Acta Neurol Scand 2004: 109: 324–329 DOI: 10.1046/j.1600-0404.2003.00234.x
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ACTA NEUROLOGICA
SCANDINAVICA

Objective assessment of neurotoxicity while shifting from carbamazepine to oxcarbazepine

Clemens B, Ménes A, Nagy Z. Objective assessment of neurotoxicity while shifting from carbamazepine to oxcarbazepine.
Acta Neurol Scand 2004: 109: 324–329. © Blackwell Munksgaard 2003.

Objectives – Objective assessment of non-overt neurotoxicity of carbamazepine (CBZ) vs oxcarbazepine (OXC) in patients with difficult-to-treat partial epilepsy, who were resistant to CBZ treatment and were converted from CBZ monotherapy to OXC monotherapy. *Material and methods* – Therapeutically equivalent doses (150 mg OXC for every 100 mg CBZ) were compared in 20 adult patients. Neurological investigation, conventional and spectral EEG analysis, brainstem auditory evoked responses (BAER) were carried out in both treatment conditions. EEG and BAER data of 20 age-matched healthy controls helped interpretation. Primary target variables (electrophysiological parameters) were evaluated blindly. *Results* – There were no significant differences between treatment conditions concerning the neurological condition, lack of clinically evident neurotoxicity, seizure frequency and EEG spike frequency. OXC treatment was characterized by less delta, theta, and alpha power, more beta power, and significantly greater mean alpha frequency ($P = 0.03$ and 0.05 for the left and right occipital leads, respectively), than CBZ treatment. Interpeak latencies were prolonged in the CBZ condition as compared with normals ($P = 0.01$) and OXC ($P = 0.02$). *Conclusion* – In this cohort of patients substitution of OXC for CBZ was associated with significant normalization of electrophysiological parameters, indicating decreasing neurotoxicity while shifting from CBZ to OXC monotherapy.

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Key words: carbamazepine; oxcarbazepine; neurotoxicity

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Accepted for publication August 29, 2003

Seizure (2004) 13, 366–374

SEIZURE

www.elsevier.com/locate/yseiz

Abnormal quantitative EEG scores identify patients with complicated idiopathic generalised epilepsy

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KEYWORDS
Idiopathic generalised epilepsy;
Quantitative EEG;
Personality disorder;
Pharmacoresistance

Summary *Objective*: To investigate the relationship between quantitative EEG (QEEG) scores and ''complicating factors'' (psychopathology, true pharmacoresistance, neurological symptoms) in idiopathic generalised epilepsy (IGE). *Methods*: 35 newly referred, newly diagnosed, unmedicated IGE patients were collected in a prospective and random manner. Standard neuro-psychiatric and EEG examination was done. The patients were treated and controlled at regular visits. After 2 years of follow-up, clinical data were summarised and were compared to QEEG results. Clinical target items were neurologic and psychiatric abnormalities, proven pharmacoresistance. Patients with at least one of these items were labelled ''complicated'', whereas patients without these additional handicap were labelled as ''uncomplicated''. The 12 QEEG target variables were: Z-transformed absolute power values for three (anterior, central, posterior) brain regions and four frequency bands (1.5–3.5; 3.5–7.5; 7.5–12.5; 12.5–25.0 Hz). QEEG scores outside the ±2.5 Z range were accepted as abnormal. The overall QEEG result was classified as normal (0–2 abnormal scores), or pathological (3 or more abnormal scores). Clinical and QEEG results were correlated. *Results*: All patients with psychopathology showed 4–8 positive pathological scores (power excess not confined to a single cortical region or frequency band). The two patients with pure pharmacoresistance showed pathological negative values (delta power deficit) all over the scalp. Statistically significant ($P < 0.001$) association was found between patients with uncomplicated IGE and normal QEEG, and between complicated IGE and pathological QEEG. Patients with neurological items had normal QEEG. *Conclusion*: Higher degree of cortical dysfunction (as assessed in the clinical setting) is reflected by higher degree of QEEG abnormalities. QEEG analysis can differentiate between IGE patients with or without psychopathology. Forecasting psychopathology may be the practical application of the findings.

Seizure (2004) 13, 441–444

SEIZURE

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CASE REPORT

Orgasmic aura—a report of seven cases

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KEYWORDS
Temporal lobe epilepsy;
Right hemisphere;
Presurgical evaluation;
Lateralisation;
Orgasm;
Sexual functions

Summary We report on seven patients who experienced an orgasmic aura at the start of their seizures. The patients (five women, two men) were aged 36–58. Three of seven patients described the exact nature of their auras only many years after their appearance, when the epilepsy diagnostic procedure became more intensive due to drug resistance. Moreover, one patient even refused any new therapeutical options due to the reportedly positive role of the orgasmic aura in her life. All of our patients had temporal lobe epilepsy. The clinical picture, EEG, MRI or SPECT findings suggested a right temporal epileptic focus in six patients, while in one patient the epileptogenic region was localised in the left temporal lobe. In the latter case, the left hemisphere was speech-dominant, while in the other cases no Wada tests were done. Our results confirm that orgasmic aura could be considered as an ictal lateralising sign to the right hemisphere, however, it has no 100% lateralising value.

