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**Report**

# Depressed Responses of Facilitatory Synapses

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[1]*Department of Neurobiology Institute of Life Sciences and Interdisciplinary Center for Neural Computation, the Hebrew University, Givat Ram, Jerusalem, Israel; and* [2]*Institute of Neuroinformatics, University Zurich/Eidgenössische Technische Hochschule, Zurich, Switzerland*

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**Banitt, Yoav, Kevan A. C. Martin, and Idan Segev.** Depressed responses of facilitatory synapses. *J Neurophysiol* 94: 865–870, 2005. First published February 23, 2005; doi:10.1152/jn.00689.2004. We show that when temporal summation takes place, depression of postsynaptic responses may ensue when the underlying synaptic conductance change is constant or even facilitatory. We term this phenomenon "apparent depression." Such apparent depression is most notable for slow synaptic conductance changes, for high frequency, and when the synapse is located at distal dendritic sites. We show that, when temporal summation ensues, the erroneous estimation of short-term synaptic plasticity arises partially from the conventional measurement of synaptic dynamics at postsynaptic potential peak time. This can be corrected when measuring overlapping synaptic responses at fixed intervals after stimulus time. Somatic voltage clamp also helps to partially correct for the apparent depression, but a good model of the neuron can do even better in providing a more accurate view of the underlying synaptic conductances.

## INTRODUCTION

Short-term synaptic plasticity is a property of many central synapses. After a train of action potentials, the amplitude of the postsynaptic response may either depress for successive stimuli, or facilitate. In a depressing synapse, the amplitude of successive responses [either postsynaptic potentials (PSPs) or postsynaptic currents (PSCs)] is smaller than that of the first response; the opposite is true for a facilitatory synapse. Dynamic synapses transmit different aspects of the presynaptic activity, depending on the temporal structure of the input (Markram and Tsodyks 1996; Tsodyks et al. 1998). These synapses may govern the dynamics of the network, and they may have important computational role, such as input-specific gain control (Abbott et al. 1997) and coincidence detection (Senn et al. 1998). It is therefore of critical importance to characterize the activity-dependence dynamics of synapses accurately.

The motivation for this study came from an attempt to model the dynamics of inhibitory synapses (Tarczy-Hornoch et al. 1998) and excitatory synapses (Stratford et al. 1996) formed onto a spiny stellate cell in layer 4 of the cat primary visual cortex. Tarczy-Hornoch et al. (1998) have shown that this inhibitory connection is depressing. However, we found that, in some cases, we had to assume a facilitatory synaptic conductance change to fit the experimentally measured depressing inhibitory PSPs (IPSPs). We explore here, using both analytical and numerical analysis, the conditions whereby depression of postsynaptic responses may arise from non-depressing (and even facilitatory) synaptic conductance change. We use the term "apparent depression" to mean that the PSPs (or PSCs) are indeed depressed even though the actual synaptic conductance change is not. We show that temporal summation of synaptic inputs is the source for the apparent depression of successive postsynaptic responses. Apparent depression is more pronounced for synapses with slow kinetics, for high-input frequencies, and for synapses located at distal dendritic sites.

## METHODS

Simulations were performed using compartmental models, implemented by NEURON software (Hines 1989). In Figs. 1–3, a single passive Resistor-capacitor compartment receiving repetitive synaptic input was used. In Fig. 4, a model of a reconstructed spiny stellate cell from layer 4 of cat V1 was employed (courtesy of J. C. Anderson, INI, Zurich, Switzerland). Synaptic conductance change was simulated using the NEURON built-in synapse model that describes conductance change as a sum of two exponents, governed by a rise time constant, $\tau_{rise}$, and a decay time constant, $\tau_{dec}$. Unless otherwise specified, the amplitude of the $n$th PSP in a train of overlapping responses was measured as the difference between the voltage at its peak (attained at time $t_p$) and the voltage at $t_p$ in a train with only $n - 1$ PSPs ($V_p$–$V_b$ in Fig. 2*A*).

## RESULTS

To explore the mechanisms that turn a facilitatory conductance change into a depressing voltage response, we used a simplified neuron model consisting of one compartment and a modeled synapse receiving repetitive stimuli. The facilitatory synaptic conductance change and corresponding synaptic current are shown in Fig. 1, *A* and *B*, respectively, and the resultant depressing IPSPs are shown in Fig. 1*C*. The corresponding amplitudes of the synaptic conductance, $g_s$, the synaptic current, $i_s$, and the synaptic potential ($V$) are depicted in Fig. 1*D*.

Temporal summation of the membrane potential is accompanied with a decrease in amplitudes of successive PSPs, whereas the amplitudes of the underlying conductance change facilitates. Indeed, Fig. 1 shows that voltage may appear depressing even with a moderate facilitatory synaptic conductance change.

