

Abstract

The following document contains my thoughts on the paper ***Unsupervised learning of digit recognition using spike-timing-dependent plasticity***. It starts with a brief neuroscience primer where i account the necessary preliminaries for understanding the paper. Once the fundamental ideas are clear the paper is an easy read. Many of the equations are paper-and-pen derived by the author in an attempt to account them as very natural and intuitive. The section *extras* in chapter 2 is not part of the work but included for additional context.

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Chapter 1

Introduction

1.1 A brief neuroscience primer

For me it started with reading about great spanish painter turned neuro-anatomist Ramon Cajal. Probably his drawings marked the beginning of modern neuroscience. He proposed the neuron doctrine - neuron as the fundamental building block of brain.

The “Neuron Doctrine” and the “Theory of dynamic polarization”

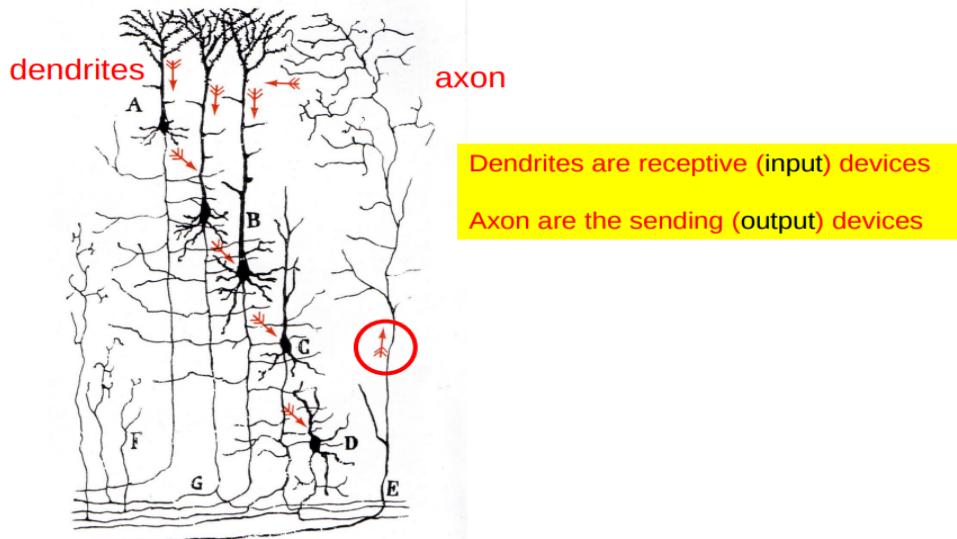


Figure 1.1: Cajal hypothesized the information flow from axon to the dendrites even though there was notable separation between the two.

Here's a neuron as an electronic device.(figure 1.3)

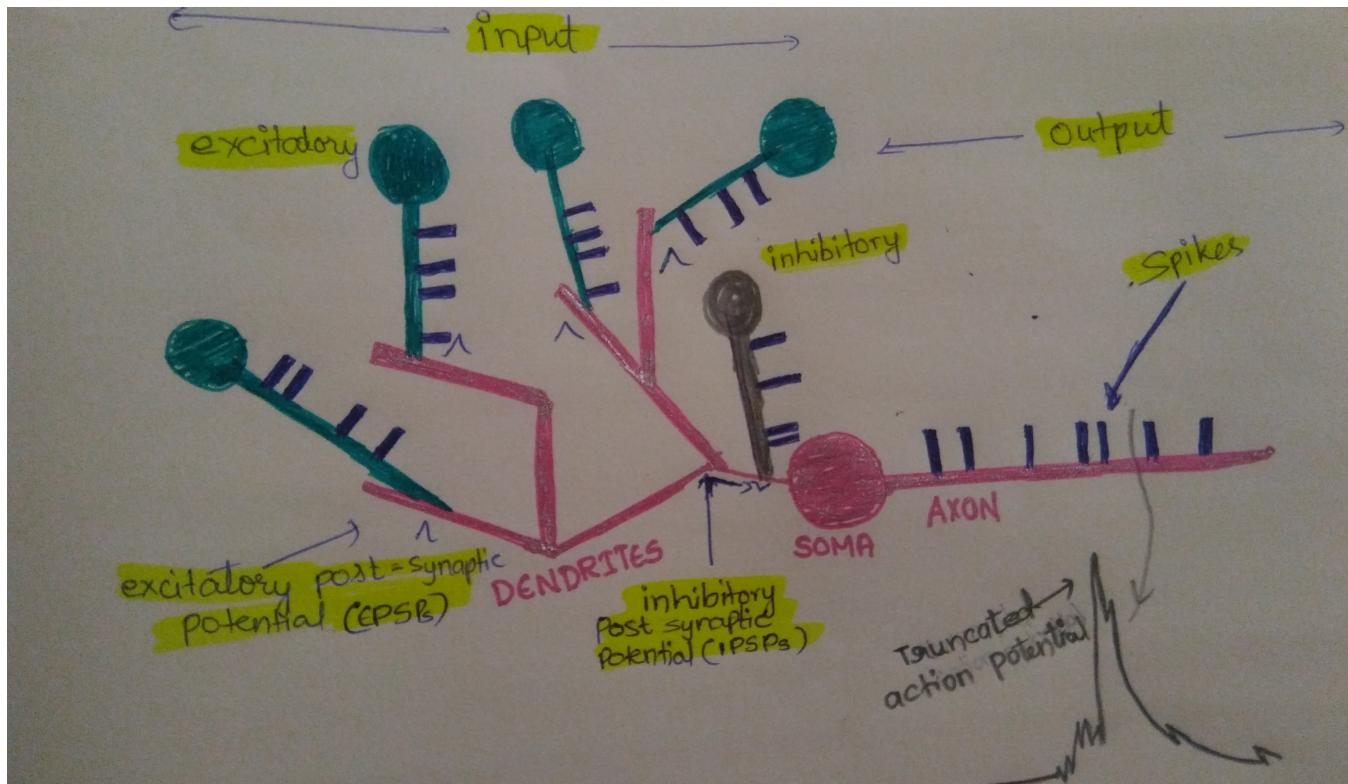


Figure 1.2: Temporal summation)

Biological neuronal network consists of thousands of axons attached to the dendritic tree of a neuron. The axon attaches to the post-synaptic spine(outgrowths on a dendritic branch) through a synapse that acts as a digital to analog converter. Axon carries only all-or-none signal(spike), whereas dendrites locally develop synaptic potential which is analog in nature. So axons from local or faraway neurons reach post-synaptic neuron's dendritic tree and instill local voltage growth which if of sufficient concentration (If helps in reaching threshold voltage) might trigger a spike that is carried through the axon. The dendritic connections might be either of inhibitory or excitatory in nature.