Introduction

Orgasm may be related to epileptic seizures in several ways. Epileptic somatosensory seizures can be manifested as a vaginal sensation, which may induce a compulsion to masturbate until orgasm. Orgasm may very rarely evoke epileptic seizures as a kind of reflex epilepsy mechanism. Orgasm may occur during epileptic seizures in women but rarely in men.[1] In our previous paper, we reported on a well-documented patient with an orgasmic aura in whom the right mesiotemporal seizure onset area was identified by ictal video-EEG monitoring with intracranial electrodes, MRI abnormality, and long-term seizure freedom after right anterior temporal lobectomy.[1] In that study, we also re-evaluated all published cases with orgasmic auras, paying special attention to the ictal onset area and we concluded that orgasmic aura is an ictal lateralisation sign to the right hemisphere which may help in identifying the epileptogenic region during presurgical evaluation. However, in our previous study, we published only one new case; the other cases were extracted from previously published case reports.[1] In the present paper, we report on seven new cases with orgasmic aura.

Seizure (2005) xxx, xxx–xxx

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Forced normalisation precipitated by lamotrigine

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Received 16 August 2004

KEYWORDS
Epilepsy;
Forced normalization;
Lamotrigine

Summary

Purpose: To report two patients with lamotrigine-induced forced normalization (FN).
Methods: Evaluation of the patient files, EEG, and video-EEG records, with special reference to the parallel clinical and EEG changes before, during, and after FN.
Results: This is the first documented report of lamotrigine-induced FN. The two epileptic patients (one of them was a 10-year-old girl) were successfully treated with lamotrigine. Their seizures ceased and interictal epileptiform events disappeared from the EEG record. Simultaneously, the patients displayed de novo occurrence of psychopathologic manifestations and disturbed behaviour. Reduction of the daily dose of LTG led to disappearance of the psychopathological symptoms and reappearance of the spikes but not the seizures.
Conclusions: Lamotrigine may precipitate FN in adults and children. Analysis of the cases showed that lamotrigine-induced FN is a dose-dependent phenomenon and can be treated by reduction of the daily dose of the drug.

Seizure (2005) 14, 52–57

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Factors affecting spiking related to sleep and wake states in temporal lobe epilepsy (TLE)

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KEYWORDS
Interictal spike;
Sleep;
Temporal lobe epilepsy

Summary The aim of the study was to investigate the influence of different clinical factors on spiking during sleep and wakefulness in temporal lobe epilepsy. The study included 38 temporal lobe epilepsy (TLE) patients who underwent long-term electroencephalography (EEG) monitoring. In addition to traditional sleep scoring, waking was subdivided into eyes opened (WEO) and eyes closed (WEC) states. The following spike measures were investigated: spiking rates for each state, mean spike rate, spiking stability across wake and sleep states and relative spike density for each state. These measures were investigated according to clinical variables, such as age, age at epilepsy onset, duration of epilepsy, seizure frequency, the presence of secondarily generalised tonic-clonic (SGTC) seizures and the data on epileptogenic lesions based on MRI. Spiking rates during most states and spiking stability showed a significant positive correlation with epilepsy duration. Relative spike density during sleep stage NREM3,4 significantly increased with age at epilepsy onset. Relative spike density during WEC was significantly higher in the presence of hippocampal sclerosis (HS). Spiking rate during REM was significantly higher if a patient had SGTC seizures. Our data provide evidence that different aspects of spiking are associated with different aspects of TLE. We suggest that spike behaviour analysis offer new aspects both for diagnosis and research.

Brain Topography, Volume 18, Number 2, Winter 2005 (©2005)
DOI: 10.1007/s10548-005-0280-z

Distribution of Spatial Complexity of EEG in Idiopathic Generalized Epilepsy and Its Change After Chronic Valproate Therapy

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Summary: The objective of this study was to investigate the global and regional spatial synchrony of the EEG background activity, and to assess the effect of chronic valproate therapy on spatial synchrony. 15 idiopathic generalized epilepsy (IGE) patients were examined and compared to 16 normal controls. Resting EEG with 19 channels was investigated before and during chronic administration of valproate (VPA). Omega, a single-valued measure of spatial covariance complexity, was calculated to assess the degree of spatial synchrony of EEG. Furthermore, a new parameter was defined to characterize the distribution of spatial synchrony (Antero-Posterior Complexity Ratio, APCR). Global Omega complexity was significantly lower in IGE compared to controls, while regional complexity showed significant differences only in the anterior region: the IGE group showed lower complexity. APCR was significantly lower in IGE. VPA therapy (1) lowered the global complexity, (2) increased regional complexity in the anterior region, but decreased it in the posterior region, and (3) increased APCR. In IGE lower complexity, i.e. enhanced spatial synchrony, was found, especially in the anterior cortical area. VPA modified the distribution of spatial synchrony in IGE patients towards that of normal controls, although the effect is not identical with full normalization of cortical bioelectric activity. Whether the observed change of spatial synchrony distribution may reflect the normalizing effect of valproate on the brain state is worth further investigation.

Key words: Human multichannel EEG; Idiopathic generalized epilepsy; Valproate; Spatial synchrony; Omega complexity; Antero-Posterior Complexity Ratio.

