over placebo regardless of previous carbamazepine (CBZ), lamotrigine (LTG), levetiracetam (LEV), or topiramate (TPM) failure. This analysis explored long-term retention of adjunctive BRV in patients with previous CBZ/LTG/LEV/TPM. **Methods:** Post-hoc analysis of double-blind, placebo-controlled trial (N01358 [NCT01261325]) and open-label extension (N01379 [NCT01339559]; cut-off 15-March-2017) of adjunctive BRV in patients (≥16 years) with focal seizures. Outcomes were assessed in patients randomized to BRV (100 or 200 mg/day) who had previous CBZ/LTG/LEV/TPM (stopped ≥90 days before BRV initiation). **Results:** 503 patients were analyzed. Baseline characteristics were generally similar in subgroups with previous CBZ/LTG/LEV/TPM (n=209/162/256/182). Overall, Kaplan-Meier-estimated BRV retention at 1-, 3-, and 5-years was 71.0%, 50.9%, and 32.4%. Across previous antiepileptic drug (AED) subgroups, Kaplan-Meier-estimated BRV retention (1-year: 64.8%–73.2%; 3-year: 41.9%–49.9%; 5-year: 31.5%–35.7%), BRV discontinuations (58.4%–63.0%), and most common reasons for discontinuation (lack of efficacy: 23.0%–25.3%; adverse event: 16.7%–22.2%) were generally similar. **Conclusions:** Post-hoc analysis demonstrated similar long-term retention rates and discontinuation reasons with adjunctive BRV in adults previously treated with CBZ/LTG/LEV/TPM. Adjunctive BRV provides long-term effectiveness in patients who failed common AED treatments, including LEV.

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P.006

Efficacy of adjunctive brivaracetam in adult patients with secondarily generalized tonic-clonic seizures at baseline: pooled results from long-term follow-up trials

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Background: Previous post-hoc analysis of three 12-week, double-blind, placebo-controlled trials showed adjunctive brivaracetam (BRV) reduced focal and secondarily generalized tonic-clonic seizures (SGTCS; Type IC) in patients with baseline SGTCS. This analysis explored long-term efficacy of adjunctive BRV in these patients. **Methods:** Patients (≥16 years) with focal seizures with SGTCS at Baseline were identified from 12-week double-blind, placebo-controlled trials (NCT00490035/NCT00464269/NCT01261325) and subsequent open-label, long-term follow-up (LTFU) trials (NCT00175916/NCT00150800/NCT01339559). Outcomes were assessed at protocol-specified time-points (up to 60 months). We report post-hoc efficacy data for patients receiving BRV (50–200 mg/day). **Results:** At double-blind Baseline, 409 patients had SGTCS (mean epilepsy duration: 22.2 years); 28.4%, 38.9%, and 32.8% had 0–1, 2–4, and ≥5 previous AEDs. Baseline median seizure frequency/28 days was 8.1 (focal) and 3.0 (SGTCS only). 325/409 patients (79.5%) entered LTFU. In the 12-month (n=150), 24-month (n=89), 36-month (n=73), 48-month (n=68) and 60-month (n=57) exposure cohorts, median percent reduction from Baseline in SGTCS frequency/28 days was 81.1%, 84.0%, 89.2%, 91.0%, and 90.6%, respectively. ≥50% responder rates for SGTCS were 75.3%, 78.7%, 80.8%, 79.4%, and 78.9%. No safety concerns were identified. **Conclusions:** Adjunctive BRV (50–200 mg/day) reduced SGTCS frequency during LTFU (up to 60 months) in patients with SGTCS at Baseline.

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P.007

Déjà vu evoked by stimulating the insula in two patients suffering from intractable temporal lobe epilepsy

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Background: Déjà vu is a common manifestation of temporal lobe seizures. It can be reproduced by electrical stimulation of the hippocampus, amygdala and temporal neocortex with stereotactically implanted electrodes. We report here déjà vu triggered by the stimulation of the insula. **Methods:** Two patients suffering from intractable temporal lobe epilepsy exhibiting auras of déjà vu underwent invasive EEG studies. One patient had a prior temporal lobectomy with reoccurrence of similar symptoms after surgery. We performed functional connectivity analyses using phase locking value (PLV) to estimate changes in connectivity between different brain regions in the standard EEG frequency bands during stimulation. **Results:** Stimulation of the insular cortex induced reproducible déjà vu symptoms in both patients. In one patient, PLV analysis showed increased synchronization in the alpha band between insular and temporal regions after an evoked déjà vu compared to a control stimulation. **Conclusions:** Our results suggest that the insula may in rare occasions generate déjà vu. This implies that insular epileptogenicity cannot be ruled out in patients exhibiting such an aura nor in patients with persisting déjà vu despite an initial amygdalo-hippocampectomy.

P.008

Triphasic waves in powassan encephalitis: a case report

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Background: Powassan virus is a tick borne virus which can lead to encephalitis. **Methods:** 63 year old woman with history of migraine presented with 2 days of fever, headache, language difficulty and right sided facial droop. Her examination revealed right upper motor neuron type facial weakness and expressive aphasia. She rapidly deteriorated within 24 hours becoming non verbal and ultimately comatose. **Results:** MRI brain revealed T2 hyperintensities in bilateral caudate and putamen. Subsequent MRI brain showed progression of the caudate and basal ganglia changes and new T2 hyperintensities in bilateral thalami and midbrain with no abnormal enhancement. CSF revealed lymphocytic pleocytosis with normal protein and glucose. Viral Encephalitis was suspected and she was continued on Acyclovir until Varicella zoster and Herpes simplex virus serology in CSF returned negative. Prolonged video EEG showed near continuous generalized triphasic pattern without any evolution or seizure pattern. There was no improvement in clinical status or EEG with antiepileptic treatment. Paraneoplastic panel , serum HIV, Lyme and 14-3-3 protein were negative. Extensive viral serologies were sent and ultimately Powassan serology came back positive. **Conclusions:** This case highlights powassan virus as a cause of encephalitis and occurrence of triphasic waves in non metabolic causes of encephalopathy such as infectious encephalitis.

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