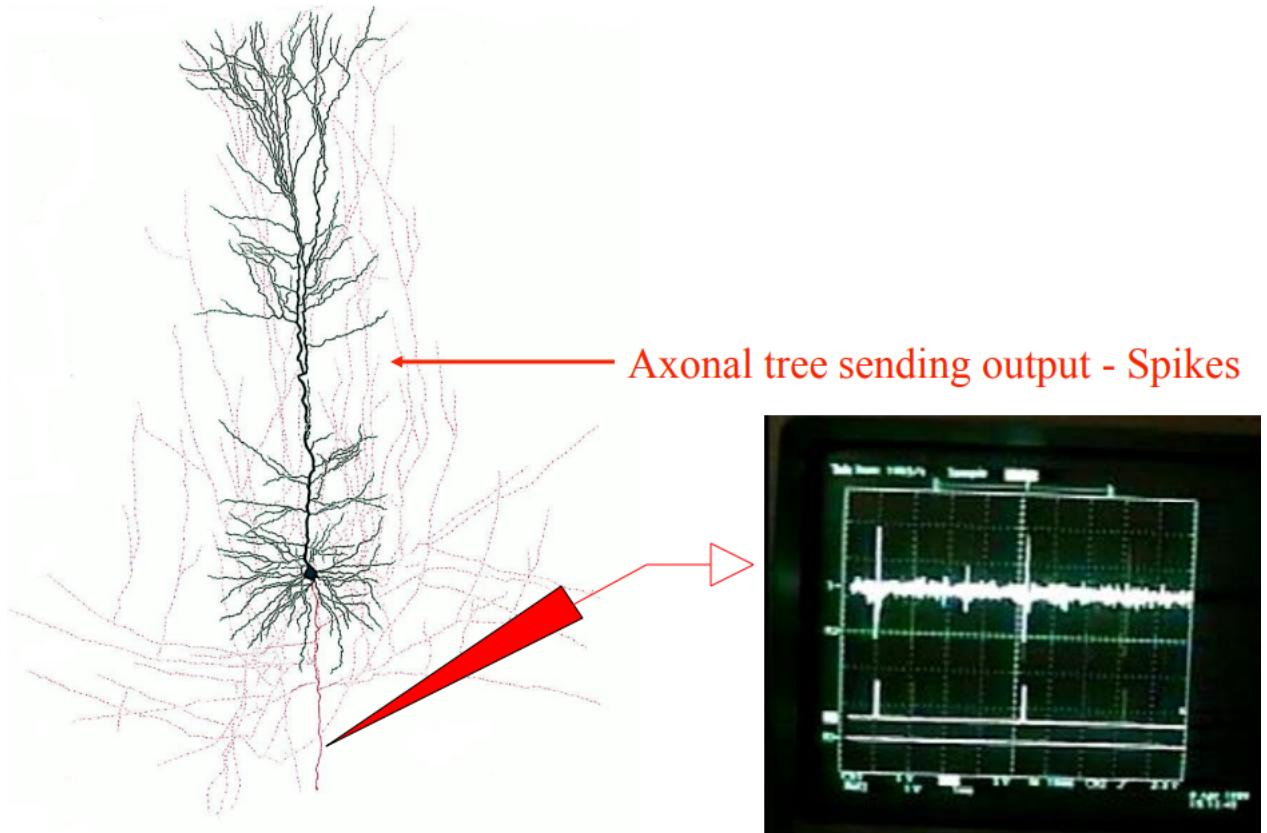


Figure 1.3: Axon-spike train(brain-code)

Here, I will try to develop mathematical model of the neuron. The figures below are self contained and they terminate at the celebrated Hodgkin-Huxley model. These form the backdrop for understanding the modeling in next chapter.

Let's start with modeling the dendritic membrane (figure 1.4, 1.5). We will assume that it's equipotential. In early twentieth century people have stuck electrodes into these membranes and sent small currents. They observed that the following electric potential profile which resembles a typical RC circuit.

