at the right medial frontal convexity covering the suspected lesion and in several ipsilateral interhemispheric contacts.

Typical seizures were induced by stimulation of right (but not left) interhemispheric contacts.

Functional mapping localised the epileptogenic lesion anterior to right medial premotor cortex (Fig.3). Congruent results of intracranial monitoring and MRI postprocessing allowed a lesionectomy. The lesion was histologically classified as FCD Palmini type IIb. The patient is currently seizure-free (follow-up 6 months).

Conclusion: Combining MRI postprocessing with clinical and neurophysiologic data allowed establishing a hypothesis for resective epilepsy surgery in a MRI-negative patient with gelastic seizures due to a right fronto-medial FCD. The results of EEG recordings and mapping demonstrate the role of the medial frontal cortex in the motor but not the affective component of laughter.

Poster session 2
Clinical epileptology

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BESA EPILEPSY: A NEW CLINICAL TOOL FOR THE FAST EVALUATION OF INTERICTAL SPIKES IN LONG-TERM EEG

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Introduction: Traditional spike detection programs mark detected events in long-term EEG but lack a good overview. BESA epilepsy uses a new hypercluster technique and a file independent data management to combine similar events over 24 h of EEG. On a daily basis, the physician inspects each hypercluster and decides whether it is epileptiform or not. Optimized source waveform and EEG segment displays, 3D maps, and localization of the events in a head scheme allow for fast decision and assessment of the likely region of origin.

Methods: A new spike detection and clustering algorithm based on an EEG transformation into 29 regional brain sources was developed. Clusters were calculated in 2 h epochs and combined into daily hyperclusters using empirical rules on similarity in waveshape and topography. 24 h EEG data of 44 epilepsy patients (21 children) were evaluated by independent raters using traditional visual inspection versus fast hypercluster evaluation.

Results: Visual rating resulted in 107 epileptiform spike types. Hypercluster rating agreed in 85% (temporal lobe spikes 94%, extratemporal 78%). Agreement was similar in adults and children. In a 24 h recording, about 15–25 hyperclusters had to be inspected to decide whether they reflected artifacts, normal EEG patterns, or epileptiform discharges. As a benefit of the rapid inspection tools, the decision and reporting process was typically completed within 5 min by an experienced physician.

Conclusion: The traditional hourly evaluation of 2–5 min epochs of long-term EEG can be readily supplemented by a computer-based hypercluster evaluation. The latter adds a fast, comprehensive overview and report, an independent control of the existence of one or multiple spike foci, and an estimation of their origin. The involvement of the physician in the decision process allows to increase sensitivity on the one hand. On the other hand, more hyperclusters with artifacts are formed. Yet, they can be easily rejected by the fast decision process. Long-term EEG evaluation is facilitated by a file independent graphical data management. This enables the rapid clinical inspection of the same, fixed 24 h interval every day without the time-consuming need to open and close different EEG files.

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INTERMITTENT EXPLOSIVE DISORDER: FRONTAL LOBE EPILEPSY?

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Introduction: Intermittent explosive disorder (IED, episodic dyscontrol) is characterized by episodes of failure to resist aggressive impulses that result in serious destructive acts, out of proportion to precipitating stressors, not better explained by another mental conditions.

Case report: An 18-year-old man brought up in a home came to our attention for chronic aggressive behabior due to a difficult familiar setting. He had aggressive outbursts easily explained by situative contexts, but others without evident reasons, as observed by himself or others. Neuroleptic treatment failed to significantly reduce the spontaneous outbursts, while hospitalization reduced environmental triggered ones. Neurological and physical history did not contribute. Neurological exam showed soft signs suggestive of cerebral disintegration and intermittent facial cloni without any semiological resemblence to epileptic myocloni. Clinically, there were signs of emotional and cognitive dysexecutive syndrome. Technical workup included normal routine MRI, EEG with left frontal theta dysrhythmia. Holter-EEG failed to documentate an outburst of aggression, but displayed left frontal thetas of high voltage for up to 12 s. SPECT disclosed interictal left frontal and frontomesial relative hyperperfusion. A therapeutic trial of carbamazepine (CBZ) led to marked improvement of the spontaneous attacks, but the patients mental state remained otherwise unchanged.

Discussion: The clinical picture seemed at first glance compatible with maladaptive behavior but extended to IED. The signs of dysexecutive syndrome and lateralized frontal brain dysfunction indicates epileptic genesis of spontaneous aggressive outbursts, and responded well to CBZ treatment. Whether this represents frontomesial or frontoorbital seizures or subclinical seizures weakening executive functions remains open.

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UNCOMMON COURSE OF NONCONVULSIVE STATUS EPILEPTICUS IN CDKL5-MUTATION-RELATED ENCEPHALOPATHY

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Objective: Severe epileptic encephalopathies are well described in patients with CDKL5-mutation. A slow onset of nonconvulsive status epilepticus as seen in our 36 year old patient is uncommon. The i.v.-administration of Levetiracetame was able to improve the clinical course but not the findings in eeg.

Methods: We report a girl with atypical Rett's syndrome with a primary severe retardation of psychomotor development and pharmacoresistance (valproate, clobazam, pyridoxine, sulthiame, topiramate, ACTH, phenobarbital, vigabatrin und levetiracetam).

In 2009 a point mutation of the CDKL5-gene (c.1751C>G) (p.S584X) was found. Repeated MRI scans revealed a delayed myelination and a progressive global atrophy. Starting with BNS-seizures at the age of 2 weeks the further course implied absences, complex partial seizures and polytopical myoclonic seizures. Since summer 2009 multiple daily generalized tonic seizures were prominent. The child was responsive in the interictal time. Still having a high seizure frequency while treating with lamotrigine and valproate we started an additional dexamethasone pulse therapy in December 2009. The eeg findings still consist of a generalized functional disorder and multifocal epileptic discharges. Comparing to the age of 2 years the EEG findings deteriorated continuously without evidence of a bioelectrical status.