Epilepsy Research

Volume 70, Issues 2–3, August 2006, Pages 190–199

Quantitative EEG effects of carbamazepine, oxcarbazepine, valproate, lamotrigine, and possible clinical relevance of the findings

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http://dx.doi.org/10.1016/j.eplepsyres.2006.05.003

Abstract

Quantitative EEG (QEEG) effects of therapeutic doses of carbamazepine (CBZ), oxcarbazepine (OXC), valproate (VA) and lamotrigine (LA) monotherapy were investigated in patients with beginning epilepsy. Baseline waking EEG (EEG1) was recorded in the untreated state, the second EEG (EEG2) was done after 8 weeks of reaching the therapeutic dose. Left occipital data were used for analysis. QEEG target parameters were absolute band-power (delta: AD, theta: AT, alpha: AA, beta: AB), and alpha mean frequency (AMF). Group effects (untreated versus treated condition in the CBZ, VA, OXC, LA groups) were computed for each target parameter. One group with benign rolandic epilepsy remained untreated for clinical reasons and served to estimate the QEEG test–retest differences. In addition, the individual QEEG response to each drug was calculated as (EEG2 – EEG1). Results: statistically significant ($p < 0.05$) group differences indicated the QEEG domain systematically affected by the drugs. CBZ caused AT increase and AMF decrease. OXC caused AMF decrease. VA and LA did not decrease AMF (LA even increased it), but reduced broad-band power. Individual power and AMF changes showed considerable variability in each group. >0.5 Hz AMF decrease (that was reported to predict cognitive impairment in prior studies) occurred in 10/41 patients in the CBZ group but never in the OXC, VA, LA groups. The results may be utilized in planning further studies addressing the relationship between antiepileptic drugs and their CNS effects. In addition, the relationship of AED-related cognitive impairment and AMF changes was discussed.

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Clinical Neurophysiology xxx (2007) xxx–xxx

www.elsevier.com/locate/clinph

Lamotrigine decreases EEG synchronization in a use-dependent manner in patients with idiopathic generalized epilepsy ☆

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Accepted 23 November 2006

Abstract

Objective: To investigate the quantitative EEG effects of lamotrigine (LTG) monotherapy. Hypothesis: LTG was predicted to decrease thalamo-cortical neuronal synchronization in idiopathic generalized epilepsy (IGE).
Methods: Waking EEG background activity of 19 IGE patients was investigated before treatment and in the course of LTG monotherapy. Raw absolute power (RAP), raw percent power (RRP), and raw mean frequency (RMF) were computed for 19 electrodes and four frequency bands (delta = 1.5–3.5 Hz, theta = 3.5–7.5 Hz, alpha = 7.5–12.5 Hz, and beta = 12.5–25.0 Hz). Inter- and intrahemispheric coherence was computed for eight electrode pairs and the four frequency bands. In addition, scalp-averages were calculated for each variable. Group differences were computed by means of nonparametric statistics including correction for multiple comparisons.
Results: Main results were decreased delta and theta RAP ($p < 0.05$ for scalp-averages). LTG compressed the delta, theta, and alpha RAP datasets, reducing the upper limit of the scatter in particular. Spearman *r*-values indicated marked correlation between the starting values (RAPuntreated) and the LTG-related decrease (RAPtreated − RAPuntreated) in three bands: delta ($r = -0.72$; $p = 0.0005$), theta ($r = -0.59$; $p = 0.007$), and alpha ($r = -0.61$; $p = 0.006$). Thus, the greater the baseline neuronal synchronization, the marked the dampening effect of LTG on it. The remaining findings were decreased theta RRP, theta RMF, and increased alpha RMF ($p < 0.05$ for scalp-averages). The electrode-related changes were small but topographically consistent across the 19 electrode sites. LTG did not affect coherence.
Conclusions: 1. LTG partially normalized the spectral composition of EEG background activity. LTG decreased pathological thalamo-cortical synchronization in use-dependent manner. 2. LTG did not cause quantitative EEG alterations suggesting worsening of the physiological brain functions. Instead, its profile suggested a mild psychostimulant effect.
Significance: The results contribute to the understanding of the effect of LTG at the network level.

Keywords: Lamotrigine; Quantitative EEG; Idiopathic generalized epilepsy

Epilepsia, **48**(5):941–949, 2007
Blackwell Publishing, Inc.

Characteristic Distribution of Interictal Brain Electrical Activity in Idiopathic Generalized Epilepsy

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Summary: *Purpose:* To demonstrate the anatomic localization of the cortical sources of the interictal EEG activity in human idiopathic generalized epilepsy (IGE).

Methods: Multiple cortical and hippocampal sources of the interictal spontaneous EEG activity were investigated by low-resolution electromagnetic tomography in 15 untreated IGE patients and in 15 healthy controls. EEG activity (current density) in four frequency bands (delta: 1.5–3.5 Hz, theta: 3.5–7.5 Hz, alpha: 7.5–12.5 Hz, beta: 12.5–25.0 Hz) was computed for 2,397 voxels. Voxel-by-voxel group comparison was done between the patient and the control group. Voxels with $p < 0.01$ differences (between the two groups) were correlated with cortical anatomy.

Results: Areas of significantly increased or decreased activity were characterized by their anatomical extension and the frequency bands involved. Five areas of bilaterally increased activity were found: rostral part of the prefrontal cortex (delta, theta); posterior part of the insula (delta); hippocampus and mediobasal temporal cortex (all frequency bands); medial parietooccipital cortex (theta, alpha, beta); dorsal and polar parts of the occipital cortex (alpha). Bilaterally decreased delta, theta, alpha activity was found in the majority of the frontal and anterior parietal cortex on the lateral surface, and in parts of the medial surface of the hemispheres. The area of decreased beta activity was less extensive. The right lateral and laterobasal temporal cortex showed decreased delta, theta, alpha, and beta activity, while its left counterpart only showed decreased delta and alpha activity in a limited part of this area.

