Novel Reconfigurable Computing Architectures for Neural Information Processing

John DiCecco, PhD and Jason Gaudette, PhD
Contributions by Alexander Batrakov, Georges Gauthier, and Christopher Toole

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Neural Reconfigurable Architectures
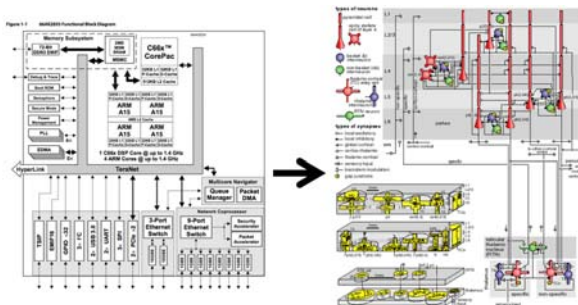
- Need exists to bend the complexity curve of linear systems
- On demand incorporation of neural components
- Maintain computational capacity with fewer systems/components
- Reexamine dependence on interconnectedness
- Goal: Design and build proof-of-concept printed circuit board with interconnected neurons exhibiting realistic and variable firing patterns (at right, simulated)
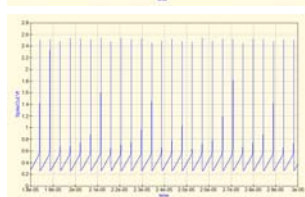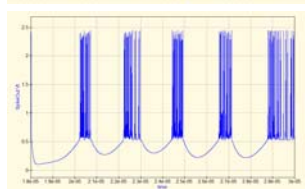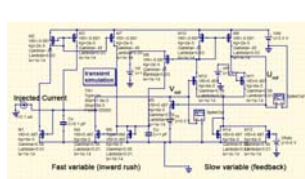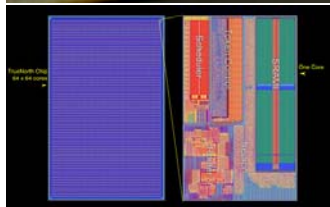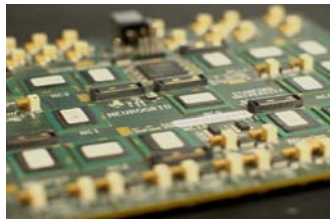
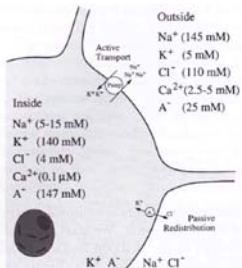# Before We Go Any Further, Consider ...
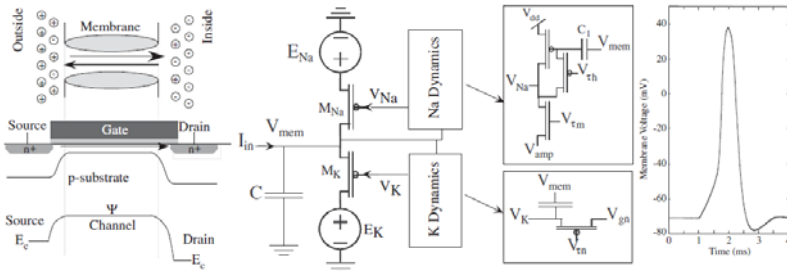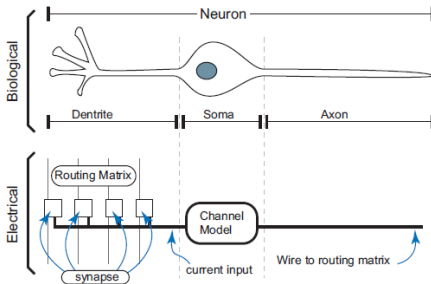
A Few Current Players

- Stanford Neurogrid
- IBM TrueNorth (DARPA)
- GaTech FPAA
- Hasler, Indiveri, Boahen (as a very small subset)
    - Easy to read background by Hasler (GaTech), Indiveri (Univ. Zurich INI)
- Massive research in analog vice digital transistors (MOSFETS), memristors, neuristors

