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Local and cortical contributions to the field potentials recorded in the somatosensory thalamic nuclei

Local field potentials (LFP) recording is a tool well suited for the monitoring of neuronal activity. It is generally believed that LFP changes are mainly caused by synaptic and corresponding return currents. However, due to volume conduction, changes in electric field propagate widely within the brain tissue and thus, signal recorded in one place may have a substantial contribution from distant neuronal populations. There is no consensus among researchers on the magnitude of this passive spread. Some reports claim that recorded potential is almost exclusively sum of signals originating in a radius about 250 μm from an electrode while others consider larger distances up to several millimeters.

Determining this effect is crucial in the model we use in our research: the rat vibrissae-barrel system. The cortical representation of mystacial vibrissae is located relatively close above somatosensory thalamic nuclei. Moreover, due to geometry and organization of cells, thalamic population activated by sensory input produce weaker and more closed potential field than cortical population. In our previous experiments we showed in a rat that the early (~ 10 ms post-stimulus, N_{12}) thalamic wave coincides with the cortical N_1 wave and the amplitude of the thalamic N_{12} was modulated during cortical cooling. We interpreted this as a result of active influence of cortical feedback connection over thalamic activity. However, in the light of our later results and those from other groups such similarity of thalamic and cortical recording, to some extent, must be a result of passive spread of the strong cortical signal to the thalamus. To examine this possibility, we recorded evoked potentials (EPs) from multiple locations through the barrel cortex and ventrobasal thalamus in urethane-anesthetized rats. Then we applied kernel Current Source Density method (kCSD) to estimate the distribution of current sinks and sources along the shaft of the electrode. Statistical significance for CSD maps were estimated using percentile bootstrap method. Next, we used the calculated CSD distribution in forward modeling to compute the contribution to the measured potential coming from particular structures. To assess the extent to which the potentials recorded in the thalamus can be explained by local and by distant sources we compared the shapes of averaged recorded thalamic EPs and their model counterparts. As a simple measure of similarity we used Pearson's linear correlation coefficients which were calculated for 22 ms waveform sweeps starting 3 ms to 25 ms after the stimulus.

We found clusters of statistically significant sinks and sources in the thalamus within analyzed 25 ms post-stimulus time window. Cortical sinks and sources were order of magnitude stronger. Model EPs calculated from thalamic CSD was highly similar to raw EPs recorded in the thalamus which indicates that local currents determine time course of EPs recorded in the thalamus. Contribution from cortical currents to potentials recorded in the thalamus came out noticeable but not dominating. This confirmed that the recording of local field potentials can be used as a valid representation of neuronal thalamic activity.