Introduction to Neural Networks U. Minn. Psy 5038 Spring, 1999 Daniel Kersten

Lecture 2a





From Anderson (1995)

Basic Structure

Information flow: dendrites -> (soma -> axon hillock) -> axon -> terminal buds

Dendrites

A tree-like structure consisting of "dendrites" with special processes or connection sites called synapses. Much computational power is thought to reside in the strength of connections, and in the dendritic tree itself. In this course, we will primarily examine the computational properties of groups of simple neurons, rather than aggregates of dendrites in a single neuron.

Dendrites play the role of wires that convey information. Neural processes are tubes of ionized cytoplasm sitting in a bath of ionized fluid whose ionic composition is not that much different from "seawater". The inside of these tubes during resting state sits at about minus 70 millivolts relative to the outside of the cell. The tubes are on the order of microns in diameter, (but other processes, such as the axons discussed below, can reach half a millimeter or so) and the membranes 50 Angstroms (50 x 10-10 meters).

Signal transmission is limited by high resistance of the axoplasm, and high capacitance of the neural membrane. Information transmission consequences of these properties are:

• the voltage potential changes have a short range of influence, with the amplitude decreasing rapidly as one moves away from the synaptic source.

• the signals travel relatively slowly.

■ Soma and axon hillock

• Integrates dendritic signals

• The storage of electrical charge across the membrane, and the chemical nature of synaptic transmission leads to temporal integration of signals. This observation together with spatial integration of signals from the dendritic tree arriving at the axon hillock will lead to our basic model of the neuron.

■ Axon



How can the range and speed be increased?

Certain neurons are equipped with a specialized process called an axon that serves to "digitize" the data into all-or-none responses called action potentials or spikes. This digitization occurs at the axon hillock near the cell body. There is passive or electrotonic conduction along the dendrites up to the axon hillock at which point, if there is a sufficient potential change to reach threshold, an active process of depolarization kicks in leading to a spike.



Signals are carried by rapid (1 msec) voltage depolarizations going from -70 to +40 mV via Na+ influx, and K+ outflow through the membrane. From the axon hillock on, a myelin sheath serves to lower the capacitance and speed up conduction. However it interferes with the regenerative processes that preserve the all-or-none response. At periodic points (Nodes of Ranvier) the myelin sheath is interrupted where high extracellular concentrations of Na+ ions exist with sodium gates. When a small depolarization arrives, this decreases membrane conductance allowing an increased depolarizing influx of Na+, regenerating the spike.

Terminal arborization and terminal buds

Neurons with axons end in a terminal arborization. The terminal buds make synaptic contacts with the dendrites of subsequent neurons, and we have the beginnings of a neural network. Synaptic contacts can either be electrical or chemical, but more about these later.

Basic electrophysiology

Passive properties

Above we noted that the potential maintained by ionic imbalance (excess Na+ outside, and K+ inside). Balance between ionic concentration and electric field forces - determined by the Nernst equation (see Anderson text for a derivation).

We noted two problems: passing a signal over a long distance and with sufficient speed--BIG problem for an organism that has to transmit signals fast over a few feet. Let's take a more quantitative look at these problems that arise from the passive electrical properties of neuronal "electronics".

Model the passive electrical properties:

• across a small portion of the membrane by an "RC circuit" - low pass, i.e. favors signals with low temporal frequencies,

• over time and short lengths by the "Cable equation" (see pages 25-32 of Anderson, and Lecture 3 notes)

exponential drop-off of voltage with distance for constant current. Length constant about 2 mm. (1/e drop-off or 63% drop)

By solving the cable equation governing the voltage change over distance and time, we can get quantitative idea of how voltage drops with distance, and how voltage changes with time--change is not instantaneous.



From Segev (1992)

■ Solutions of the cable equation

The cable equation is given by:

$$V = \lambda^2 \frac{\partial^2 V}{\partial x^2} - \tau \frac{\partial V}{\partial t}$$

In the Notebook for Lecture 3, we'll see how to use *Mathematica* to find solutions to equations, including differential equations. For now, let's take the steady-state solution for a fixed voltage, V0, at a specific place, say x = 0, and see how the voltage drops away from zero. The voltage drops exponentially. Lambda is the "space constant", which for an axon would be about 3 to 5 mm.

$$V = \lambda^2 \frac{\partial^2 V}{\partial x^2}$$
$$V(x) = V_0 e^{-x/\lambda}$$

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V0 = 1; lambda = 2; (*Space constant*)
V[x_] := V0 Exp[-Abs[x]/lambda];
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Plot[V[x], {x,-4,4}, PlotRange->{0,1}];



Let's also plot the dynamical solution to the cable equation. Suppose a 1 volt step is applied across the neuron membrane at a point x=0. How does voltage change with time at this point? It doesn't immediately change to 1 volt, but grows gradually. A typical time constant is on the order of 1 or 2 msec.

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V(t) = 1 - e^{-t/\tau}
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Active (non-linear) properties



Action potentials, spike trains are nature's solution to the problem of fast long distance signalling.

"toilet flush model" - small push of the handle and a little leak, but reach a threshold, and the whole thing goes. Refractory period (absolute, and relative)

Relative refractory period- threshold gradually lowers with time--frequency coding.

.. and the solution to the decline in voltage signal over distance due to passive properties?

Action potential at one location provides the depolarization stimulus at a nearby spatial location, travels like a lit fuse. And how about the speed problem?

myelin sheath (insulation reduces leakage)

Nodes of Ranvier, action potentials jump from node to node, up to 30x.

Synaptic Integration

pre-synaptic potentials

post-synaptic potentials

excitatory (make the cell more likely to fire) and inhibitory (less likely to fire) post-synaptic potentials

long duration - fast EPSP is 1 to 2 msec rise time and 3 to 5 msecs decay (action potential 1-2 msecs)

synaptic delay - about 500 micro-seconds.

Interaction of synaptic potentials

axon hillock

algebraic? sometimes but not always

Our generic connectionist model introduced later will assume linear algebraic summation.



Anderson (1995). Interaction of inhibitory postsynaptic potentials (IPSP) and excitatory postsynaptic potentials (EPSP) can be linear (left column, A) or non-linear (right column, B). From Rall (1967). Dotted line shows linear prediction.





Slow potential at axon hillock waxes and wanes (because of low-pass temporal characteristics and the spatial distribution of the inputs) depending on the number of active inputs and their arrival times.

The slow integrated voltage potential now and then exceeds threshold producing an axon potential.

(note retina example: of the 6 types of cells, only 1 uses action potentials, the others communicate via slow potentials).

Further, if the slow potential goes above threshold, frequency of firing related to size of slow potential.

Noise

ion channels open and close probabilistically, quantized

neurotransmitter release in discrete packages - Poisson

over long distances spike train frequency is roughly like a Poisson process (better--an interval Gamma distribution) whose mean is modulated by the already noisy slow potential.

References

Koch, C. (1998). Biophysics of Computation : Information Processing in Single Neurons . Oxford: Oxford Univ. Press.

Koch, C., & Segev, I. (1998). <u>Methods in Neuronal Modeling : From Ions to Networks</u>. Cambridge, MA: MIT Press, 671 pages.

Segev, I. (1992). Single neurone models: oversimple, complex and reduced. Trends in Neuroscience, 15(11), 414-421.

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