Basic Stuff

Figure 2.1: Ion concentrations and Nernst equilibrium potentials (2.1) in a typical mammalian neuron (modified from Johnston and Wu 1995). A^- are membrane-impermeant anions. Temperature $T = 37°C$ (310°K).
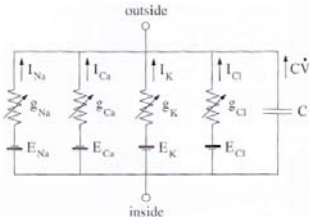

Figure 2.3: Equivalent circuit representation of a patch of cell membrane.

Animals

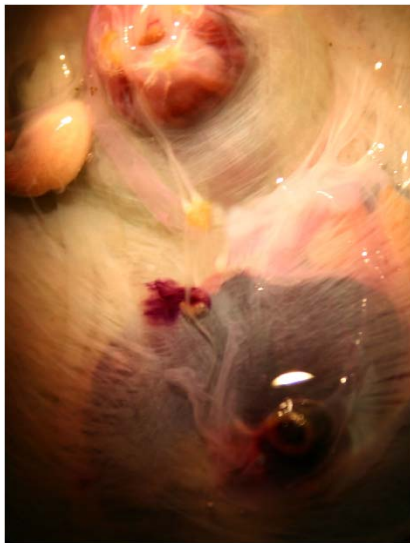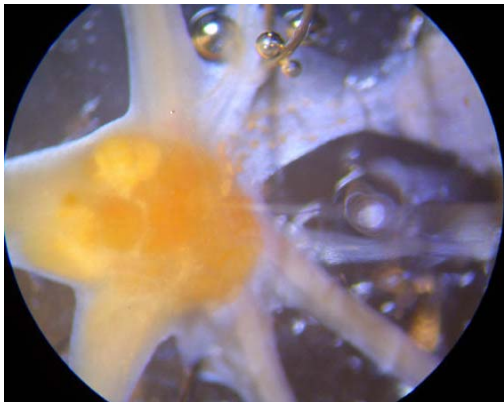*Lymnaea stagnalis*

Aplysia californica

Aplysia californica Dissection

Aplysia californica Visceral Ganglion

Artwork by J. DiCecco

Aplysia Action Potential Patterns
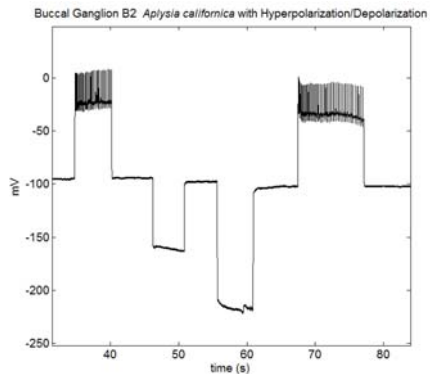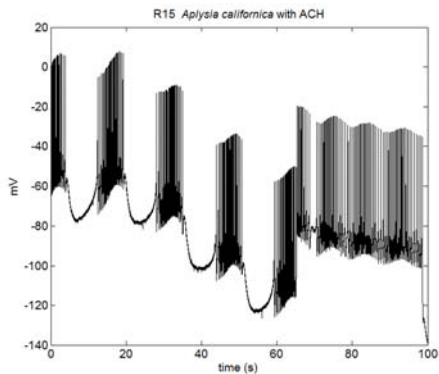
Lymnaea Dissection
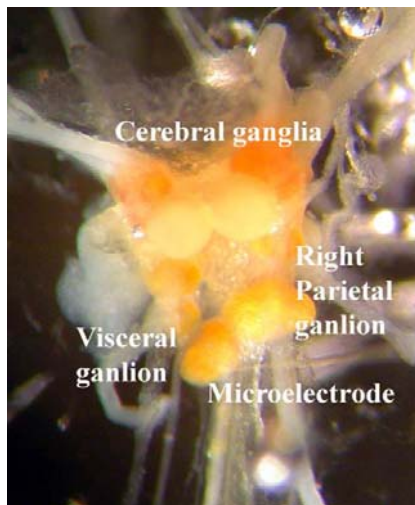
Lymnaea Action Potential Patterns
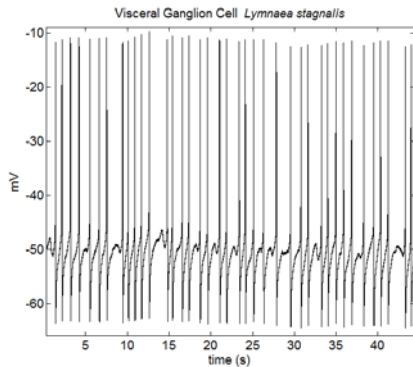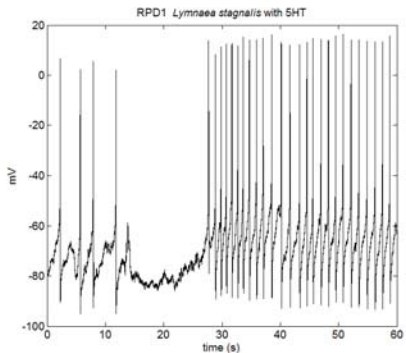
Hodgkin-Huxley