### *Two sources of apparent voltage depression*

Figure 2*A* shows the voltage response to three identical excitatory conductance transients, $g_s(t)$, repeated at 20-ms

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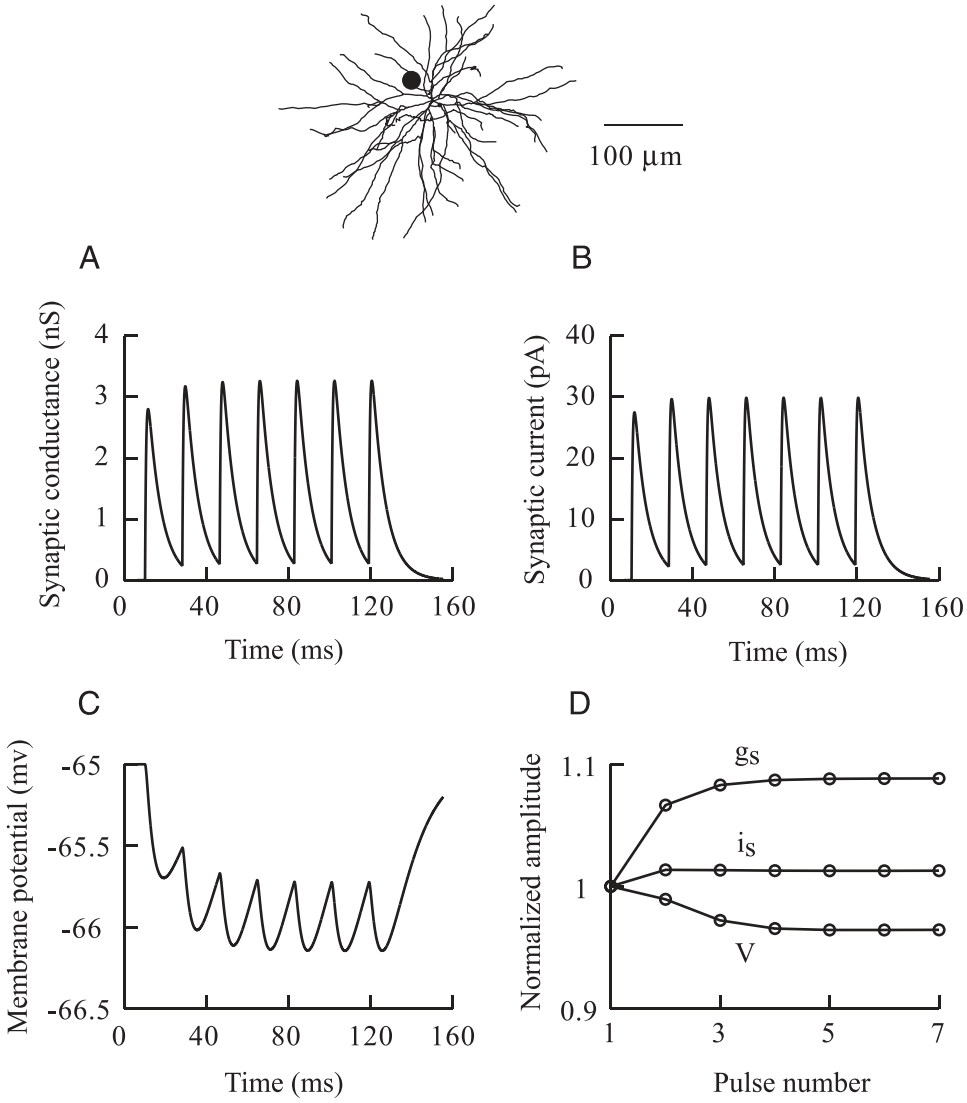


FIG. 1. Facilitatory synaptic conductance turns into depressing inhibitory postsynaptic potentials (IPSPs). *A*: inhibitory synaptic conductance change undergoing facilitation, activated at 55 Hz. Corresponding synaptic current (*B*) and postsynaptic potential (*C*). *D*: amplitudes of synaptic conductance change ($g_s$), synaptic current ($i_s$), and membrane potential ($V$), normalized to the amplitude of the 1st response. A single compartment model was used with membrane parameters, $C_m$ = 1 $\mu$F/cm$^2$, $R_m$ = 12,000 $\Omega \cdot$ cm$^2$, which gives a 79.5 M$\Omega$ input resistance; $V_{rest}$ was set to $-65$ mV. Synaptic conductance change was modeled as a sum of 2 exponents governed by a rise time constant, $\tau_{rise}$ = 0.55 ms, and a decay time constant, $\tau_{dec}$ = 6.5 ms. Maximal conductance of the 1st input was $g_{max}$ = 2.8 nS with synaptic battery, $E_S = -75$ mV. Synaptic facilitation was modeled as in Varela et al. (1997). Facilitation parameters: $\tau_F$ = 13 ms, $F$ = 0.27. When this facilitatory synapse was activated at the dendritic location marked by a circle on the model of reconstructed spiny stellate cell (*inset*), a depressing IPSP similar to that measured by Tarczy-Hornoch et al. (1998) was obtained (data not shown).

