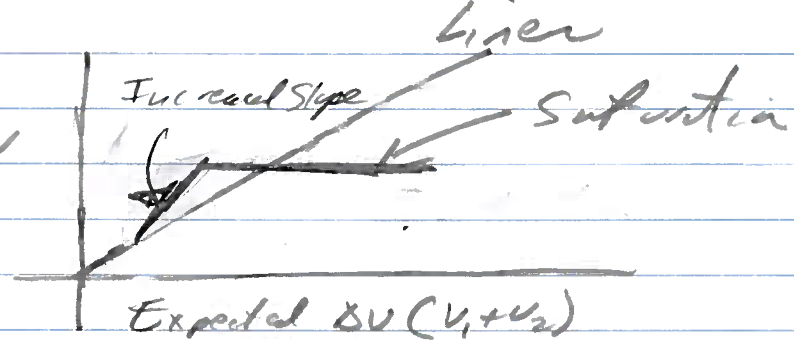
Dendritic Computation Notes:

(23 Feb 2011)

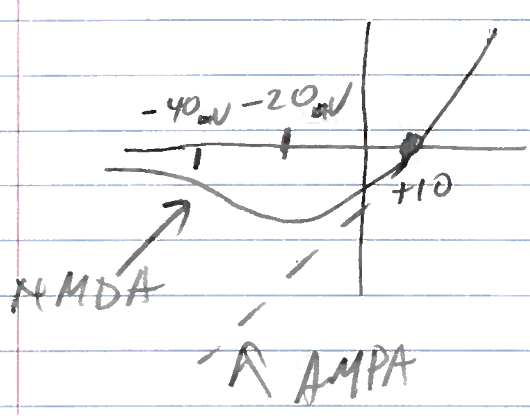
1/3

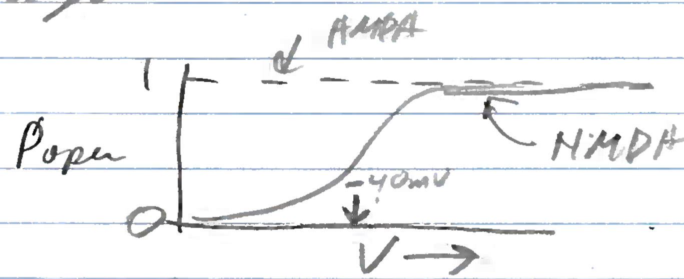
The experiments of Schiller looked at the amplitude of the EPSP in response to pairs of inputs into the same dendritic branch versus a different branch. Schiller finds



The idea here is that some non-linearity in a group of fine dendrites leads to increased gain and saturation, so that the group has a voltage dependence not unlike the output of a Perceptron.

The idea is that the synaptic current increases with depolarization as one of the two glutamate channels depends on voltage as well as agonist





$$P(V) = \frac{1}{1+e^{-ze(V-V_{on})/k_BT}}$$

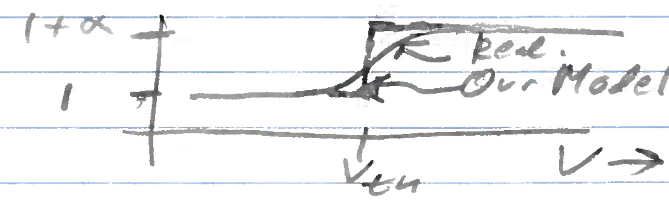
↑ about -40mV

Ckt Model

AMPA → 1, NMDA → $\alpha P(V)$, α = fraction of NMDA current.

$$0 = G_L(V-V_L) + G_{syn}\underbrace{[1+\alpha P(V)]}_{=Q(V)}(V-V_{syn}) = 0$$

Q(V)



With no synaptic input, $V_{ss} = V_L$

<u>Weak Input</u>: $Q(V) = 1$

$$V_{ss} = \frac{G_{syn}V_{syn} + G_L V_L}{G_{syn} + G_L}$$

$$\Delta V_{ss} = V - V_L = \frac{G_{syn}}{G_{syn}+G_L}(V_{syn}-V_L)$$

$$\xrightarrow[G_{syn} \ll G_L]{} \frac{G_{syn}}{G_L}(V_{syn}-V_L)$$

Twice this change is $\frac{2G_{syn}}{G_L}(V_{syn}-V_L)$

On the other hand, if we doubled the input to $2G_{syn}$, change in voltage is still

$$\Delta V_{ss} = \frac{2G_{syn}}{G_L}(V_{syn}-V_L)$$

So linear response for "weak" input

and $V < V_{th}$, even with $2G_{syn}$ input.

Now, suppose that input with $2G_{syn}$ gives a voltage change that exceed V_{th}. Then $2G_{syn}$ is replaced by $2(1+\alpha)G_{syn}$.

$$\therefore \Delta V = \frac{2(1+\alpha)G_{syn}}{G_L + 2(1+\alpha)G_{syn}}(V_{syn} - V_L)$$

$$\xrightarrow[G_{syn} \ll G_L]{} \frac{G_{syn}}{G_L}(1+\alpha)[V_{syn} - V_L]$$

It is is larger by a factor of $(1+\alpha)$! So the boost by turning on the NMDA receptor leads to increased slope.

How large can G_{syn} be? Nonlinear gain is lost when

$$\frac{G_{syn}}{G_L} > \frac{\alpha}{1+\alpha}$$

This model accounts for increased slope but it <u>fails</u> to capture saturation, which is further <u>not</u> caused by Ca^{2+} spike but possibly of Ca^{2+}-activated K-channels that blunt the effect of increased depolarization. Recall that NMDA receptors but not AMPA-receptors pass Ca^{2+}.