The Hodgkin-Huxley equations are given by:

$$C\frac{dV}{dt} = I_{inj} - \bar{g}_{Na}m^3h(V - V_{Na}) - \bar{g}_K n^4(V - V_K) - g_L(V - V_L) \quad (1)$$

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n \quad (2)$$

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m \quad (3)$$

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h \quad (4)$$

Where $C\frac{dV}{dt}$, $\frac{dn}{dt}$, $\frac{dm}{dt}$, $\frac{dh}{dt}$ are the total current flow across the membrane, potassium channel activation, sodium channel activation, and sodium channel inactivation, respectively. L is the leak current. α and β are the rate constants for the respective ion channels.

Hodgkin-Huxley

$$\alpha_n(V) = \frac{0.01(V + 55)}{1 - exp[-(V + 55)/10]} \tag{5}$$

$$\beta_n(V) = 1.125 exp[-(V + 65)/80] \tag{6}$$

$$\alpha_m(V) = \frac{0.1(V + 40)}{1 - exp[-(V + 40)/10]} \tag{7}$$

$$\beta_m(V) = 4 exp[-(V + 65)/18] \tag{8}$$

$$\alpha_h(V) = 0.07 exp[-(V + 65)/20] \tag{9}$$

$$\beta_n(V) = \frac{1}{1 + exp[-(V + 35)/10]} \tag{10}$$

The values of the constants are:
$C = 1$, $g_{Na} = 120$, $V_{Na} = 50$, $g_K = 36$, $V_K = -77$, $g_L = 0.3$, an $V_L = -54$
In these equations voltages are in mV, current densities in $\mu A/cm^2$, capacitance in $\mu F/cm^2$ and time in ms.

Reducing HH - Izhikevic

There exists a relationship between gating variables $n(t)$ and $h(t)$ (K^+ activation and Na^+ inactivation, resp.) such that $n(t) + h(t) \approx 0.84$. Plotting the variables on the (n, h) plane reveals that the relationship can be better described by $h = 0.89 - 1.1n$.

By substituting this relationship for h and assuming that the activation kinetics of the Na^+ current is instantaneous ($m = m_\infty(V)$), :

$$C \frac{dV}{dt} = I - g_{Na} m_\infty^3 (0.89 - 1.1n)(V - V_{Na}) - g_k n^4 (V - V_K) - g_L(V - V_L) \quad (11)$$

$$\frac{dn}{dt} = \frac{n_{\infty(V)} - n}{T_n(V)} \quad (12)$$
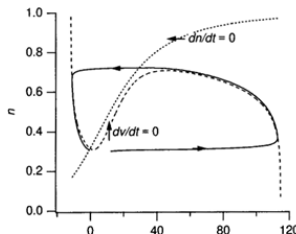
Nullcline - Briefly