intervals (Fig. 2*B*). The vertical lines denote the EPSP peak time. The amplitude of the third EPSP is defined as the sum of $V_p - V_0$ ($a \uparrow$) and of $V_0 - V_b$ ($a \downarrow$); this amplitude, divided by the amplitude of the first EPSP ($amp_1$), is conventionally used to measure short-term synaptic plasticity (Otis et al. 1993). However, careful observation of Fig. 2 shows that, relative to the start of each successive $g_s(t)$, the corresponding EPSP peak is shifted to the left (i.e., appears earlier). This peak-shift effect is simply a consequence of summing the rising phase (positive slope) of a second EPSP with the decaying phase (negative slope) of the first EPSP. This necessarily implies that the peak of the summed response (where the derivative of the *sum* is 0) will appear earlier (shifted to the left) compared with the peak of the second EPSP individually. As we will show below, this peak-shift effect leads to a problem in unveiling the true dynamics of the synaptic process.

Figure 2, *C* and *D*, depicts an alternative new way to characterize the short-term plasticity of a synapse. We define $\Delta t$ as the time-to-peak of the first EPSP and $\Delta V$ is measured at an interval $\Delta t$ after the time of the third stimulus (Fig. 2*C*). In the new measure suggested here, the ratio $\Delta V/amp_1$ is used to characterize short-term synaptic plasticity. In a linear system, for identical current source repeated at constant intervals, this ratio is unity. In contrast, the ratio used as the conventional measure (Fig. 2, *A* and *B*), is smaller than unity in this linear case (apparent depression).

However, even if this problem is avoided, temporal summation may still lead to an apparent depression. In the following, we computed analytically the amplitude of the *n*th PSP in response to a train of step conductance changes in a single compartment model. We note that in the particular case of a step conductance change, there is no peak-shift effect discussed above because the peak of each successive EPSP appears always at the end of the corresponding step conductance change. This enabled us to isolate another source for apparent depression. For a step conductance change, $g_s$, the total current through the membrane compartment is

$$C \times \frac{dV}{dt} + g_s \times (V - E_s) + g_\ell \times (V - E_\ell) = 0 \qquad (1)$$

Where $V$ is the membrane voltage, $C$ is the membrane capacitance, $E_s$ is the synaptic battery, $g_l$ is the membrane leak conductance, and $E_l$ is the reversal potential of the leak current, which for simplicity, was set to zero.

