Cortical oscillations as seizure markers in photosensitive epilepsy

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Introduction

Photosensitive epilepsy (PSE) is the most common reflex epilepsy, where seizures are triggered by a visual stimulus denoted by a 'photoparoxysmal response' (PPR) on an electroencephalogram (EEG).[1] PSE provides a reproducible model to investigate the changes in neuronal oscillatory networks leading to interictal to ictal transition,[2] and understand the pathophysiology of epilepsy overall.[3,4] The aim of this study is to investigate the interictal to ictal transition in PSE using intermittent photic stimulation (IPS) by analysing cortical oscillations on scalp EEG.

Methods

Clinical EEG data with IPS, a common clinical procedure used for investigation of epilepsy, were collected for patients with genetic generalised epilepsies (GGE), and two groups were established: one with photosensitivity and another without. These data were signal processed in Matlab, epochs of IPS were extracted, PPRs were identified, and segments of EEG signal preceding the PPR were extracted from the photosensitive group. Corresponding segments from the non-photosensitive group were extracted. The various frequency components of the signal underwent a Herbert transform to produce time–frequency spectra, and average power at the stimulation frequency band and its harmonics was calculated for the frontal and occipital EEG channels. Then, the phase clustering index (PCI) was calculated to compare the phase synchrony of neurons in the photosensitive group and non-photosensitive group.

Results and discussion

The following patient groups were included:

- GGE with photosensitivity, 28 patients (mean±SD age 18±12 years)
- juvenile myoclonic epilepsy (a type of GGE), 26 patients (mean±SD age 22±8 years). The average power at the stimulation frequency was increased for both the occipital and frontal channels in the photosensitive group vs non-photosensitive, indicating that there is a greater degree of entrainment or locking of neuronal oscillation to the visual stimulus, albeit not statistically significant (frontal: p=0.1187, occipital: p=0.0595, unpaired t-test adjusted for multiple comparisons). There was also significantly increased phase clustering for frontal and occipital channels (channel F4: p=0.0195, F7: p=0.0174, O2: p=0.0174, unpaired t-test adjusted for multiple comparisons).

Conclusions

The enhanced phase synchronisation and degree of entrainment of neuronal oscillations can be used as potential biomarkers of epileptic transition in PSE. These results help understand mechanisms of underlying perceptual processes involved in the pathophysiological changes that occur in brain networks in epilepsy, to improve diagnosis and develop effective treatment in the future. ■

References

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