentials (PSPs) that tend to change the membrane potential of the neuron. Large PSPs can be amplified by the voltage sensitive processes embedded in the neuronal membrane and lead to generation of spike. A typical neuron receives input from more than 10,000 other neurons and fire spikes with different regimes. So the fundamental question is why the response of neurons to their inputs are so much different. Is there a simple model that can describe the response with acceptable accuracy?

In this chapter we investigate models that are based on of electrophysiology of neurons which primarily focused on dynamics of membrane voltage in the classic approach. Then using the mathematical tools provided by dynamical systems and bifurcation theory, we proceed to know more involved model, named simple model [1].

The core concept is treating neurons as dynamical systems and using mathematical tools such as phase analysis and bifurcation theory to find a minimal model, capable of describing the wide range of neural activities. But first let's take a quick look at neuronal electrophysiology to have a better understanding of the dynamics of a neuron.

2.1 Electrophysiology of Neurons

Neurons use ionic currents to conduct their electrical activities. The transmembrane ionic currents mostly include one of the four ions, namely sodium(Na^+), potassium(K^+), calcium (Ca^{2+}), or chloride (Cl^-). The concentration of ions is different on the inside and the outside of a cell, which is the driving force of their movement. As depicted in Fig. 2.1, the concentration of (Na^+) and (Cl^-) is high in extracellular medium while inside the cell there is more concentration of (K^+) and negatively charged molecules denoted by (A^-).

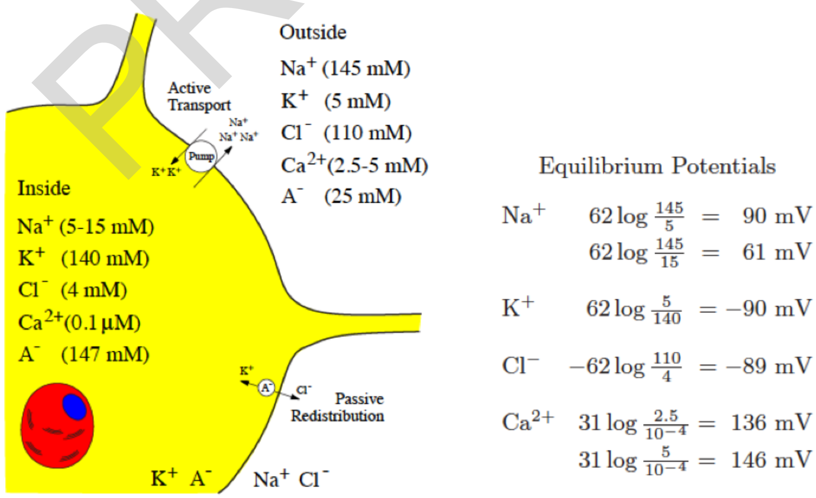

Figure 2.1: Ion concentration and Nernst equilibrium potential [1]

Active (pumping of ions via ionic pumps) and passive (attraction of (K^+) and

repelling of (Cl^-) by impermeable (A^-)) redistribution of ions through membrane protein channels maintain the stability of membrane voltage in the rest state. The process involves two opposite forces. For instance, consider (K^+) ions. Since the concentration is lower on the outside, the ions start to flow outward and make the inside medium more negative. This induces an increasing voltage difference on two sides of membrane which makes it harder for (K^+) currents to flow outward. At one point the equilibrium is achieved when concentration gradient and electric potential gradient cancel each other and the net current becomes zero. The value of equilibrium potential depends on the ionic spices and called the *Nernst* potential. Based on that the net current is given by:

$$I_{\mathrm{K}} = g_{\mathrm{K}}(V - E_{\mathrm{K}}) \tag{2.1}$$

In which V is membrane potential in volts, g_K is the (K^+) conductance in ($\mathrm{mS/cm^2}$) and E_{K} is the Nernst potential of (K^+). The same type of equation can describe other ionic currents which lead to the equivalent circuit of Fig. 2.2, for a single neuron. Note that the capacitance of membrane is in ($\mu\mathrm{F/cm^2}$). Based on electrical equivalent circuit of Fig. 2.2, the equation that describes the ionic currents is:

$$\begin{aligned} C\dot{V} &= I - I_{\mathrm{Na}} - I_{\mathrm{C}a} - I_{\mathrm{K}} - I_{\mathrm{C}l} \\ &= I - g_{\mathrm{Na}}(V - E_{\mathrm{Na}}) - g_{\mathrm{Ca}}(V - E_{\mathrm{Ca}}) - g_{\mathrm{K}}(V - E_{\mathrm{K}}) - g_{\mathrm{Cl}}(V - E_{\mathrm{Cl}}) \end{aligned} \tag{2.2}$$

If there is no additional source or injected current then $I = 0$. In this case the membrane potential is typically bounded by :

$$E_{\mathrm{K}} < E_{\mathrm{Cl}} < V_{\mathrm{rest}} < E_{\mathrm{Na}} < E_{\mathrm{Ca}} \tag{2.3}$$

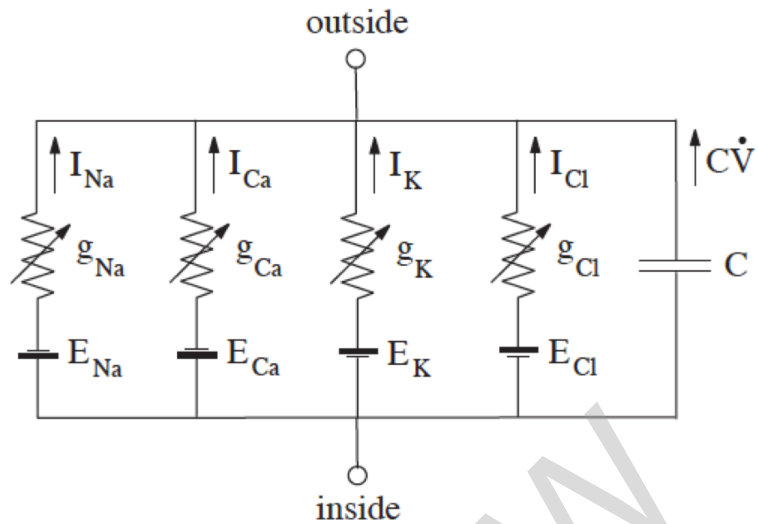

Figure 2.2: Equivalent electrical circuit representation of a neuron

Also note that I_{Na} , $I_{\mathrm{Ca}} < 0$ (inward currents) that make the membrane potential more positive or cause depolarization while I_{K}, $I_{\mathrm{Cl}} > 0$ (outward currents) that make the membrane potential more negative or cause hyperpolarization. At V_{rest} we have $\dot{V} = 0$ and hence:

$$V_{\mathrm{rest}} = \frac{g_{\mathrm{Na}}E_{\mathrm{Na}} + g_{\mathrm{Ca}}E_{\mathrm{Ca}} + g_{\mathrm{K}}E_{\mathrm{K}} + g_{\mathrm{Cl}}E_{\mathrm{Cl}}}{g_{\mathrm{Na}} + g_{\mathrm{Ca}} + g_{\mathrm{K}} + g_{\mathrm{Cl}}} \tag{2.4}$$

The denominator of 2.4 is the total input conductance, g_{inp}, of the neuron. The quantity $R_{\mathrm{inp}} = 1/g_{\mathrm{inp}}$ is called the input resistance. The bigger value of resistance results in larger displacement from V_{rest} due to injection of DC current I.

The input resistance is a function of membrane voltage and time. The common experiment to see the relationship is holding the membrane voltage at fixed value V_c by means of voltage-clamp. Then reset the value of voltage to V_s and measure the instantaneous and asymptotic currents. The instantaneous I-V curves usually has non-monotone N-shape curve which shows the nonlinear positive feedback trans-

membrane process and can be assumed fast enough to have instantaneous kinetic [3]. The steady state I-V relation can be monotone or not depending on the properties of membrane current.