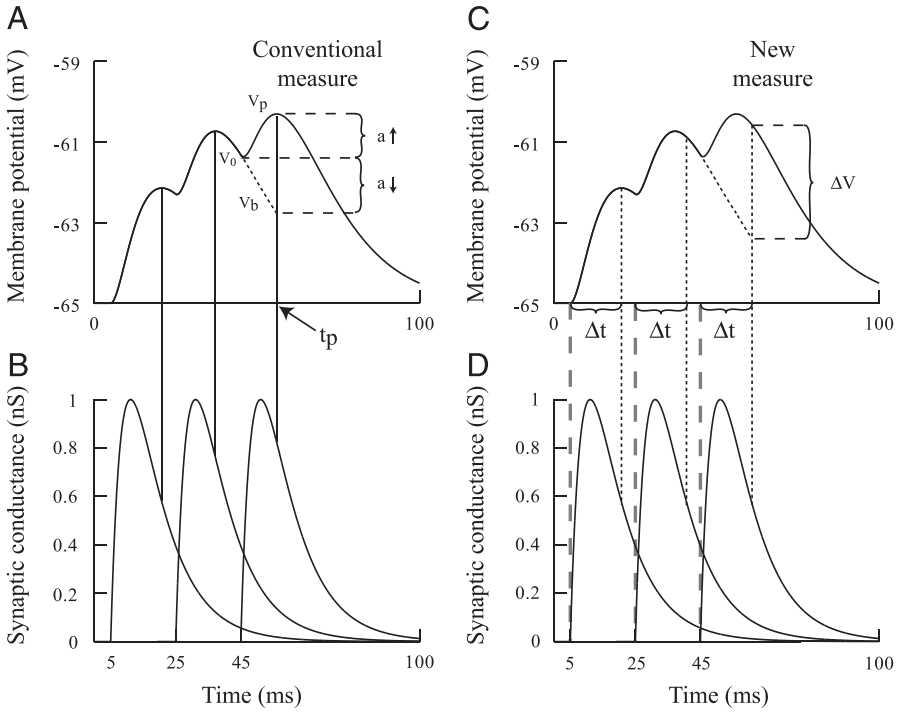


FIG. 2. Measurement of synaptic responses and short-term plasticity. *A*: measurement of PSP amplitude in a train of overlapping responses. Amplitude of the 3rd excitatory PSP (EPSP) is calculated as $V_p - V_b$, where $V_p$ is the voltage at its peak (attained at time $t_p$), and $V_b$ is the voltage at $t_p$ in a train with only 2 PSPs. Voltage at the beginning of the 3rd EPSP is denoted by $V_0$. Amplitude of this EPSP is therefore the sum of $V_p - V_0$ (marked by $a\uparrow$) and of $V_0 - V_b$ (marked by $a\downarrow$). Vertical lines mark time of peaks of EPSPs; this time is conventionally used to characterize short-term synaptic plasticity. *B*: 3 underlying synaptic conductance transients that give rise to the EPSPs in *A*. Vertical lines show that peak times of successive EPSPs in *A* fall at a different time of the underlying synaptic conductance response (peak-shift effect). *C*: same as in *A*. Dashed vertical lines mark beginning of synaptic responses. Dotted vertical lines depict timing of measurements of short-term plasticity (new measure) using a fixed interval ($\Delta t$) from stimulus time. The new definition of amplitude of the 3rd EPSP is now $\Delta V$. *D*: same transients as in *B*. Now dotted vertical lines fall always at the same time of the underlying synaptic conductance response. Synaptic conductance was modeled as a sum of 2 exponents: $\tau_{rise} = 4$ ms and $\tau_{dec} = 10$ ms. Peak synaptic conductance was fixed at $g_{peak} = 1$ nS. Synaptic battery, $E_s = 0$ mV. Single compartment model as in Fig. 1 was used.

Using the same notations as in Fig. 2*A*, for a step conductance change $g_s$

$$a\downarrow = V_0 \times (1 - e^{-t/\tau_r}); \quad a\uparrow = V_{ss} \times (1 - e^{-t/\tau_s}) - V_0 \times (1 - e^{-t/\tau_s}) \quad (2.1)$$

and the amplitude (*amp*) of the EPSP is

$$amp = V_{ss} \times (1 - e^{-t/\tau_s}) - V_0 \times (e^{-t/\tau_r} - e^{-t/\tau_s}) \quad (2.2)$$

where $t$ is the duration of the step conductance change, $\tau_r$ is the passive membrane time constant, $\tau_s$ is the effective time constant when the synapse is activated, and $V_{ss}$ is the steady-state synaptic potential

$$\tau_s = C/(g_\ell + g_s); \quad \tau_r = C/g_\ell; \quad V_{ss} = (g_s \times E_s)/(g_s + g_\ell) \quad (3)$$

$a\uparrow$ is the rising phase of the EPSP during the step conductance change, whereas $a\downarrow$ is a passive decay phase from $V_0$ to $V_b$. When temporal summation ensues and $V_0$ (the membrane potential at the beginning of the pulse) becomes increasingly larger for consecutive pulses, $a\uparrow$ becomes smaller ($-V_0 \times (1 - e^{-t/\tau_s})$ becomes more negative), whereas $a\downarrow$ increases (*Eq. 2.1*). For $a\uparrow$, $V_0$ is multiplied by an exponent that is governed by the effective time constant, $\tau_s$, whereas for $a\downarrow$, $V_0$ is multiplied by an exponent that is governed by a larger time constant, $\tau_r$. Thus when temporal summation ensues ($V_0 > 0$), the decrease in $a\uparrow$ is larger than the increase in $a\downarrow$ for consecutive pulses, leading to progressively smaller PSPs, whereas the underlying synaptic conductance change is of constant amplitude (leading to an apparent depression). Note that the cause for the apparent depression in *Eq. 2.2* is the result of both the change in driving force during temporal summation and the difference in time constants that govern the rising phase and the decaying phase of the PSP (*Eq. 2.2*).

We conclude that when temporal summation of PSPs takes place, there are two sources for the apparent depression. The first concerns with peak-shift effect, whereby synaptic dynamics in overlapping PSPs is measured at peak time, rather than at a fixed time, after stimulus onset. The second source is the change in driving force during temporal summation and the different time constants governing the rising and the decaying phases of the PSP. As a result, the synaptic conductance may be facilitatory, whereas the corresponding PSPs may be depressing.