The decision to fire or not fire is made at the resting state, which is at the intersection of the nullclines (or zero growth isoclines). This intersection is the location(s) of the fixed point(s) of the dynamical systems. Approximating the nullclines in this region of phase space is sufficient for maintaining the subthreshold dynamics of the system.
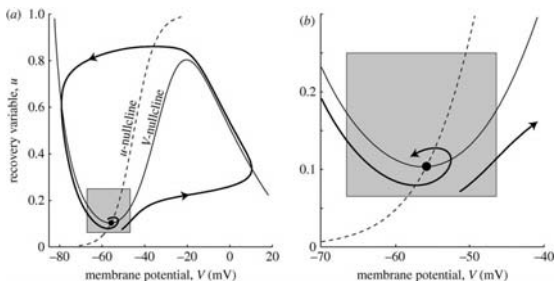
Nullcline - Briefly

Using these nullclines the subthreshold and spike initiation dynamics can be approximated by the system:

$$\frac{dV}{dt} = T_f p(V - V_{min})^2 - (u - u_{min}) \tag{13}$$

$$\frac{du}{dt} = T_s s(V - V_0)u \tag{14}$$

where T_f and T_s describe the fast and slow time scales. This system models the upstroke of an action potential. To model the downstroke the system is reset at Vmax $(V, u) \leftarrow (V_{reset}, u + u_{reset}), when V = V_{max}$. p and s are non-negative scaling coefficients derived from ionic time constants.

Reducing HH - Izhikevic

A typical V_{min} value of -60mV (human cortical) has been experimentally derived:

$$\frac{dV}{dt} = 0.04(V + 60)^2 - (u - u_{min}) \tag{15}$$

$$\frac{dV}{dt} = 0.04V^2 + 4.8v + 144 - u + I \tag{16}$$

$$\approx 0.04V^2 + 5V + 140 - u + I \tag{17}$$

$$\frac{du}{dt} = a(bV - u) \tag{18}$$

if $V \geq 30$mV, then $\begin{cases} V \leftarrow c \\ u \leftarrow u + d \end{cases}$

a (T_s), b (s), c and d are dimensionless parameters, V is the membrane potential and u is the membrane recovery variable, accounting for K^+ current and Na^+ inactivation. u also provides negative feedback to v.
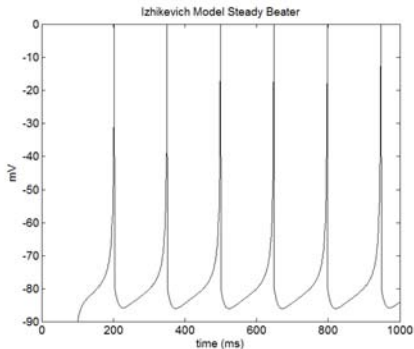
Izhikevic Model Software Simulation (MATLAB®)

```
C = 100; vr = -90; vt = -70; k = 0.7;
a = 0.03; b = -2; c = -80; d = 100;
vpeak = 0;

T = 1000; tau = 1;
n = round(T/tau);

v = vr*ones(1,n); u = zeros(1,n);
I = [zeros(1,0.1*n),70*ones(1,0.9*n)];

for i = 1:n-1

    v(i+1) = v(i) + tau*(k*(v(i)-vr)*(v(i)-vt)- u(i) + I(i))/C;
    u(i+1) = u(i) + tau*a*(b*(v(i)-vr) - u(i));
    if v(i+1)>vpeak
        v(i) = vpeak;
        v(i+1) = c;
        u(i+1) = u(i+1) + d;
    end
end

plot(tau*(1:n),v)
```
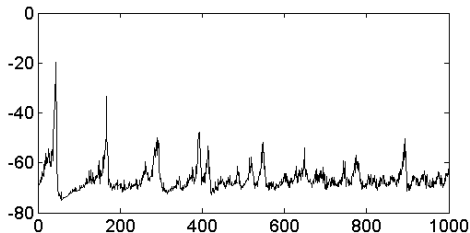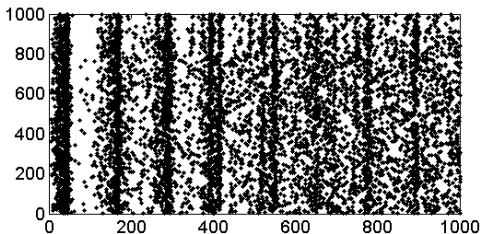