Conclusions: (1) Pathological interictal EEG activity is not evenly distributed across the cortex in IGE. The prefrontal area of increased activity corresponds to the area that is essential in the buildup of the ictal spike-wave paroxysms (absence seizures). The existence of the posterior "center of gravity" of increased EEG activity in IGE was confirmed. The frontal area of decreased activity might be related to the cognitive deficit described in IGE patients. (2) Increased activity in a lot of ontogenetically older areas (including the hippocampi) and decreased activity in the majority of the isocortex is a peculiar pattern that argues for a developmental hypothesis for IGE. **Key Words:** Idiopathic generalized—Epilepsy—EEG—LORETA

Seizure (2007) xxx, xxx–xxx

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Valproate decreases EEG synchronization in a use-dependent manner in idiopathic generalized epilepsy

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Received 8 April 2007; received in revised form 25 June 2007; accepted 10 July 2007

KEYWORDS
Valproate;
Use-dependent effect;
EEG;
Epilepsy;
Idiopathic generalized epilepsy

Summary

Introduction: In order to explore the mechanism of action of valproate (VPA) in idiopathic generalized epilepsy (IGE), the effect of VPA on cortical EEG activity was investigated. Hypothesis: VPA decreases EEG synchronization in the delta and theta frequency bands in a use-dependent manner in IGE patients.
Methods: First setting: EEG records of 17 untreated IGE patients (NAE group) were analyzed and compared to those of 15 healthy controls (NC group). Second setting: EEG recorded in the untreated condition (NAE) was compared to the EEG recorded in the treated condition (VPA) of the patient group. Technique and analysis: 2 min of eyes-closed, waking EEG background activity (without epileptiform potentials and artifacts) were analyzed. Absolute power (AP) and mean frequency (MF) were computed for 19 electrodes and four frequency bands (δ = 1.5–3.5 Hz, θ = 3.5–7.5 Hz, α = 7.5–12.5 Hz, β = 12.5–25.0 Hz). Log-transformed data entered further analysis. Group differences were computed by means of parametric statistics including correction for multiple comparisons. The VPA-related changes (APvpa – APnae) were correlated with the degree of the baseline abnormality (APnae) and the daily dose/serum levels of VPA.
Main results: Statistically significant ($p < 0.05$, corrected) changes in the first setting: diffuse delta, theta, alpha AP increase, mainly right hemispheric beta AP increase was found in the NAE group, as compared to the NC group. Second setting: VPA decreased delta and theta AP. Strong correlation was demonstrated between the degree of the initial AP abnormality and the VPA-related AP decrease. AP decrease did not correlate with the daily dose and the serum level of the drug.
Conclusion: The hypothesis that VPA decreased EEG synchronization in the delta and theta frequency bands in a use-dependent manner was supported. The findings contribute to the understanding of the action of VPA at the network level.

Epilepsy Research (2007) 75, 186–191

journal homepage: www.elsevier.com/locate/epilepsyres

Valproate selectively reduces EEG activity in anterior parts of the cortex in patients with idiopathic generalized epilepsy
A low resolution electromagnetic tomography (LORETA) study

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Received 11 March 2007; accepted 2 June 2007

KEYWORDS
Valproate;
Idiopathic generalized epilepsy;
LORETA

Summary
Purpose: To localize the cortical area where the anticonvulsive drug valproate (VPA) exerts its effect in patients with idiopathic generalized epilepsy (IGE).
Methods: In a prior study we investigated 15 IGE patients in the untreated condition and compared their low resolution electromagnetic tomography (LORETA) results to a normal control group. The investigation of these patients was continued in the present study. All the 15 patients were treated with VPA and were followed by the authors. EEG was recorded after 3 months of VPA treatment in the seizure-free patients. A total of 2 min of 19-channels, common reference-recorded, waking-relaxed background activity (without paroxysmal and other, non-stationary elements) was analyzed. ''Activity'' (current density, amper/meters squared) was given in four frequency bands (delta, theta, alpha, beta). Band-related group differences between the present LORETA results (treated condition) and the prior LORETA results (untreated condition) were computed for all the 2394 voxels by *t*-tests for interdependent datasets. The statistically significant ($p < 0.01$, uncorrected) differences of activity were projected to real cortical anatomy using the Talairach Brain Atlas.
Results: Statistically significant differences between the untreated and treated condition emerged in the delta and theta bands. VPA decreased delta and theta activity in the entire frontal cortex, insula, anterior temporal cortex and hippocampus, and in the anterior part of the parietal cortex.

Epilepsy Research (2008) xxx, xxx–xxx

journal homepage: www.elsevier.com/locate/epilepsyres

Imaging the cortical effect of lamotrigine in patients with idiopathic generalized epilepsy: A low-resolution electromagnetic tomography (LORETA) study

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Received 25 June 2007; received in revised form 7 June 2008; accepted 12 June 2008

KEYWORDS
Lamotrigine;
Idiopathic generalized epilepsy;
LORETA

Summary
Purpose: Anatomical localization of the cortical effect of lamotrigine (LTG) in patients with idiopathic generalized epilepsy (IGE).
Methods: 19 patients with untreated IGE were investigated. EEG was recorded in the untreated condition and 3 months later when LTG treatment abolished the seizures. 19-channel EEG was recorded, and a total of 2 min artifact-free, waking EEG was processed to low-resolution electromagnetic tomography (LORETA) analysis. Activity (that is, current source density, A/m^2) was computed in four frequency bands (delta, theta, alpha, and beta), for 2394 voxels that represented the cortical gray matter and the hippocampi. Group differences between the untreated and treated conditions were computed for the four bands and all voxels by multiple *t*-tests for interdependent datasets. The results were presented in terms of anatomical distribution and statistical significance.
Results: $p < 0.01$ (uncorrected) changes (decrease of activity) emerged in the theta and the alpha bands. Theta activity decreased in a large cluster of voxels including parts of the temporal, parietal, occipital cortex bilaterally, and in the transverse temporal gyri, insula, hippocampus, and uncus on the right side. Alpha activity decreased in a relatively smaller cortical area involving the right temporo-parietal junction and surrounding parts of the cortex, and part of the insula on the right side.