### *Which key parameters make a nondepressing synapse look depressed?*

In *Eqs. 1–3*, synaptic input was modeled by a step conductance change, which enabled an analytical solution. In Figs. 3 and 4, we simulated the more realistic case in which an excitatory synaptic conductance was modeled by a sum of two exponents, governed by a rise time constant ($\tau_{rise}$) and a decay time constant ($\tau_{dec}$). We examined two key parameters that affect the apparent synaptic depression: the input frequency, $f_{in}$ (Fig. 3*A*), and the duration of the synaptic conductance change (governed by both $\tau_{rise}$ and $\tau_{dec}$, see Fig. 3*B*). Continuous lines depict short-term synaptic plasticity when measurements were made at PSP peak times, as done conventionally (Fig. 2, *A* and *B*). The dotted lines show the alternative new measure at fixed time intervals (Fig. 2, *C* and *D*). For a high-input frequency (100 Hz) and a "moderately slow" synapse ($\tau_{rise} = 1$ ms, $t_{dec} = 10$ ms), EPSP amplitudes at steady state dropped to near 0.65 of the first response (continuous line with triangles), although the amplitude of the conductance change was always fixed at 1 nS (Fig. 3*A*). Using the new measure, apparent depression at steady state is now only 0.95 (dotted line with triangles). Decreasing the input frequency reduced the apparent depression.

The effect of changing the duration of synaptic conductance is shown in Fig. 3*B*. Here we changed only $\tau_{dec}$, but similar results are obtained by changing $\tau_{rise}$. With a slow synapse ($\tau_{dec} = 20$ ms) activated at 50 Hz, apparent depression measured at EPSP peak times falls below 0.8 of control (Fig. 3*B*,

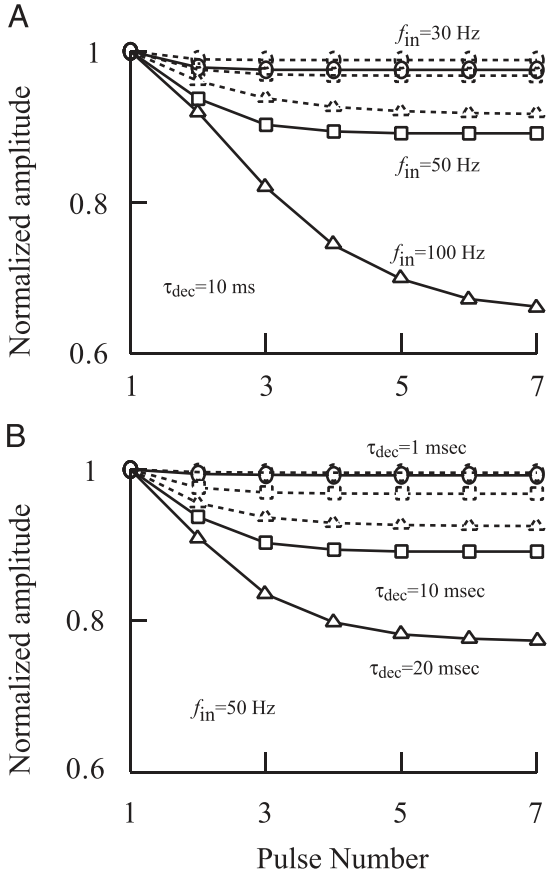


FIG. 3. Effect of duration of synaptic conductance change and input frequency on apparent depression. *A*: EPSP amplitude measured at peak (filled lines) and at constant intervals from stimulus times (dotted lines), normalized to amplitude of 1st EPSP. Input frequencies, $f_{in}$, were 30 (○), 50 (□), and 100 Hz (△). In all cases, $\tau_{rise}$ = 1 ms and $\tau_{dec}$ = 10 ms. *B*: normalized EPSP amplitudes measured at peak (filled lines) and at constant intervals from stimulus times (dotted lines) are plotted for $\tau_{dec}$ values of 1 (○), 10 (□), and 20 ms (△). In all cases, $\tau_{rise}$ = 1 ms and input frequency $f_{in}$ = 50 Hz. Other parameters are as in Fig. 2.

continuous triangles); when the new measure is used, apparent depression is only 0.95 of control (Fig. 3*B*, dotted triangles). The faster the synapse is, the smaller apparent depression is.