Izhikevich Model Steady Beater

Izhikevich NN Software Simulation (MATLAB$^{\circledR}$)

Basis for MOSFETs as Model for Differential Equations

One of the more basic electrical engineering concepts is that of current through a capacitor:

$$I(t) = C \frac{dV(t)}{dt}$$

where C is Capacitance in Farads. (Incidentally, this is the same equation for current through a lipid bilayer.)

By placing capacitors (or other energy storing units) in certain configurations, we can both describe systems with differential equations as well as solve them. It should be clear that in order to find the voltage across the capacitor, we need to integrate the current:

$$\int I(t)dt = C \int dV(t) \tag{19}$$

$$v(t) = \frac{1}{C} \int I(t)dt + k \tag{20}$$

This exactly what a capacitor does! And if ions are "injected" into a cellular membrane, the "sum" is analogous to the integration of charge.

Basis for MOSFETs as Model for Differential Equations

Lots of capacitors in MOSFETs, the gate capacitance in the oxide C_{ox} being the primary driver for current through the drain:

$$I_D = \underbrace{\mu C_{ox}}_{K_p} \frac{W}{L}(V_{GS} - \frac{V_{DS}}{2} - V_T)V_{DS}, \text{ for } V_{DS} > 0, V_{GS} > V_T, V_{GD} > V_T, \text{ linear region}$$

$$I_D = \underbrace{\mu C_{ox}}_{K_p} \frac{W}{2L}(V_{GS} - V_T)^2(1 + \lambda(V_{DS} - V_{DSat})), V_{DS} \geq (V_{GS} - V_T), V_{GS} > V_T, \text{ sat}$$

Transconductance parameter (K_p) is similar in many ways to the conductance parameters in the HH model (g_{Na}, g_K, g_L). We can achieve specific W/L ratios with specific transconductance coefficients.
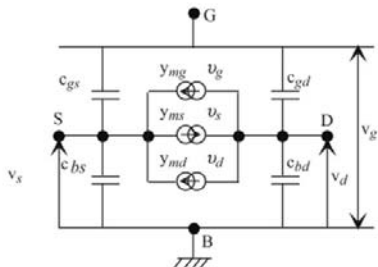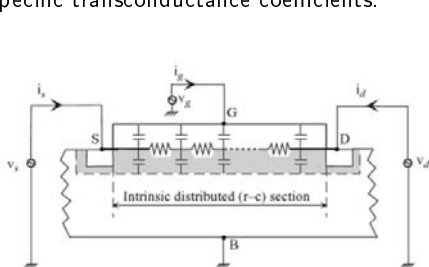
Wijekoon - Izhikevich Model

$0.35\mu m$ Process MOSFETs
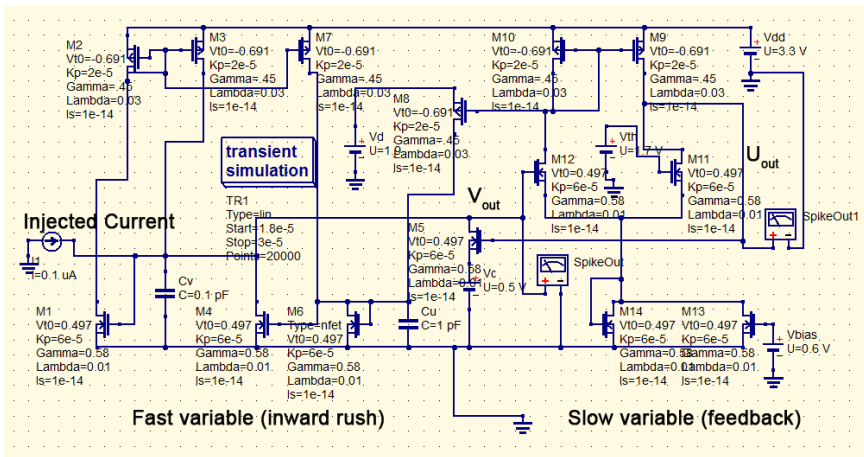
$$\frac{dV}{dt} = 0.04\,V^2 + 5\,V + 140 - u + I \qquad \frac{du}{dt} = a(bV - u)$$