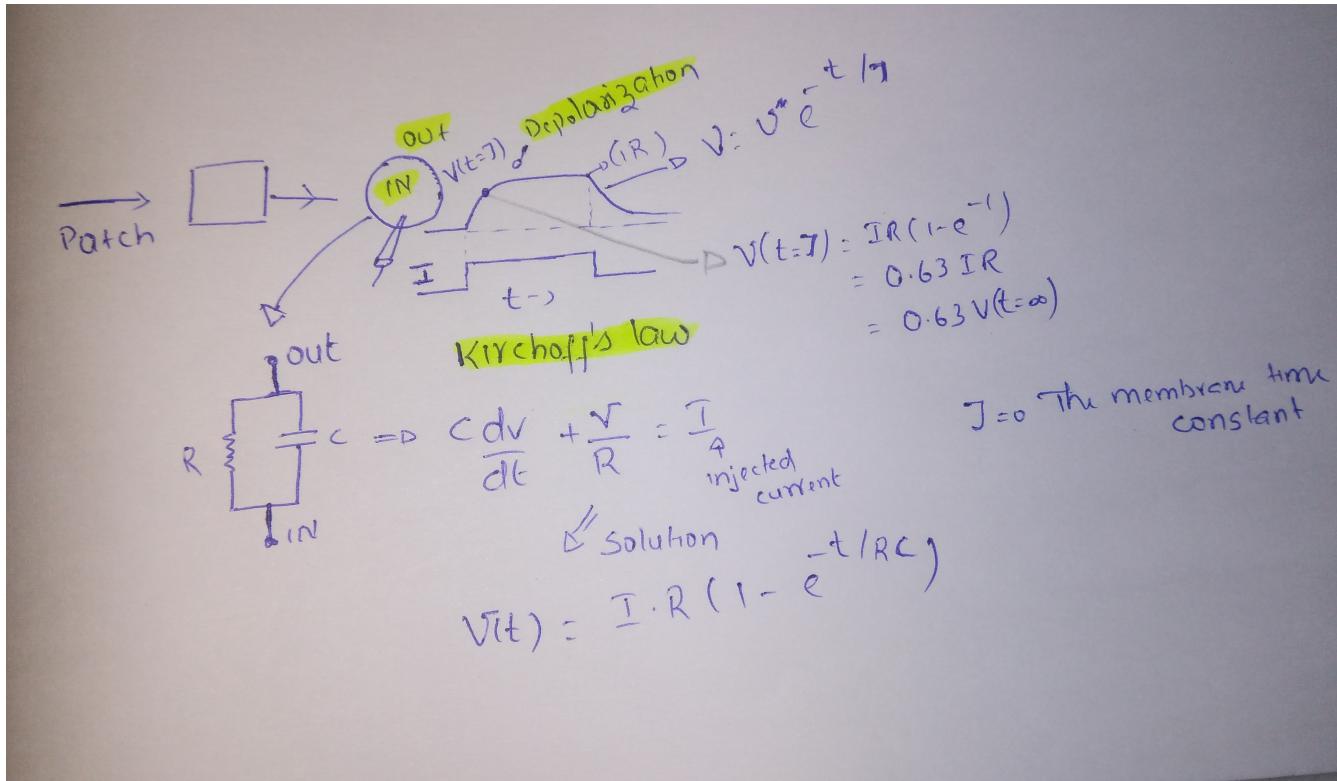


Figure 1.4: A Naive first order approximation

Now let's get a bit more realistic (figure 1.6). In a real neuron the potential difference is created due to the presence of charged ions. (typically sodium and potassium). The dendritic spine has a membrane which has some permeable channels that are geared towards maintaining potential difference of -70mv (approx). In the context of present paper, it's important to see how a train of spikes affect the dendritic potential (figure 1.5). Each new spike grows the potential from the previous spikes memory (presynaptic trace).

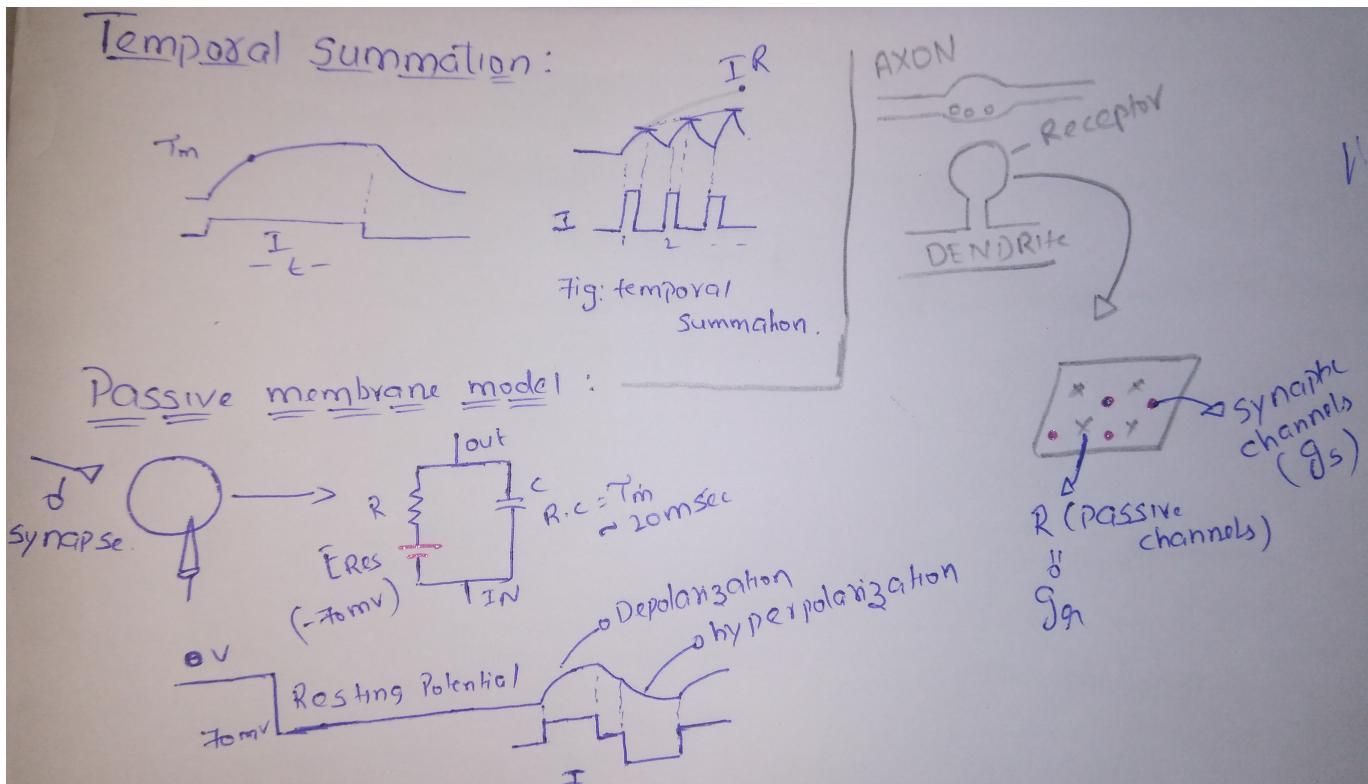


Figure 1.5: Temporal summation

When a spike propagates through the axon, at the dendritic terminal neurotransmitters are released that control the selective opening of ion channels. This further changes the current flow into the dendrite. Thus we model it through active dendritic battery and conductance.