### *How dendritic trees affect apparent synaptic depression?*

The cable properties of the dendritic tree are another important factor that determines the degree of temporal summation and therefore may affect the degree of apparent synaptic depression. As a consequence of the cable properties of dendrites, somatic PSPs originated at distal synapses are broadened (Rall 1967). Thus for the same conductance change, the more distal the synapse is, the larger the temporal summation at the soma. Figure 4 shows the effect of the distance of the synapses from the soma on apparent depression at the soma, using a model of a reconstructed spiny stellate cell from cat visual cortex. A modeled synapse with a constant peak conductance was placed at increasing distances from the soma and stimulated at a 100 Hz. In Fig. 4*A*, a fast synapses was simulated, and Fig. 4*B* shows the case of a slow synapse. As in Fig. 3, we examined here the effect of the duration of the synaptic input by changing only the decay time constant ($\tau_{dec}$) of the conductance change; a similar effect is obtained by changing the time constant that governs the rising phase (data not shown).

For fast synapses, apparent depression was negligible for all dendritic locations. Note that there is no temporal summation of the EPSPs at the input site (blue trace in *inset*); however, at the soma, there is a significant temporal summation, although the input is brief (red trace). Indeed, apparent depression is not a necessary consequence of temporal summation.

For slow synapses, however, apparent depression was much more prominent (Fig. 4*B*). For somatic EPSPs originating from the most distal synapse, the EPSP amplitude at steady state was as small as 0.4 of the first peak, despite the fact that the peak synaptic conductance was constant (Fig. 4*B*, continuous green line). Again, this apparent depression can be partially corrected by using the new measure (Fig. 4*B*, dotted green line). Next we repeated the simulation with a somatic voltage clamp and measured the amplitudes of the somatic EPSCs. The synaptic current at the soma still appeared depressed to a level of 0.6 for the distal synapse (Fig. 4*B*, brown continuous line); this is only slightly corrected using the new measure (Fig. 4*B*, brown dotted line). The *inset* shows that, even with clamped soma (red), a strong temporal summation of EPSPs at the synaptic location (blue) occurs, giving rise to apparent depression. This re-emphasizes that the membrane potential at distal sites is almost unaffected by the somatic voltage clamp (Carnevale and Johnston 1982; Clements and Redman 1989; Rall and Segev 1985).

To conclude, temporal summation and apparent depression of synaptic responses are expected to intensify with *1*) increase in input frequency, *2*) increase in duration of synaptic conductance change, and *3*) increase in distance of the synapse from the soma. Somatic voltage clamp partially alleviates the problem of apparent depression, thus enabling the actual dynamics of the synaptic conductance to be more accurately reconstructed.

## DISCUSSION

We have shown that, when repetitive activation of a synapse induces temporal summation, there is an apparent depression of successive responses. Thus a synapse with a fixed or even facilitatory conductance change may appear depressed; a depressing synapse may look even more depressed and a strongly facilitatory synapse may appear only slightly facilitatory. We have shown two causes for this apparent depression: changes in driving force during overlapping synaptic responses and different time constants governing the rising and decaying phases of the PSPs (*Eq. 2.2*) and measurement of short-term synaptic plasticity at PSPs peak times. We show that a consistent characterization of synaptic dynamics in linear systems would be at a fixed time interval after successive stimuli (Fig. 2, *C* and *D*), rather than at peak times. This new measure may correct for a significant degree of the apparent depression.

It is important to note that when characterizing the dynamics of a specific synaptic connection, for example, that of pyramidal cells and interneurons in CA1 of rat hippocampus (Ali et al. 1998; Wierenga and Wadman 2003), analyzing synaptic dynamics using PSPs would give different results than analyzing the corresponding PSCs. This is valid when using both the conventional measure (peak times) and the new measure suggested here. In any case, somatic voltage clamp helps to