Brain Topogr
DOI 10.1007/s10548-008-0061-6

ORIGINAL PAPER

Three-dimensional Localization of Abnormal EEG Activity in Migraine

A Low Resolution Electromagnetic Tomography (LORETA) Study of Migraine Patients in the Pain-free Interval

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Accepted: 15 July 2008

Abstract Investigating the brain of migraine patients in the pain-free interval may shed light on the basic cerebral abnormality of migraine, in other words, the liability of the brain to generate migraine attacks from time to time. Twenty unmedicated "migraine without aura" patients and a matched group of healthy controls were investigated in this explorative study. 19-channel EEG was recorded against the linked ears reference and was on-line digitized. 60 × 2-s epochs of eyes-closed, waking-relaxed activity were subjected to spectral analysis and a source localization method, low resolution electromagnetic tomography (LORETA). Absolute power was computed for 19 electrodes and four frequency bands (delta: 1.5–3.5 Hz, theta: 4.0–7.5 Hz, alpha: 8.0–12.5 Hz, beta: 13.0–25.0 Hz). LORETA "activity" (=current source density, ampers/meters squared) was computed for 2394 voxels and the above specified frequency bands. Group comparison was carried out for the specified quantitative EEG variables. Activity in the two groups was compared on a voxel-by-voxel basis for each frequency band. Statistically significant (uncorrected $P < 0.01$) group differences were projected to cortical anatomy. Spectral findings: there was a tendency for more alpha power in the migraine that in the control group in all but two (F4, C3) derivations. However, statistically significant ($P < 0.01$, Bonferroni-corrected) spectral difference was only found in the right occipital region. The main LORETA-finding was that voxels with $P < 0.01$ differences were crowded in anatomically contiguous cortical areas. Increased alpha activity was found in a cortical area including part of the precuneus, and the posterior part of the middle temporal gyrus in the right hemisphere. Decreased alpha activity was found bilaterally in medial parts of the frontal cortex including the anterior cingulate and the superior and medial frontal gyri. Neither spectral analysis, nor LORETA revealed statistically significant differences in the delta, theta, and beta bands. LORETA revealed the anatomical distribution of the cortical sources (generators) of the EEG abnormalities in migraine. The findings characterize the state of the cerebral cortex in the pain-free interval and might be suitable for planning forthcoming investigations.

Keywords Migraine · EEG · Quantitative EEG · LORETA

Abbreviations

EEG	Electroencephalography
QEEG	Quantitative electroencephalography
LORETA	Low resolution electromagnetic tomography
MEG	Magnetoencephalography

A leggyakrabban használt klinikai neurofiziológiai vizsgálómódszerek és indikációs területük az orvosi gyakorlatban

Clemens Béla, Kondákor István, Diószeghy Péter

Klinikai neurofiziológia alatt legtöbben a régebben elektrofiziológiának nevezett klinikai vizsgálómódszerek összességét értik. Közülük széles körben elterjedt az elektroencefalográfia, a kiváltott válaszok vizsgálata, az elektromiográfia és elektroneurográfia. E módszerek a korszerű képalkotók (CT/MR) korszakában elvesztették korábbi indikációs területük egy részét, főleg azokat, amelyeken az említett képalkotók egyértelműen informatívabbnak bizonyultak. Azonban semmi nem magyarázza, miért történt tévesztés azokon a területeken, amelyeken továbbra sem nélkülözhetők. Téves elgondolás, hogy az elektrofiziológia és a morfológiai képalkotók viszonya vetélkedő; az „intelligens medicina" feladata éppen a különféle dimenziókban keletkező információ összehangolása egymással és a klinikai adatokkal. Ez a nézet az utóbbi években világszerte elterjedt, visszahelyezve jogaiba és elismertségébe a klinikai neurofiziológiát, amely alatt nagyrészt ma is az elektrofiziológiai módszereket értik. Az elismertség egyértelmű kifejezése, hogy az Európai Unió a közelmúltban önálló orvosi diszciplínaként ismerte el a klinikai neurofiziológiát. Az alábbi áttekintésben megkíséreljük a szemléleti változást elősegíteni azzal, hogy röviden összefoglaljuk a leggyakoribb, széles körben alkalmazott vizsgálómódszerek jellemzőit és diagnosztikus értékét az ideg-elme gyógyászat területén. (Az itt tárgyalandókon kívül még számos elektrofiziológiai vizsgálat létezik, amelyeket egy-egy speciális funkció vizsgálatára fejlesztettek ki.)

Az elvégzett vizsgálat még nem minden. Az elkerülhetetlen specializáció átka ugyanis, hogy a klinikai neurofiziológiai módszerek a klinikus számára egyre kevésbé érthető nyelven adják meg az eredményt, és ez a helyzet a módszerek és lehetőségek bővülésével csak rosszabbodni fog. Ezért az eddiginél is intenzívebb eszmecserére van szükség a klinikus és a neurofiziológus között. Az előbbinek kellő részletességgel ismertetnie kell, milyen kórkép gyanújában, pontosan mire kíváncsi. A módszer vagy módszerek kombinációjának megválasztását, különösen bonyolult esetekben, jobb a laboratóriumi szakemberre bízni. Az ő részéről viszont a klinikus kérdésének kellően alapos megválaszolása szükséges. Könnyen belátható, hogy az intenzív kapcsolat nélkül hiába a költséges felszerelés és a szakértelem, a két értetlenkedő fél között elveszhet a lényeg, néha a beteg is.

