Modification of brain activity following the introduction of antiseizure medications: an EEG-microstates study

C. Catania, S. Gallotto, E. Ménétré, M. Seeck
EEG and epilepsy unit, Neurology division, University Hospital of Geneva

cecilia.catania@unige.ch

Introduction
Evidence of the impact of ASM introduction on brain electrical activity and its correlation with therapeutic outcomes is limited [1]. The support of visual EEG interpretation is scarce and more advanced methods should be considered. EEG microstates represent brief, quasi-stable scalp potential topographies, reflecting the alternating dynamics within resting-state networks [2]. We investigated the impact of ASM introduction on EEG microstates and evaluated the potential of first EEG recording in predicting seizure recurrence compared to follow-up EEGs.

Methods
We included retrospectively 42 patients with newly diagnosed epilepsy (30 focal structural, 5 IGE, 7 focal of unknown etiology) who underwent a first routine EEG before ASM prescription. All patients performed a follow-up EEG after an average of 96 days from ASM introduction. We identified 11 patients who had seizure relapse in the 6 months following treatment initiation. EEG-microstate parameters (A-D) were compared between the initial and follow-up EEGs, and between relapsers and non-relapsers.

Results
In the overall analysis, a significant decrease in duration (p < 0.001) and increase in occurrence (p = 0.013) of all microstate maps (A-D) was identified after ASM introduction compared to baseline EEG.

We found significant differences in the first drug-naive EEG between relapsers and non-relapsers (p = 0.04). Post-hoc decomposition showed that relapsers had significantly higher global explained variances (GEV) for Map A (p = 0.03) and higher GEV for Map D (p = 0.07) compared to non-relapsing patients.

No differences were found in follow-up EEG between relapsers and non-relapsers.

Discussion
Our study shows that ASM introduction affects neurophysiological activity of the brain and induces major modifications in EEG microstate dynamics. Regarding the identification of future relapers, map A is the most promising candidate. Differences between relapsers and non relapsers were significant only during the first EEG. Thus, our results suggest that the initial, drug-naive EEG recording holds greater potential for predicting seizure recurrence compared to follow-up EEGs, where ASM influence brain activity. Further studies should determine map A usefulness as clinical marker for relapse. If confirmed, patients with increased GEV in map A require closer monitoring.

References