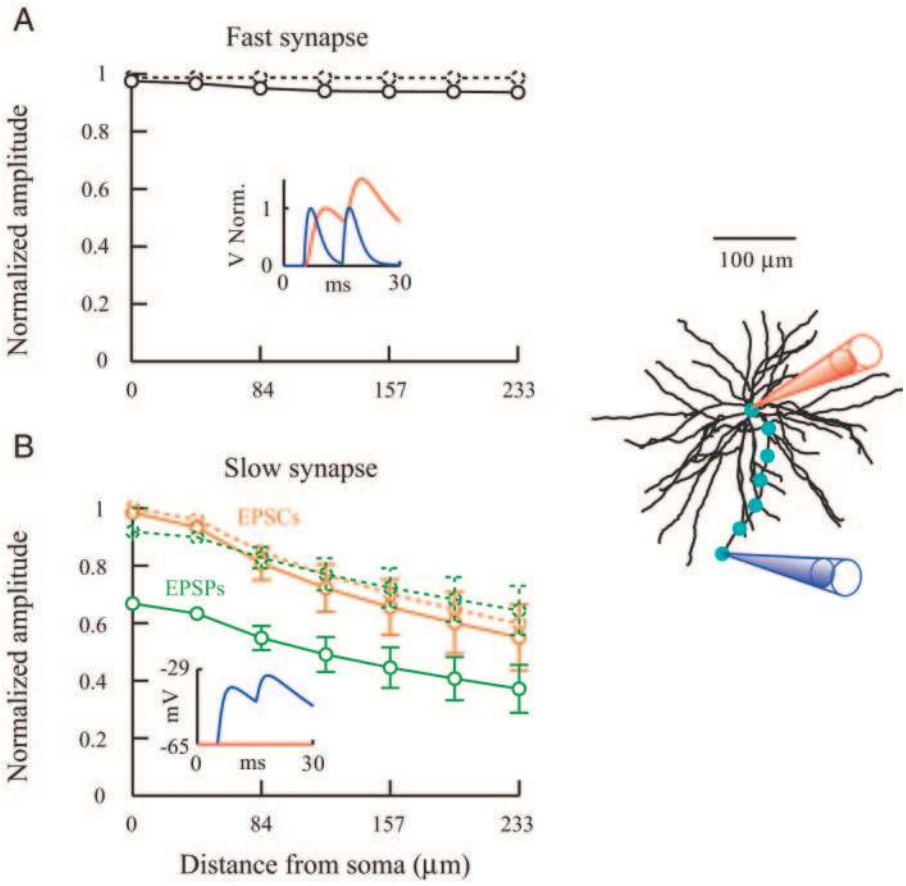


FIG. 4. Effect of the dendritic tree on apparent synaptic depression. A model synapse was placed at increasing distances from the soma and stimulated at 100 Hz. For each distance, peak response at steady state was normalized to peak of 1st response (mean ± SD for 3 different dendritic branches at any given location). Responses were measured at peak times (filled lines) and at constant intervals (dotted lines). Schematic on the *right* depicts locations of the synapse on 1 of the dendritic branches in the modeled dendritic tree. Simulations were performed under current clamp (EPSPs) and, for the slow synapse in *B*, repeated under somatic voltage clamp [excitatory postsynaptic currents (EPSCs)]. Synaptic conductance change was simulated as a sum of 2 exponents. *A*: fast synaptic input ($\tau_{rise}$ = 1 ms, $\tau_{dec}$ = 1 ms). *Inset*: transient responses for synaptic input located at distal dendritic site (blue electrode); blue trace was measured at the synaptic location, whereas red trace was measured at the soma. EPSPs are normalized to amplitude of 1st response. Note the temporal summation of EPSPs at soma (red trace). *B*: slow synaptic input ($\tau_{rise}$ = 1 ms, $\tau_{dec}$ = 10 ms). Green lines are for current-clamp conditions, whereas brown lines are for the case of somatic voltage clamp. *Inset*: transient voltage responses for distal synaptic input under somatic voltage clamp. Note the "escape" of distal EPSPs from voltage clamp and the temporal summation at the synaptic site (blue transients). Membrane parameters same as in Fig. 1 with axial resistivity, $R_i$ = 200 Ω · cm. Maximal synaptic conductance change in *A* and *B*, $g_{max}$ = 1 nS.

partially correct for the apparent depression (Fig. 4*B*), but a good model of the neuron can do even better in providing a more accurate view of the underlying synaptic conductances.

Why is it of functional importance to extract the correct synaptic dynamics? Let us take as an example the work of Tarczy-Hornoch et al. (1998), which was the starting point for this study. This work studied the dynamics of the inhibitory synapses formed by basket cells onto spiny stellate cells in layer 4 of cat V1. They found that the IPSPs of these synapses undergo depression that is proportional to the input frequency. Namely, a stronger steady-state depression is obtained for higher input frequencies. This implies that, at high-input frequencies, the effect of the basket cells inhibition on the spiny stellate cells is reduced. If, however, the underlying synaptic conductance of inhibitory synapses is facilitatory, the shunting effect of synapses formed by the basket cells on the dendritic tree of the postsynaptic spiny stellate cells would increase rather than decrease with the firing frequencies of the presynaptic basket cells. The role of the dynamic synapses in this case would be to augment the inhibitory effect of the presynaptic basket cells, especially at high frequencies.

Underestimation of excitatory synaptic shunts will also lead to an incomplete understanding of many important aspects of the neuron's activity. With massive excitatory input as is the case in vivo, the dendritic tree undergoes a significant shunt (see Pare and Destexhe 1998). Under strong dendritic shunt, the temporal resolution of the dendritic tree is sharpened (due to decrease in the effective membrane time constant), individual EPSPs become smaller, and attenuation along the dendritic tree is augmented (Bernander et al. 1991; Rapp et al. 1992). Furthermore, with a strong dendritic shunt, current threshold and the voltage threshold for spike generation increase. In addition, the back-propagating action potentials in dendrites are expected to be less secure, and a local dendritic spike is less likely to be generated. It is therefore of great importance to characterize accurately the shunt that neurons experience when they are embedded in active networks.

