

A neurovascular decoupling during ictal activity in rats with focal epilepsy.

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Abstract Details

The spatiotemporal profiles of neuronal activity, hemodynamics and metabolic rate in epileptic cortices during focal ictal activity were recently evaluated using modern imaging techniques in acute situations (4-aminopyridine). However, no studies have verified these profiles in chronic models of focal epilepsy. In this study, we combined electrophysiology and optical techniques to untangle the relationships between neuronal activities and hemodynamic responses during ictal periods in the cortices of rats with chronic focal epilepsy.

13 Wistar rats with focal epilepsy were used in this study. Candidate regions for a craniotomy were obtained from EEG source imaging and BOLD effect associated with interictal activity. Intracranial electrical recordings were obtained by inserting a 16-channel probe into the irritative cortex. CBF from the irritative cortex was recorded by using a laser Doppler flowmetry system. Dual-wavelength intrinsic optical imaging system was later employed to obtain spatiotemporal variations of CBV and local concentration of de-oxy hemoglobin [dHb]. Seizure-related MUA responses and spectral perturbations in different LFP frequency bands were obtained.

Ictal activities were frequently observed in several rats. The averaging event-related response of CBF locked to the ictal periods showed a significant decrease preceding the ictal onset, and gradually increased until the end of the ictal period. All LFP frequency bands, as well as the MUA, contributed to the CBF dynamics Fig. 1B. We also observed spatiotemporal fluctuations of CBV and [dHb] before and after the ictal onset. These fluctuations explain the existence of activation and deactivation BOLD responses.

Our study suggests that the deactivation BOLD signal previously reported during ictal periods is caused by a decrease in CBF before the seizure-onset. We also observed increases in [dHb] at the end of the ictal period probably reflecting an increase in oxygen metabolism associated with an early "epileptic dip".

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