2.1.1 Gated Channels

Ionic channels are large transmembrane protein that facilitate the flow of ions trough the membrane. The electrical conductance of the channel may be controlled by gating particles which switch the channels between close and open states [4]. The net current generated by large population of identical channels can be described by the equation

$$I = \bar{g}p(V - E) \tag{2.5}$$

in which parameter p is the average proportion of channels in open state. If the channel is selective for a single ion, the value of E will the the relevant Nernst potential. When the gating variable is sensitive to the membrane voltage the channels are called voltage-gated. Also the gates are divided to activation gates and inactivation gates. The probability of an activation gate is being open is denoted by variable m and the probability of inactivation gate in open state is denoted by variable h. Therefore

$$p = m^a h^b \tag{2.6}$$

where a and b are the number of activation and deactivation gates respectively. Some channels do not have inactivation gates($b = 0$). Such channels do not inactivate which results in persistent currents. In contrast channels with inactivation results in transient currents. The dynamic of activation variable is given by a first order

differential equation [3]:

$$\dot{m} = (m_\infty(V) - m)/\tau(V) \tag{2.7}$$

The values of steady state activation function and time constant can be measured experimentally. Likewise the dynamics of inactivation variable is described by first order differential equation

$$\dot{h} = (h_\infty(V) - h)/\tau(V) \tag{2.8}$$

Inactivation kinetics is usually slower than activation kinetics. $h_\infty(V)$ has the same sigmoid shape of $m_\infty(V)$.

2.1.2 Hodgkin-Huxley Equations

Using experimental techniques, Hodgkin and Huxley (1952) determined that giant squid axon carries three major currents : voltage gated persistent K^+ current with four activation gates, voltage gated transient Na^+ current with three activation gates and one inactivation gate, and ohmic leak current which carried mostly by Cl^-. The model is described by the following set of equations [5].

$$\begin{aligned}
C\dot{V} &= I - \bar{g}_{\mathrm{K}} n^4 (V - E_{\mathrm{K}}) - \bar{g}_{\mathrm{Na}} m^3 h (V - E_{\mathrm{Na}}) - \bar{g}_{\mathrm{L}} (V - E_{\mathrm{L}}) \\
\dot{n} &= (n_\infty - n)/\tau_n(V) \\
\dot{m} &= (m_\infty - m)/\tau_m(V) \\
\dot{h} &= (h_\infty - h)/\tau_h(V)
\end{aligned} \tag{2.9}$$

Where the steady state and time constant values of gating variables are function of V and time. It is common to approximate the steady state activation curve by

Boltzmann function [3], e.g. for m

$$m_\infty(V) = \frac{1}{1+\exp\{(V_{1/2}-V)/k\}} \tag{2.10}$$

The parameter $V_{1/2}$ satisfies $m_\infty(V_{1/2}) = 0.5$ and k is the slope factor (negative for h). Smaller values of $|k|$ results in steeper curves. The time constant is approximated by Gaussian function

$$\tau(V) = C_{\text{base}} + C_{\text{amp}}\exp\frac{-(V_{\max}-V)^2}{\sigma^2} \tag{2.11}$$

Hodgkin-Huxley model is constructed based on evidence gathered from a single experiment. However for more general modeling purpose, a normal form that universally describes the neuron behavior is required.

2.2 Integrate and Fire

The most popular neural model is known as integrate-and-fire [6]. In this model, neurons are integrator units that sum up the incoming PSPs and compare the sum with a threshold value. If the sum is greater than the threshold, neuron will fire a spike and then the membrane potential reset to a rest value. In other words, spikes are generated whenever the membrane potential u crosses the threshold value ϑ. The firing time is defined as the crossing moment, i.e. $u(t^{(f)}) = \vartheta$.

The standard equation of linear leaky integrate-and-fire neuron is given by [6]:

$$\tau_m\frac{du}{dt} = -u(t) + RI(t) \tag{2.12}$$

in which $\tau_m = RC$ is membrane time constant and R and C are equivalent resistance