



Large, invariable action potential amplitude in neocortical nerve terminals revealed by high-resolution current-clamp recordings

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The presynaptic action potential critically controls synaptic transmission. At experimentally-accessible large nerve terminals, sodium and potassium channels control the amplitude and duration of action potentials, respectively. Recently, potassium channels were found to dynamically regulate action potential amplitude in small nerve terminals and this effect was proposed as a novel mechanism for synaptic plasticity. To investigate this apparent difference between large and small terminals, we established high-resolution current-clamp recordings from small nerve terminals of cultured neocortical neurons. Using an electrical equivalent circuit and quartz glass pipettes, we systematically investigated errors related to pipette capacitance and the performance of current-clamp amplifiers. We found rapid action potentials with an amplitude of ~ 120 mV. Blocking potassium channels prolonged action potentials but did not affect their amplitude. Furthermore, the spike amplitude was unaffected during short-term and homeostatic plasticity. Thus, our data indicate large and stable action potential amplitude at small neocortical nerve terminals.

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Clinic for Epileptology, Seminar Room, Ground Floor



If you would like to meet with the speaker, please contact:

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