Circuits Imitate Life
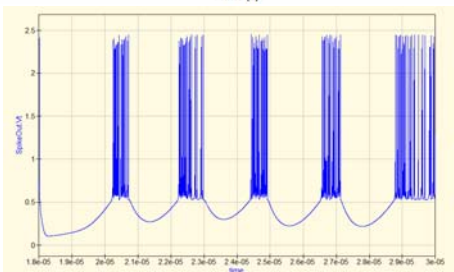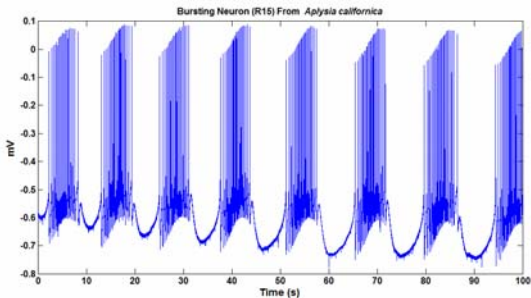
Circuits Imitate Life
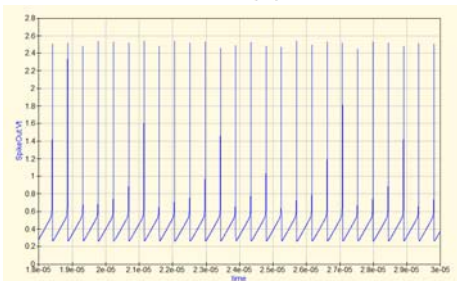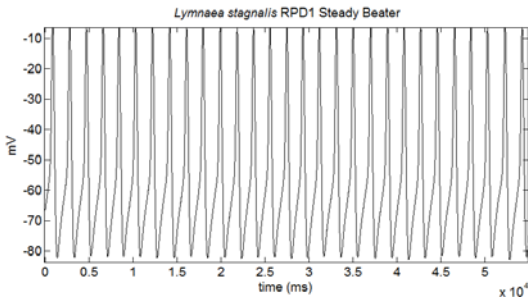
Wijekoon Circuit
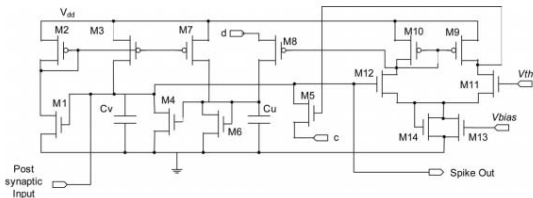
Figure: Original Circuit (0.35μm process)
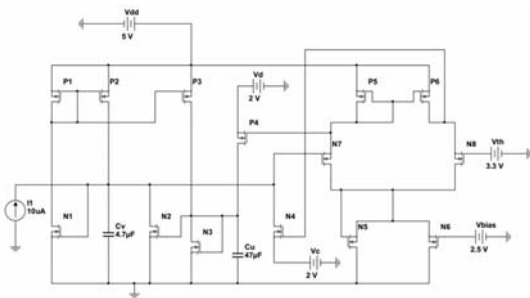
Figure: Adaptation (much bigger)