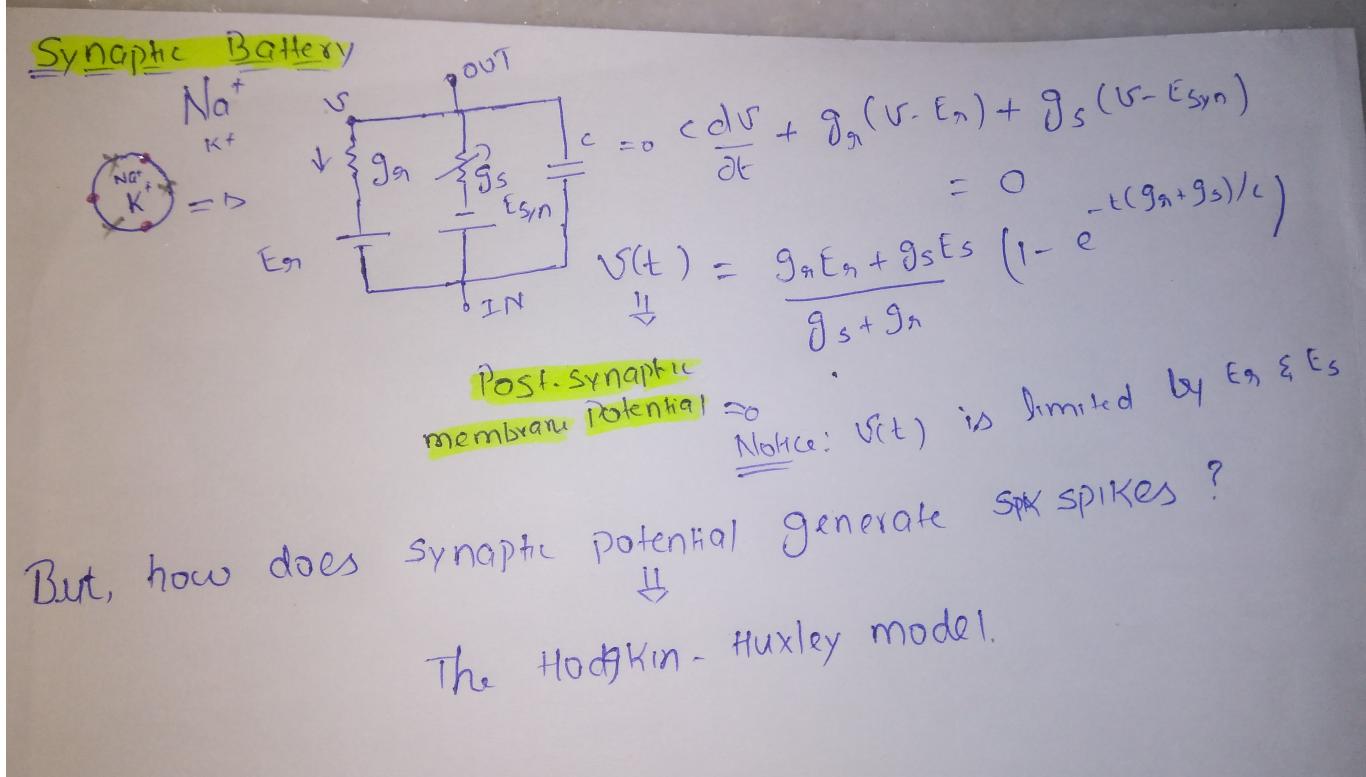


Figure 1.6: Synaptic Battery

1.1.1 The spiking heroes- Hodgkin and Huxley

Probably, the most successful theory in neuroscience is given by these guys.

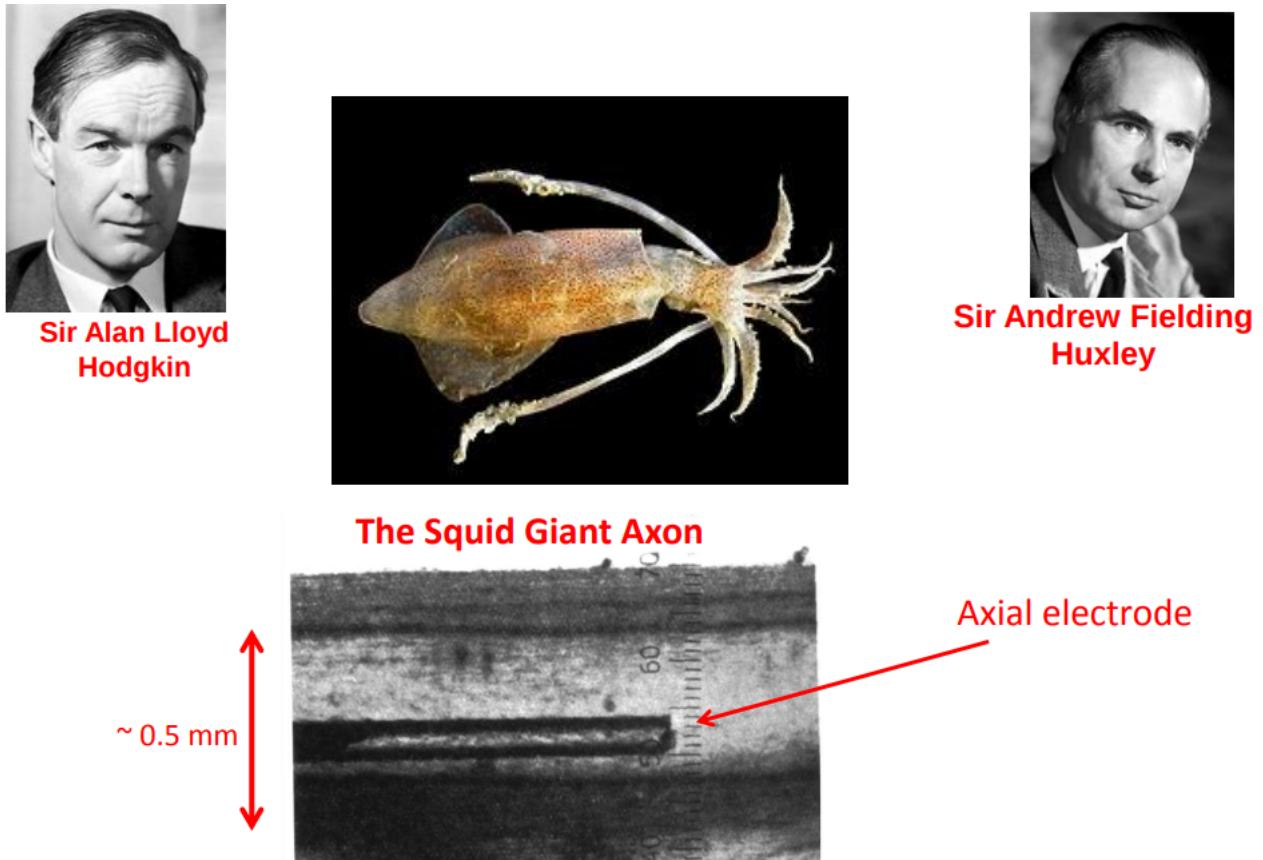


Figure 1.7: The hodkin-huxley experiment

They drove an electrode into the squid's axon and measured the spike directly through external stimulation. This marked the beginning of series of experiments that culminated in a set of four ordinary differential equations. Together they beautifully explain the phenomenon of spike(all or none signal) in the neuron through membrane potential.

$$I = C_m \frac{dV}{dt} + g_{Na} h m^3 (V - V_{Na}) + g_K n^4 (V - V_K) + G_L (V - V_L) \quad (1)$$

$$\frac{dm}{dt} = \alpha_m (V) (1 - m) - \beta_m (V) m \quad (2)$$

$$\frac{dn}{dt} = \alpha_n (V) (1 - n) - \beta_n (V) n \quad (3)$$

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

Figure 1.8: The spiking equations

1952 was a magical year for neuroscience. Six papers by three noble laureates Hodgkin, Huxley and Katz were published that year. The major driving point was development of two ingenious experimental techniques - the voltage and space clamp techniques. The details and derivation of the equations are omitted here for the lack of relevance to the present paper. But the following inferences are important for the paper:

1. The frequency of spikes is limited. (Refractory period of about 10ms.)
2. Post synaptic conductance change is modeled through an exponential raised to the power 4.