We have initially uncovered the phenomenon of apparent depression during an attempt to model a synapse formed by a basket cell onto a spiny stellate cell in the visual cortex. The model enabled us to predict that this synapse, which appears to depress, is actually facilitating. The process of reconciliation between model response and experiments provides a chance of uncovering the real dynamics of synapses. It still remains to be shown directly whether this specific connection is depressing or facilitatory; measuring changes in input resistance of the spiny stellate cell at different input frequencies at steady state will help in resolving this issue.

## ACKNOWLEDGMENTS

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## REFERENCES

**Abbott LF, Varela JA, Sen K, and Nelson SB.** Synaptic depression and cortical gain control. *Science* 275: 220–224, 1997.

**Agmon-Snir H and Segev I.** Signal delay and input synchronization in passive dendritic structures. *J Neurophysiol* 70: 2066–2085, 1993.

**Ali AB, Deuchars J, Pawelzik H, and Thomson AM.** CA1 pyramidal to basket and bistratified cell EPSP's: dual intracellular recordings in rat hippocampal slices. *J Physiol* 507: 201–217, 1998.

**Bernander O, Douglas RJ, Martin KA, and Koch C.** Synaptic background activity influences spatiotemporal integration in single pyramidal cells. *Proc Natl Acad Sci USA* 88: 11569–11573, 1991.

**Carnevale NT and Johnston D.** Electrophysiological characterization of remote chemical synapses. *J Neurophysiol* 47: 606–621, 1982.

**Clements JD and Redman SJ.** Cable properties of cat spinal motoneurones measured by combining voltage clamp, current clamp and intracellular staining. *J Physiol* 409: 63–87, 1989.

**Dean AF.** The relationship between response amplitude and contrast for cat striate cortical neurones. *J Physiol* 318: 413–427, 1981.

**Hines M.** A program for simulation of nerve equations with branching geometries. *Int J Biomed Comput* 24: 55–68, 1989.

**Maffei L, Fiorentini A, and Bisti S.** Neural correlate of perceptual adaptation to gratings. *Science* 182: 1036–1038, 1973.

**Markram H and Tsodyks M.** Redistribution of synaptic efficacy between neocortical pyramidal neurons. *Nature* 382: 807–810, 1996.

**Otis TS, De Koninck Y, and Mody I.** Characterization of synaptically elicited GABAB responses using patch-clamp recordings in rat hippocampal slices. *J Physiol* 463: 391–407, 1993.

**Pare D, Shink E, Gaudreau H, Destexhe A, and Lang EJ.** Impact of spontaneous synaptic activity on the resting properties of cat neocortical pyramidal neurons in vivo. *J Neurophysiol* 79: 1450–1460, 1998.

**Rall W.** Distinguishing theoretical synaptic potentials computed for different soma-dendritic distributions of synaptic input. *J Neurophysiol* 30: 1138–1168, 1967.

**Rall W and Segev I.** Space-clamp problems when voltage clamping branched neurons with intracellular microelectrodes. In: *Voltage and Patch-Clamping With Microelectrodes*, edited by Smith TG Jr, Lecar H, and Redman SJ. Bethesda, MD: American Physiological Society, 1985, p. 191–215.

**Rapp M, Yarom Y, and Segev I.** The impact of parallel fiber background activity on the cable properties of cerebellar purkinje cells. *Neural Comput* 4: 518–533, 1992.

**Senn W, Segev I, and Tsodyks M.** Reading neuronal synchrony with depressing synapses. *Neural Comput* 10: 815–819, 1998.

**Stratford KJ, Tarczy-Hornoch K, Martin KA, Bannister NJ, and Jack JJ.** Excitatory synaptic inputs to spiny stellate cells in cat visual cortex. *Nature* 382: 258–261, 1996.

**Tarczy-Hornoch K, Martin KA, Jack JJ, and Stratford KJ.** Synaptic interactions between smooth and spiny neurones in layer 4 of cat visual cortex in vitro. *J Physiol* 508: 351–363, 1998.

**Tsodyks M, Pawelzik K, and Markram H.** Neural networks with dynamic synapses. *Neural Comput* 10: 821–835, 1998.

**Varela JA, Sen K, Gibson J, Fost J, Abbott LF, and Nelson SB.** A quantitative description of short-term plasticity at excitatory synapses in layer 2/3 of rat primary visual cortex. *J Neurosci* 17: 7926–7940, 1997.

**Wierenga CJ and Wadman WJ.** Excitatory inputs to CA1 interneurons show selective synaptic dynamics. *J Neurophysiol* 90: 811–821, 2003.