Circuit Simulation
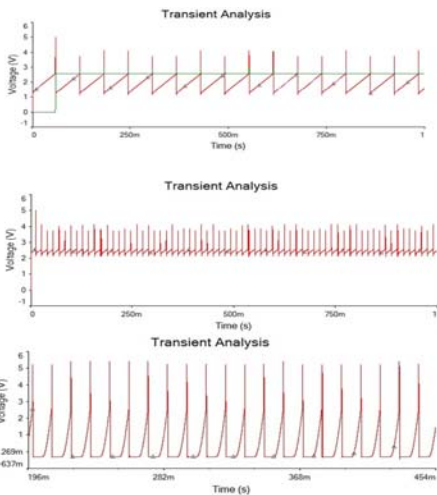
- Spice simulation has proven successful but ...
- Various neuronal firing patterns have been captured but simulation requires heavy computational power
- Designed circuit based on COTS MOSFETS

COTS MOSFET Neuron

- Still only 14 transistors (6 P-FETS, 8 N-FETS)
- Fully COTS
- Current Efforts to go to SMD and multiplicity
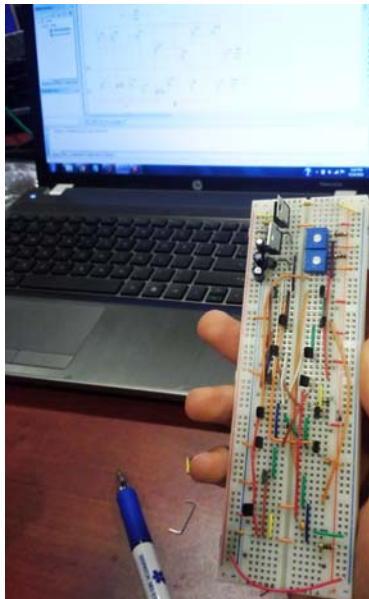
Our Hardware vs MATLAB®
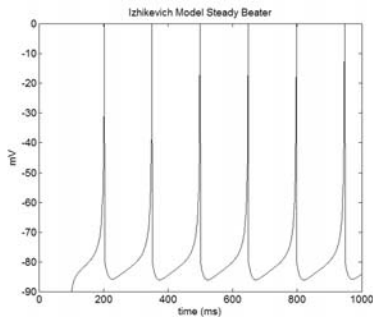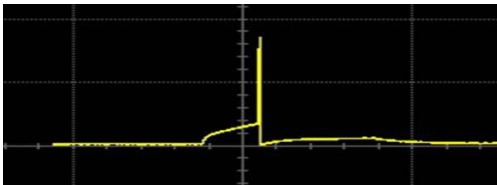
Step Response
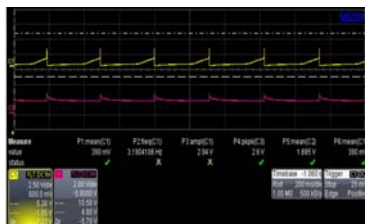
Regular spiking and low threshold spiking (interneurons)

Step Response
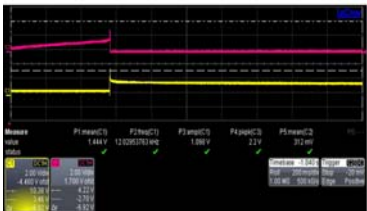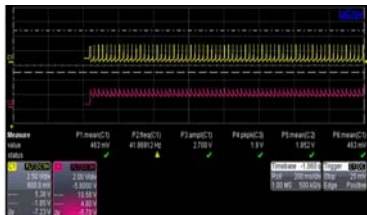
Fast spiking and Phasic spiking (stimulation detection)

Impulse Response

- After an impulse is transmitted to a neuron, a small sub-threshold oscillation occurs.
- It is the precursor to bursting activity.
- Caused by the interplay between the slow Calcium T current and the inward h-current.

- Characterized by a barely sub-threshold stimulation evoked by an impulse stimulation.
- Creates a significant delay between stimulation and fire.
- It is still not clear how or when the brain uses it but is suggested that latency encodes the strength of the input.
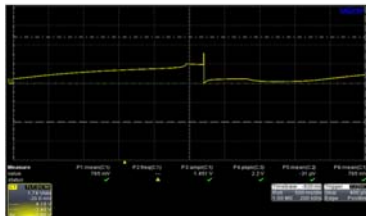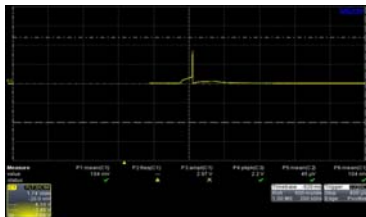
Impulse Response

- Excitable systems are defined based on two features: the coexistence of resting and spiking states and the existence of sub-threshold oscillations.