When Hodgkin-Huxley increased the voltage inside the axon to superthreshold region (using voltage clamping) they observed the weird nature of the current profile (figure 1.9).

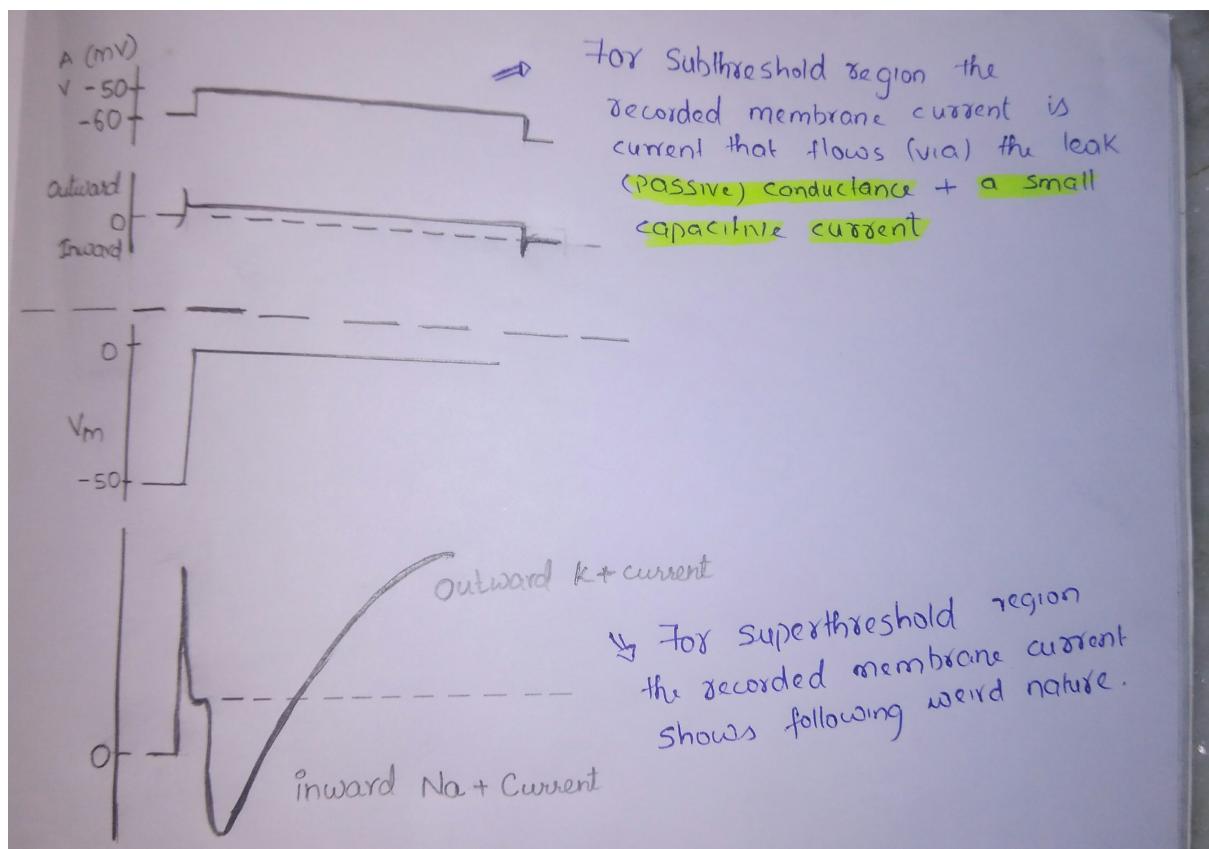


Figure 1.9: The observed current profile

which they attributed to the time variation of the conductances of sodium and potassium channels. The precise variation is modeled by n which is raised to power 4

to fit the experimental results. Also the phenomenon of the refractory period is also well explained by the parameters Na inactivation(h) and slow kinetics of potassium conductance which make the following spike impossible.

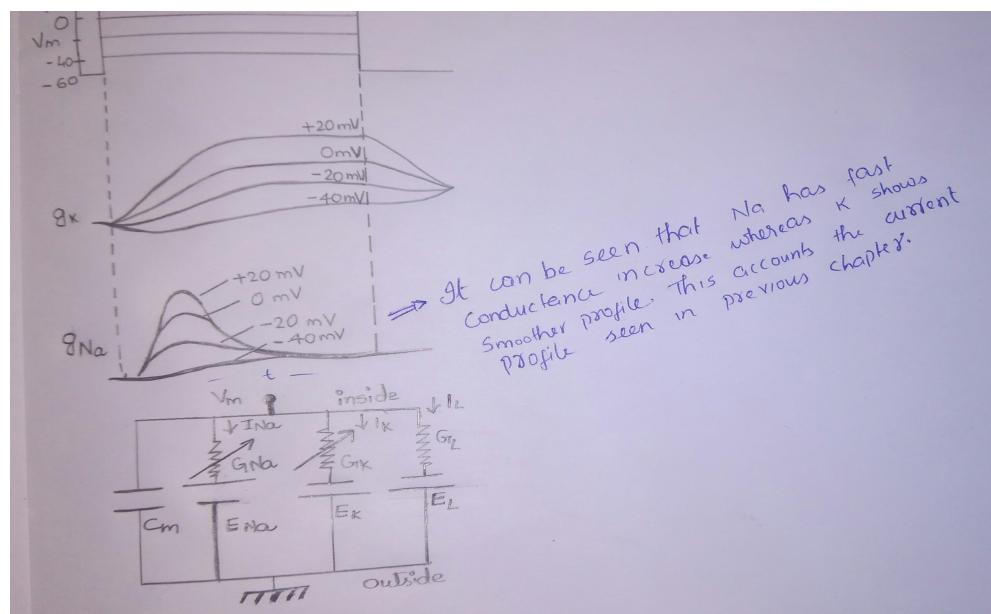


Figure 1.10: Experimental post synaptic conductance variation.

Chapter 2

The details of the experiment.

2.1 Introduction

Now that we have covered considerable background let us see things from authors point of view. They wanted to build biologically plausible neuronal network that also excels at learning. As outlined in previous section we can model brain at varying levels of detail. What level of detail is essential for intelligence is still a matter of debate. But they chose simple leaky integrate and fire model (which is a nice balance between biological plausibility and ease of implementation).

