Supplementary Method

The extent of NMDARs activation (Supplementary Fig. 1) during spiking or non-spiking depolarizations of off-response of tectal neurons (from the same cell shown in Fig. 1b) was simulated using the asymmetric trapping block model (ref. 16). The pattern of presynaptic glutamate release was approximated by using experimentally recorded spike trains from retinal ganglion cells in response to light stimuli (ref. 17). We assumed that each spike-triggered glutamate release leads to an increase in the glutamate concentration from basal level of 25 µM to 1 mM for 1-ms duration (Supplementary Fig. 1a). The model used for simulating NMDAR currents consisted of 20 NMDARs (single channel conductance is 50 pS, with extracellular 1.5 mM Mg\(^{2+}\)) on the postsynaptic neuron (without considering the neuronal morphology). The simulation results showed a much larger extent of NMDAR activation by spiking waveforms than that by non-spiking ones. The change in the total charge relative to the baseline was quantified by integrating the NMDA-mediated currents from 360 to 1200 ms (see Supplementary Fig. 1c,d). In summary, the total charge estimated was at least 5-fold larger in spiking forms than that in non-spiking forms (Supplementary Fig. 1d).