- Our circuit exhibits both.

- Bursts in response to a sufficiently long hyperpolarizing current.

- A long pulse of current invokes inward h-current, resulting in a voltage sag, and upon termination, generates a spike.

- Even if rebound depolarization does not elicit a spike, it may increase the excitability of the neuron.
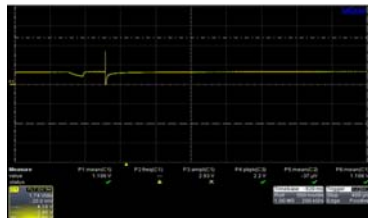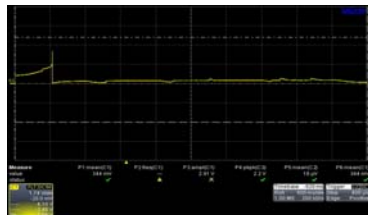
Current Configuration

- Code written to interface single-computer board to neuron through DAC
- Uses SPI protocol
- 16-bit resolution "should" provide necessary precision

Technical Challenges

- We have expertise available to us to march down the path of making a MOSFET with variable gate transconductance if necessary, which, to our knowledge, has not been attempted previously
- We MAY be able to avoid this in the digital interface by bringing multiple neurons into parallel and series configurations to effectively change W/L ratios
- Designing and fabricating the FPGA - will need collaboration here
- Implementing neural network algorithms to test hardware
  - Restricted Boltzmann Machine (RBM) or Pulse-Coupled Neural Network (PCNN)
- Mapping the models for various systems (next slide) onto a suitable number of neurons (and interneurons) with the correct dynamics
- Compensating for vagaries in the production of the MOSFETS

Vision

The ability to characterize the neuronal dynamics of a real biological system of interest (a.k.a. wetware) and drop it into a processor chip

- Biological systems of interest to replicate:
  - Sensorimotor control in fish
  - Vision in mammals, insects
  - Active acoustic sensing in bats, dolphins, whales
  - Passive acoustic sensing in owls, fish, insects, humans, etc
  - Olfactory sensing
  - Regulation of biochemical processes
  - General pattern matching circuits, i.e. human cortex
- Brain-machine interfacing
  - Basic motion control of prosthetics has already been demonstrated in chimpanzees and humans

Path Forward

- Solve issue with lack of bursting activity
- Fabricate circuit with multiple neurons with input/output interfaces - doing
- Grow the model onto an approximately 50 neuron board
  - The ability to interconnect neurons will require some mechanical (large) connections that will disappear with the FPGA implementation.
- Examine connectedness
  - This is the real bottleneck
  - Accurate (or at least efficient) modeling of the synapse and information coding
  - We cannot compete with large labs and their ability to fabricate smaller, exotic, devices
- Work to date is novel and publishable - no known example of a COTS implementation of this circuit
- Neuron interconnection dynamics will be the principal focus of follow-on funding
- Modeling complete systems (neural networks) such as the wetware systems highlighted on the previous slide is the principal cost driver in this funding request

Final Thoughts

At a conference during late 2009, IBM announced that it had used a supercomputer to simulate a brain with complexity similar to that of a cat's. The project simulated 1 billion neurons that share 10 trillion interconnections (synapses). But even with its abundant 144 terabytes of storage and some 147,000 microprocessors, the $\frac{1}{2}$-petaflop-per-second computing grid ran the cat-brain model 83 times slower than a real cat's brain.

Today, TrueNorth (IBM), 1 million neurons, 256 million synapses, from 5.4 billion transistors and 4096 neurosynaptic cores, using just 70mW. Human brain - 100 billion neurons, 100 trillion synapses, at 20W. We still have a LONG way to go!

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