Most learning rules are formulated in terms of mean firing rates, viz., a continuous variable reflecting the mean activity of a neuron. For example, a 'Hebbian' (Hebb 1949) learning rule which is driven by the correlations between presynaptic and postsynaptic rates may be used to generate neuronal receptive fields. Rate based approach neglects the pulse structure. Some experiments (recent) suggested to show temporal correlations of spike timing even on millisecond level - STDP. Present authors took STDP as biologically plausible mechanism for learning which i doubt but for the time being STDP is our model. Anyways before reviewing it let's look at some interesting details of their model. I have covered lot of ground in the previous chapter, particularly remember the variation of synaptic weight (due to Na and K channels). Here we will try to model them reasonably faithfully:

1. A leaky-integrate and fire model is used to model a neuron.

$$\tau \frac{dV}{dt} = (E_{rest} - V) + g_e(E_{exc} - V) + g_i(E_{inh} - V)$$

Figure 2.1

2. As i explained in previous section how conductance change at synaptic junction gives rise to neuronal plasticity (figure 2.2 shows the weight update for hebb's rule but here i cannot account for the incompleteness of the rule but similar modeling can be done for STDP.)

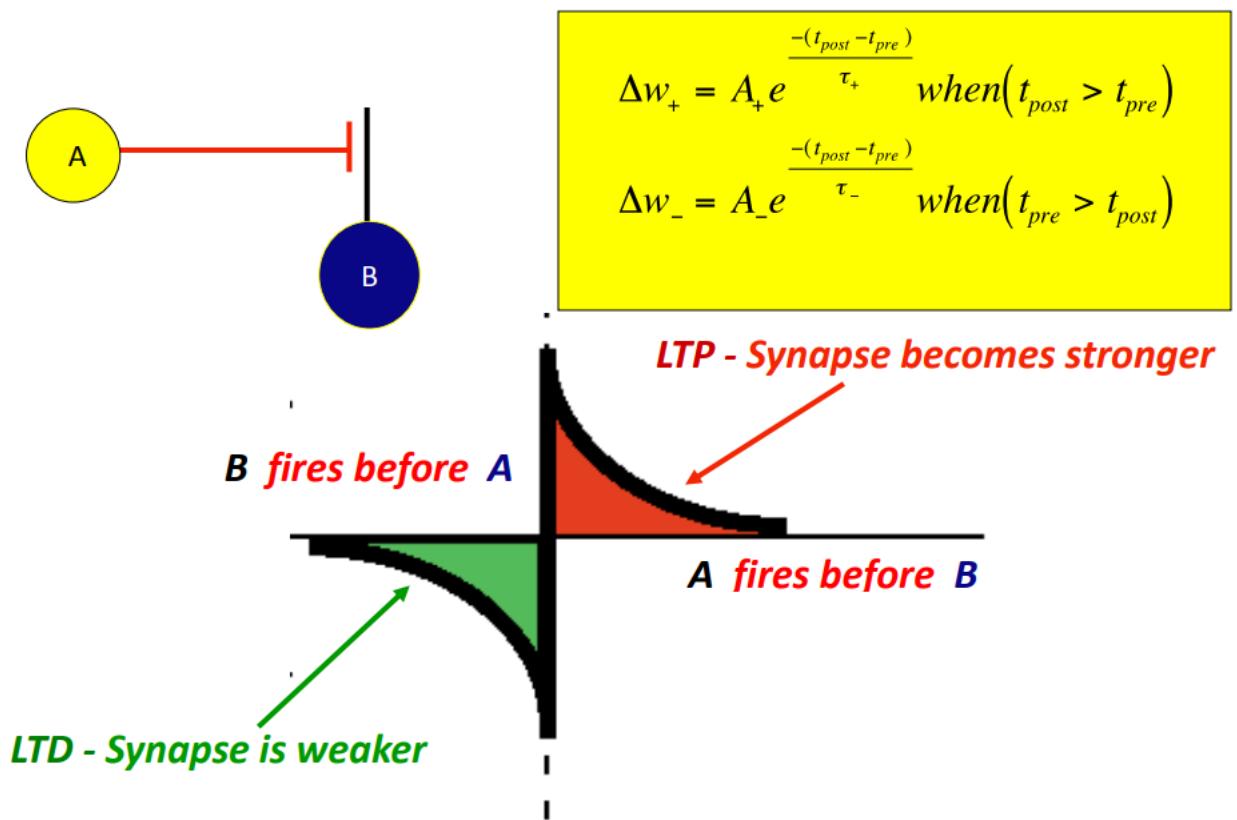


Figure 2.2: Hebbian weight change

3. Synaptic conductance increase by weight 'w', otherwise decreases exponentially. (If this is not clear refer figure 1.10)

$$\tau_{g_e} \frac{dg_e}{dt} = -g_e$$

Figure 2.3

4. They used membrane time constant of 100ms (note in previous chapter we found the actual values to be around 20 ms). But, this shouldn't be of concern as it is used to protect from the affect of noise (Note: Input is modeled as poisson spike train with average rate proportional to intensity of pixel.)

The network architecture.

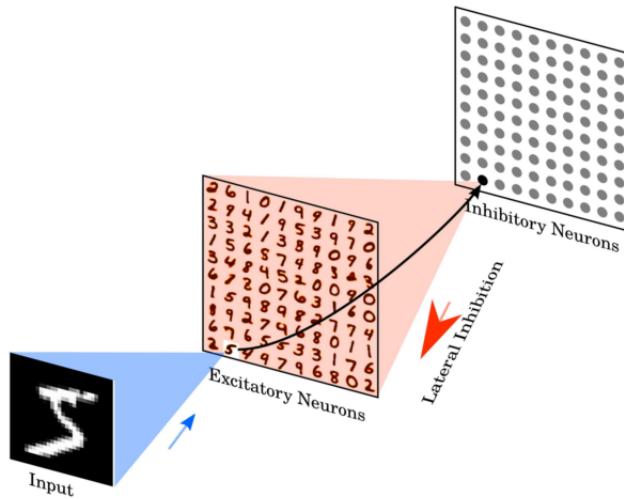


FIGURE 1 | Network architecture. The intensity values of the 28×28 pixel MNIST image are converted to Poisson-spike with firing rates proportional to the intensity of the corresponding pixel. Those Poisson-spike trains are fed as input to excitatory neurons in an all-to-all fashion. The blue shaded area shows the input connections to one specific excitatory example neuron. Excitatory neurons are connected to inhibitory neurons via one-to-one connections, as shown for the example neuron. The red shaded area

denotes all connections from one inhibitory neuron to the excitatory neurons. Each inhibitory neuron is connected to all excitatory neurons, except for the one it receives a connection from. Class labels are not presented to the network, so the learning is unsupervised. Excitatory neurons are assigned to classes after training, based on their highest average response to a digit class over the training set. No additional parameters are used to predict the class, specifically no linear classifier or similar methods are on top of the SNN.