Téves elgondolás, hogy az elektrofiziológia és a morfológiai képalkotók viszonya vetélkedő.

Elektroencefalográfia

Az elektroencefalográfia (EEG) kizárólag az agykéregben termelődő áramokból keletkezik. A 0,5–30 Hz frekvenciasávba tartozó hullámok alakja nagyban torzul, amíg az agykéregtől az elektromosan eltérő ellenállású szöveti rétegeken át eljutnak a fejbőrig, ahol regisztráljuk őket. Fontos tudni, hogy az EEG-aktivitás valamennyi (ismert és ismeretlen) agykérgi folyamat eredője, de nagyban befolyásolják az agykérgi állapotot vezérlő, kéreg alatti szerkezetek is. Az EEG-hullámformák és -ritmusok fő meghatározói az életkor és tudati állapot (alvás-ébrenlét ciklus). Az EEG-leletezés során megítélendők az állandóan jelen levő, úgynevezett háttértevékenység jellemzői, a frekvencia és amplitúdó eloszlása az egyes agyi területek felett, továbbá

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Epilepsy Research (2009) xxx, xxx–xxx

ELSEVIER

journal homepage: www.elsevier.com/locate/epilepsyres

The electrophysiological ''delayed effect'' of focal interictal epileptiform discharges. A low resolution electromagnetic tomography (LORETA) study

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Received 16 September 2008; received in revised form 24 March 2009; accepted 27 March 2009

KEYWORDS
Epilepsy;
Spike;
Delayed effect of the spike;
EEG;
LORETA

Summary Collating the findings regarding the role of focal interictal epileptiform discharges (IEDs) on CNS functions raises the possibility that IEDs might have negative impact that outlasts the duration of the spike-and-wave complexes. The aim of this study was the electrophysiological demonstration of the ''delayed effect'' of the IEDs. 19-channel, linked-ears referenced, digital waking EEG records of 11 children (aged 6–14 years, eight with idiopathic, three with cryptogenic focal epilepsy, showing a single spike focus) were retrospectively selected from our database. A minimum of 20 (preferably, 30), 2-s epochs containing a single focal spike-and-wave complex were selected (Spike epochs). Thereafter, Postspike-1 (Ps1), Postspike-2 (Ps2) and Postspike-3 (Ps3) epochs were selected, representing the first and second seconds (Ps1), the third and fourth seconds (Ps2) and the fifth and sixth seconds (Ps3) after the Spike epoch, respectively. Interspike epochs (Is) were selected at a distance at least 10 s after the Spike epoch. *Individual analysis*: the frequency of interest (FOI = the individual frequency of the wave component of the IEDs), and the region of interest (ROI = the site of the IEDs) were identified by reading the raw EEG waveform and the instant power spectrum. Very narrow band LORETA (low resolution electromagnetic tomography) analysis at the FOI and ROI was carried out. Age-adjusted, Z-transformed LORETA ''activity'' (= current source density, ampers/meters squared) was compared in the Spike, Ps1, Ps2, Ps3 and Is epochs. Findings: the greatest (uppermost pathological) Z-scores and the greatest spatial extension of the LORETA-abnormality were always found in the Spike epochs, followed by the gradual decrease of activity in terms of severity and spatial extension in the Ps1, Ps2, Ps3 epochs. The lowest (baseline) level and extension

Clinical Neurophysiology 121 (2010) 848–858

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Theta EEG source localization using LORETA in partial epilepsy patients with and without medication ☆

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ARTICLE INFO

Article history:
Accepted 20 January 2010
Available online 23 February 2010

Keywords:
Epilepsy
EEG
LORETA
Theta activity

ABSTRACT

Objective: To investigate and localize the sources of spontaneous, scalp-recorded theta activity in patients with partial epilepsy (PE).
Methods: Nine patients with beginning, untreated PE (Group 1), 31 patients with already treated PE (Group 2), and 14 healthy persons were investigated by means of spectral analysis and LORETA, low resolution electromagnetic tomography (1 Hz very narrow band analysis, age-adjusted, Z-scored values). The frequency of main interest was 4–8 Hz.
Results: **Group analysis:** Group 1 displayed bilateral theta maxima in the temporal theta area (TTA), parietal theta area (PTA), and frontal theta area (FTA). In Group 2, theta activity increased all over the scalp as compared to the normative mean ($Z = 0$) and also to Group 1. Maximum activity was found in the TTA, PTA, and FTA. However, in the PTA and FTA the centers of the abnormality shifted towards the medial cortex. **Individual analysis:** all the patients showed preferential activation (maximum Z-values) within one of the three theta areas.
Conclusions: EEG activity in the theta band is increased in anatomically meaningful patterns in PE patients, which differs from the anatomical distribution of theta in healthy persons.
Significance: The findings contribute to our understanding of the sources of theta rhythms and the pathophysiology of PE.

