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Monitoring cortical neuronal activity and spreading depression in freely behaving familial hemiplegic migraine Cacna1a R192Q knockin mice

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Introduction

Experimental findings from transgenic migraine mouse models that carry a human FHM1 gain-of-function mutation in CaV2.1 (P/Q-type) calcium channels underscore the role of neuronal hyperexcitability in migraine [1,2]. However, functional data that link the excitability changes to neuronal network activity and the enhanced propensity to cortical spreading depression (CSD), the likely mechanism underlying migraine aura, are largely lacking.

Purpose/background/objectives

Here, we aimed to set up an electrophysiology platform to study changes in cortical neuronal network activity in relation to CSD in freely behaving transgenic migraine mice.

Methods

We developed an electrophysiology system for long-term recordings of DC-EEG and multi-unit-activity from the cortex of freely behaving mice. The system combines a counterbalanced 7 channel swivel with custom-built differential DC-EEG, AC-EEG and unit activity amplifiers. Stable DC-EEG recordings are obtained using AgAgCl epidural electrodes, while intracortical platinum electrodes are used for simultaneous recording of multi-unit-activity and AC-EEG. For CSD induction intracortical microdialysis was used for infusion of high KCl solution.

Results

Simultaneous recordings of multi-unit activity, DC- and AC-EEG were made from the sensorimotor and occipital cortex of wild-type and FHM1 migraine mice for up to 3 weeks. Apart from spontaneous cortical activity, visual evoked cortical responses were induced using 1 ms blue light pulses. Microdialysis with KCl solution resulted in successful induction of CSD events in the awake mice.

Conclusion

We established a novel platform for performing longitudinal recordings of cortical neuronal activity and spreading depression in freely behaving mice carrying migraine-specific mutations. Using this platform, we aim to characterize how cortical activity is altered by modulatory factors that predispose for migraine attacks.

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