Figure 2.4

6. But, what's the intuition? How does this simple setting at all result in learning? (refer figure 2.5, 2.6)

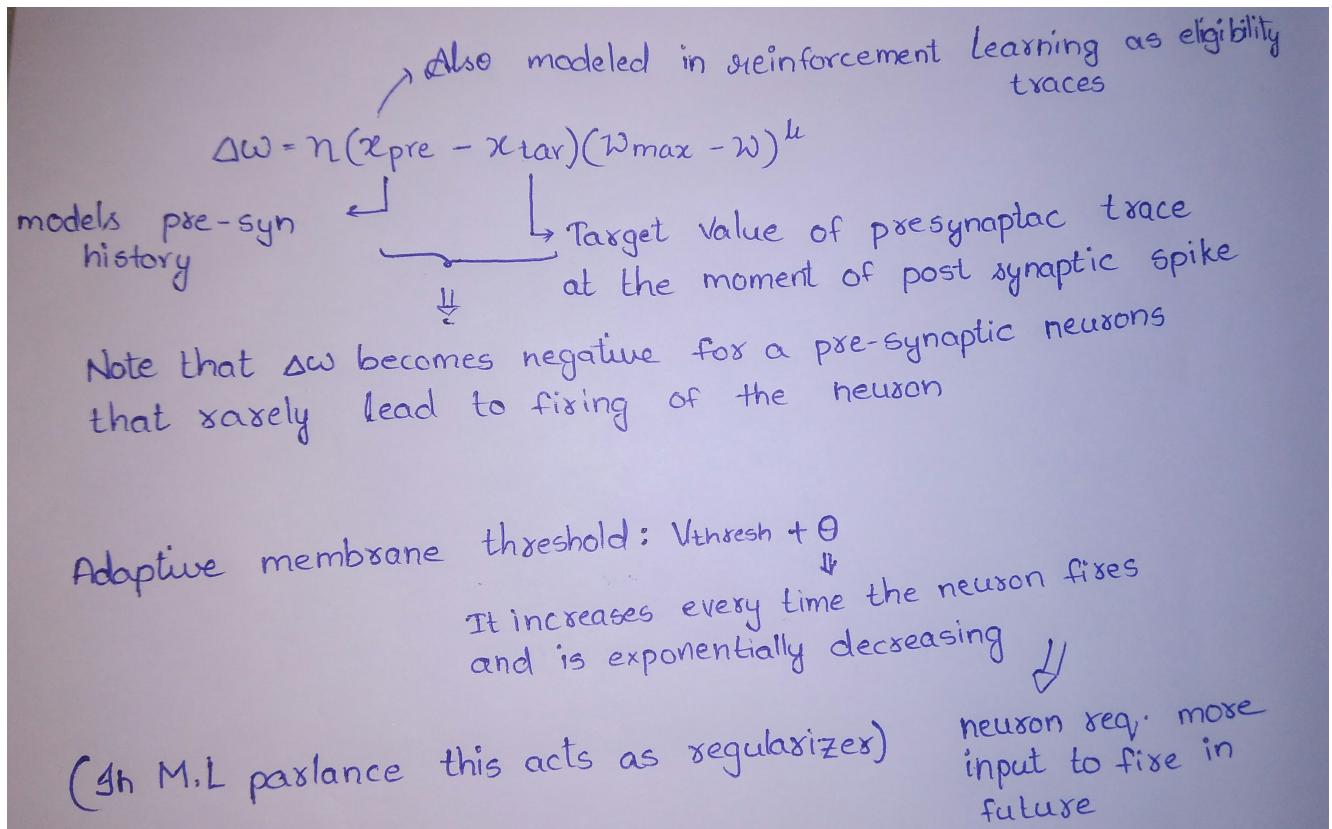


Figure 2.5

What's happening in the network ?

Sort of competition based learning is happening here

- * Each neuron learns the prototype of an input or average of similar inputs.
- * The winner prototypes are used to predict the class of input and weights are adopted towards present input.
- * Difference in lateral inhibition prevents them from becoming too similar to each other. Thus a group of neurons can capture the variability of a class.
- * Competition bt neurons forces them to learn as diff input patterns as possible. Note that change in weight depends on present weight which inhibits them becoming too large if input patterns does not reflect it.



Explains the stability of network

Figure 2.6

They have used a very specific architecture(though they tested for robustness of learning method but not of architecture) with equal number of excitatory and inhibitory neurons which is not biologically plausible. But, they explained that by incorporating many to one connections from excitatory to inhibitory neurons and increasing the network size similar effect can be achieved.

Why did they try different learning rules ?

Our brain seems to implement different learning rules based on the region.(atleast as far as experiments are concerned). Thus variability in learning rules ensures robustness of the model.(refer *extras* for further info)

2.2 A more complete look at learning in brain.

Learning invites change. Brain must undergo some physical change to encode the ongoing learning process. These can be attributed to:

1. Growing new cells - neurogenesis.
2. New synaptic connections are formed - structural plasticity. (Ramon cajal)

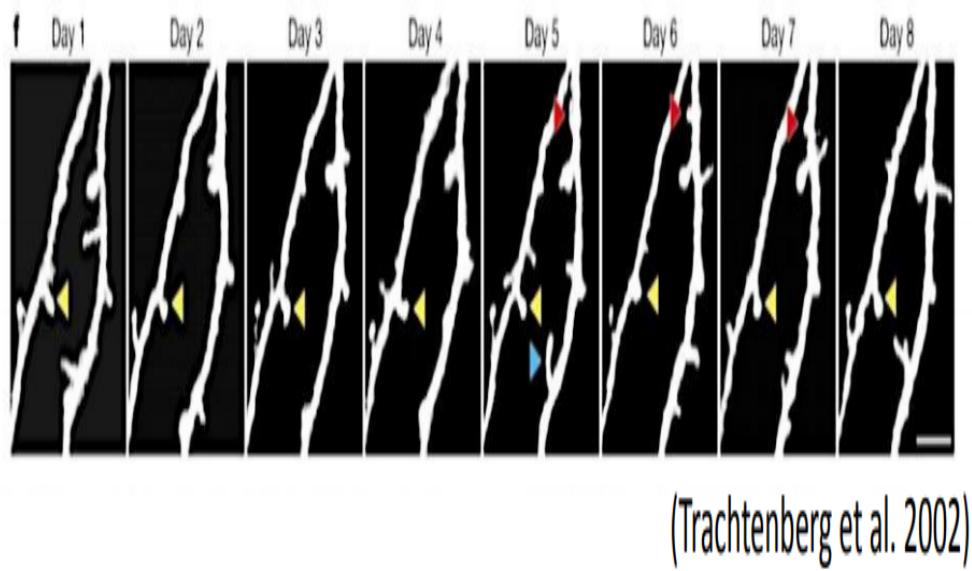


Figure 2.7: A 2002 study showing new spines appearing and disappearing frequently whose role in learning is not yet known

Synaptic plasticity is attributed to both growth of new spines and functional plasticity.