Epilepsy Research (2010) 91, 94–100

journal homepage: www.elsevier.com/locate/epilepsyres

Quantitative EEG abnormalities in persons with ''pure'' epileptic predisposition without epilepsy: A low resolution electromagnetic tomography (LORETA) study

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Received 21 December 2009; received in revised form 22 June 2010; accepted 4 July 2010
Available online 31 July 2010

KEYWORDS
Epilepsy;
Epileptic predisposition;
EEG;
LORETA

Summary
Objective: Epileptic predisposition means genetically determined, increased seizure susceptibility. Neurophysiological evaluation of this condition is still lacking. In order to investigate ''pure epileptic predisposition'' (without epilepsy) in this pilot study the authors prospectively recruited ten persons who displayed generalized tonic–clonic seizures precipitated by 24 or more hours of sleep deprivation but were healthy in any other respects.
Methods: 21-channel EEGs were recorded in the morning, in the waking state, after a night of sufficient sleep in the interictal period. For each person, a total of 120 s artifact-free EEG was processed to low resolution electromagnetic tomography (LORETA) analysis. LORETA activity (Ampers/meters squared) was computed for 2394 voxels, 19 active electrodes and 1 Hz very narrow bands from 1 to 25 Hz. The data were compressed into four frequency bands (δ: 0.5–4.0 Hz, θ: 4.5–8.0 Hz, α: 8.5–12.0 Hz, β: 12.5–25.0 Hz) and projected onto the MRI figures of a digitized standard brain atlas. The band-related LORETA results were compared to those of ten, age- and sex-matched healthy persons using independent *t*-tests. $p < 0.01$ differences were accepted as statistically significant.
Results: Statistically significant decrease of α activity was found in widespread, medial and lateral parts of the cortex above the level of the basal ganglia. Maximum α decrease and statistically significant β decrease were found in the left precuneus. Statistically not significant differences were δ increase in the medial-basal frontal area and θ increase in the same area and in the basal temporal area.

Epilepsy Research (2012) 99, 281–292

journal homepage: www.elsevier.com/locate/epilepsyres

EEG-LORETA endophenotypes of the common idiopathic generalized epilepsy syndromes

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Received 5 July 2011; received in revised form 14 November 2011; accepted 11 December 2011
Available online 11 January 2012

KEYWORDS
EEG;
LORETA;
Endophenotype;
Absence;
Juvenile myoclonic epilepsy

Summary
Objective: We tested the hypothesis that the cortical areas with abnormal local EEG synchronization are dissimilar in the three common idiopathic generalized epilepsy (IGE) phenotypes: IGE patients with absence seizures (ABS), juvenile myoclonic epilepsy (JME) and epilepsy with generalized tonic–clonic seizures exclusively (EGTCS).
Patients and methods: Groups of unmedicated ABS, JME and EGTCS patients were investigated. Waking EEG background activity (without any epileptiform potentials) was analyzed by a source localization method, LORETA (Low Resolution Electromagnetic Tomography). Each patient group was compared to a separate, age-matched group of healthy control persons. Voxel-based, normalized broad-band (delta, theta, alpha, and beta) and very narrow band (VNB, 1 Hz bandwidth, from 1 to 25 Hz) LORETA activity (=current source density, A/m^2) were computed for each person. Group comparison included subtraction (average patient data minus average control data) and group statistics (multiple t-tests, where Bonferroni-corrected $p < 0.05$ values were accepted as statistically significant).
Results: Statistically not significant main findings were: overall increased delta and theta broad band activity in the ABS and JME groups; decrease of alpha and beta activity in the EGTCS group. Statistically significant main findings were as follows. *JME group*: bilaterally increased theta activity in posterior (temporal, parietal, and occipital) cortical areas; bilaterally

Epilepsy Research (2011) 96, 11–23

journal homepage: www.elsevier.com/locate/epilepsyres

EEG functional connectivity of the intrahemispheric cortico-cortical network of idiopathic generalized epilepsy

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Received 7 December 2010; received in revised form 22 March 2011; accepted 24 April 2011
Available online 20 May 2011

KEYWORDS
Epilepsy;
Idiopathic generalized epilepsy;
Network;
LORETA Source Correlation

Summary
Aims: Intrahemispheric, cortico-cortical EEG functional connectivity (fC) was investigated in untreated patients with idiopathic generalized epilepsy (IGE) in this explorative study.
Patients and methods: Group comparison was carried out between 19, drug-naive IGE patients and 19, matched healthy persons. 90×2 s of 19 channels waking, interictal background EEG signal (without epileptiform potentials) were processed to the LORETA (low resolution electromagnetic tomography) software to compute current source density for 2394 voxels representing parcels of the cerebral cortex for 25 very narrow bands of 1 Hz bandwidth (VNBs) from 1 to 25 Hz. EEG fC was investigated among the already localized sources. Pearson correlation coefficients (R) were computed among the 33 regions of interest (ROI) within the left and within the right hemisphere, separately. Group differences were computed by means of *t*-statistics. Corrected $p < 0.05$ differences were accepted as statistically significant.
Main results: (1) The anatomical patterns of the fC differences showed great frequency-dependency. (2) Hemispheric asymmetry was prominent within most VNBs. (3) Decreased fC in the IGE group was found across all VNBs in the 1–6 Hz frequency range as compared to mixed patterns comprising both increased and decreased fC at >6 Hz frequencies. (4) In the 5–25 Hz range, decreased fC dominated in the anterior, increased fC in the posterior parts of the cortex. (5) The results delineated an anterior and a posterior network.