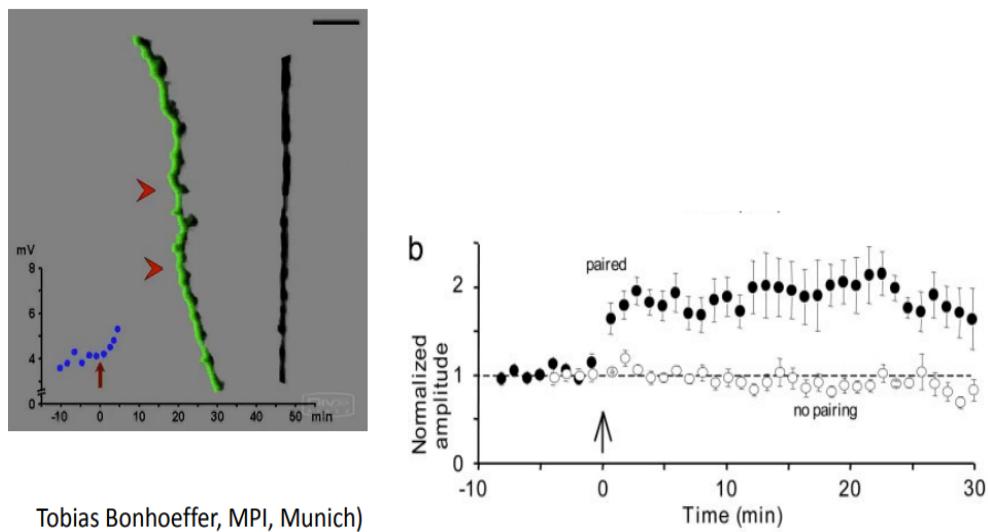


Figure 2.8: LTP lasting for minutes/hours due to structural plasticity

3. Strength of synaptic connections changing - functional plasticity.(Hebb's rule,STDP.)

This shows brain has far richer mechanism to accomodate our thoughts, reasoning fallacies and memories.

2.2.1 A deeper look at STDP.

It's important to realize that people who observed STDP,initially stuck two electrodes one into pre synaptic and other into post synaptic neurons.It is then noticed that after alternative excitation of the electrodes,depending upon the order LTP or LDP is observed.

Lisman, J., and Spruston, N. (2005) commented on STDP which is reproduced in ***Frontier in neuroscience - spike timing dependent plasticity - A comprehensive overview***.They essentially provided compelling arguments against STDP as underlying mechanism for neuronal plasticity.Again STDP can't account for long range correlations like pavlov learning(In paper it is stated to be useful in reinforcement learnig) where reward and stimuli are separated in seconds range.

2.3 Extras :

When i first read the paper i doubted the authors suggestion of superiority of unsupervised learning.Of course,there is no direct feedback of gradients to the neurons.But,there can be some sort of feedback that the correction to the earlier layers.I am impressed by Geoffrey Hinton's attachment to backpropagation.If backprop is so implausible than why is he so stuck on it ?(refer Geff's talk at stanford - Can the brain do backpropagation ?).Let's see why biologically backprop is so implausible:

1. There is no obvious source of supervised signal.
2. Cortical neurons do not communicate real valued activities.
3. The neuron need to send two different types of signals: a)the output ' y ' b)the gradient of ' y ' w.r.t input.
4. Neurons do not have symmetric reciprocal connections.

But,what if we could show that none of them are really insurmountable problems.Goeff does exactly this in his talk.I strongly suggest listening to it.

Anyways, from first principles and mounting evidence from brain studies we can conclude:

- 1.Learning happens through heirarchical layers(Hubel and wiesel) each gaining from the previous layers feature extraction.
- 2.There must be some sort of feedback signal coming to the previous layers,for learning to at all be feasible.

Now,the question is how does cortex implement learning.Though i cannot tell you that with certainty,as explained earlier STDP doesn't seem to provide enough evidence to account for synaptic plasticity.

As i noted earlier they argue that investigations of STDP process have been done under conditions in which the spike is evoked by postsynaptic current injection. Under more realistic conditions, in which the spike is evoked by the EPSP, the results do not generally support STDP. For instance, low-frequency stimulation of a group of synapses can cause LTD, not the LTP predicted by the pre-before-post sequence in STDP; this is true regardless of whether or not the EPSP is large enough to produce

a Na⁺ spike. With stronger or more frequent stimulation, LTP can be induced by the same pre-before-post timing, but in this case block of Na⁺ spikes does not necessarily prevent LTP induction. Thus, Na⁺ spikes may facilitate LTP and/or LTD under some conditions, but they are not necessary, a finding consistent with their small size relative to the EPSP in many parts of pyramidal cell dendrites. The nature of the dendritic depolarizing events that control bidirectional plasticity is of central importance to understanding neural function. There are several candidates, including backpropagating action potentials, but also dendritic Ca²⁺ spikes, the AMPA receptor-mediated EPSP, and NMDA receptor-mediated EPSPs or spikes. These often appear to be more important than the Na⁺ spike in providing the depolarization necessary for plasticity.

Presence of triplet learning rule in brain :

standard STDP models have expressed the weight change as a function of pairs of presynaptic and postsynaptic spike. Unfortunately, those paired-based STDP models cannot account for the dependence on the repetition frequency of the pairs of spike. Moreover, those STDP models cannot reproduce recent triplet and quadruplet experiments. Here, we examine a triplet rule (i.e., a rule which considers sets of three spikes, i.e., two pre and one post or one pre and two post) and compare it to classical pair-based STDP learning rules. With such a triplet rule, it is possible to fit experimental data from visual cortical slices as well as from hippocampal cultures. Moreover, when assuming stochastic spike trains, the triplet learning rule can be mapped to a Bienenstock-Cooper-Munro learning rule. - William Gerstner.