Epilepsy Research (2011) xxx, xxx–xxx

journal homepage: www.elsevier.com/locate/epilepsyres

EEG background activity is abnormal in the temporal and inferior parietal cortex in benign rolandic epilepsy of childhood: A LORETA study

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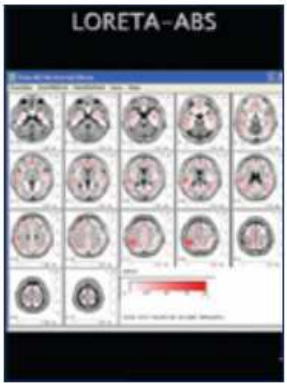
Received 29 June 2011; received in revised form 18 August 2011; accepted 20 August 2011

KEYWORDS
Benign rolandic epilepsy;
EEG;
LORETA;
Attention;
Language

Summary
Introduction: Benign rolandic epilepsy of childhood (BERS) is an epilepsy syndrome with presumably genetic-developmental etiology. The pathological basis of this syndrome is completely unknown. We postulated that a developmental abnormality presumably results in abnormal EEG background activity findings.
Patients and methods: 20 children with typical BERS and an age- and sex-matched group of healthy control children underwent EEG recording and analysis. 60×2 s epochs of waking EEG background activity (without epileptiform potentials and artifacts) were analyzed in the 1–25 Hz frequency range, in very narrow bands (VNB, 1 Hz bandwidth). LORETA (Low Resolution Electromagnetic Tomography) localized multiple distributed sources of EEG background activity in the Talairach space. LORETA activity (current source density) was computed for 2394 voxels and 25 VNBs. Normalized LORETA data were processed to voxel-wise comparison between the BERS and control groups. Bonferroni-corrected $p < 0.05$ Student's t-values were accepted as statistically significant.
Results: Increased LORETA activity was found in the BERS group (as compared to the controls) in the left and right temporal lobes (fusiform gyri, posterior parts of the superior, middle and inferior temporal gyri) and in the angular gyri in the parietal lobes, in the 4–6 Hz VNBs, mainly at 5 Hz.

Ideggyógyászati Szemle - 2011;64(9-10)

LORETA (Low Resolution Electromagnetic Tomography): háromdimenziós EEG-forráslokalizáló módszer



Clemens Béla

A szerző a LORETA (Low Resolution Electromagnetic Tomography) EEG-forráslokalizáló módszert ismerteti vázlatosan, különös tekintettel az irodalomban explicit módon nem részletezett adatokra.

Kulcsszavak

EEG, LORETA, forráslokalizáció

MAYO CLINIC

CASE REPORT

Electrical Storm in the Brain and in the Heart: Epilepsy and Brugada Syndrome

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Abstract

We describe a patient with the coincidence of 2 ion channel disorders with autosomal dominant inheritance: Brugada syndrome, a potentially fatal cardiac condition, and cryptogenic focal epilepsy, likely due to a neurologic channelopathy. Although Brugada syndrome was discovered incidentally, most of the clinical features of epilepsy in this patient shared the risk factor characteristics of sudden unexplained death in epilepsy syndrome. This case provides additional information on the potential interaction between ion channel abnormalities in the heart and in the brain. Furthermore, it may suggest that patients with epilepsy at increased risk for sudden unexplained death in epilepsy syndrome should undergo a careful cardiac evaluation.

 ■ Mayo Clin Proc. 2013;88(10):1167-1173

Brugada syndrome (BS) is a rare autosomal dominant genetic disorder hallmarked by the "Brugada electrocardiogram (ECG)," which displays a "pseudo–right bundle branch block" appearance with an ST elevation (STE) in leads V_1 to V_3, and sudden cardiac death due to ventricular arrhythmias in patients with apparently normal hearts.[1] The ECG abnormality and the susceptibility to ventricular arrhythmias are due to a loss-of-function mutation in the *HCN5A* gene encoding the α subunit of the sodium channel current of the cell membrane. Brugada syndrome is estimated to be responsible for at least 4% of all sudden deaths and at least 20% of deaths in patients with structurally normal hearts.[1] Sudden cardiac death occurs at a mean age of 41 years, typically at rest and at night. Presumed precipitating factors of the ventricular arrhythmias include an increase in vagal tone and a febrile state.

The prevalence of epilepsy is 0.5% to 1%. It is known to carry an up to 24-fold risk of sudden death compared with the general population,[2] an entity called sudden unexplained death in epilepsy (SUDEP) syndrome.[3] The role of ventricular arrhythmias is assumed in at least some of these incidents, and the coincidence of epilepsy and some potentially lethal primary cardiac ion channel disorders has recently been reported.[4-6] We describe a patient with asymptomatic BS and concomitant focal epilepsy likely related to abnormal ion channel function with an autosomal dominant inheritance.

CASE REPORT

A 41-year-old man was referred for urgent coronary intervention because of a sharp pain in the right side of his chest that had started an hour earlier. The 12-lead ECG transmitted by the ambulance team from the patient's home and that recorded on his arrival at the hospital are displayed in Figure 1. Transthoracic echocardiography indicated normal left ventricular function with no wall motion abnormality, and the levels of cardiac enzymes and electrolytes were repeatedly within the reference ranges. A computed tomographic angiogram revealed no evidence of aortic dissection or pulmonary embolism. His medical history included the diagnosis of epilepsy at age 16 years on the basis of recurrent seizure spells. His recent medications were levetiracetam and oxcarbazepine. His family history was negative for any cardiac or neurologic disease.

The patient was admitted to the hospital for evaluation of the abnormalities observed on the initial ECG. Slow intravenous administration of procainamide (400 mg) with an entirely normal starting ECG pattern initiated ventricular tachycardia (VT) at 187 beats/min, which terminated spontaneously after 85 seconds, and a type I Brugada ECG pattern appeared shortly after the VT (Figure 2, A). The patient remained conscious during the VT. A cardiac electrophysiologic study was also performed, and polymorphic VT at 250 beats/min was induced using programmed electrical stimulation from the right ventricular apex with 2 extrastimuli. This VT changed to a regular

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Epilepsy Research (2013) xxx, xxx–xxx

journal homepage: www.elsevier.com/locate/epilepsyres

Neurophysiology of juvenile myoclonic epilepsy: EEG-based network and graph analysis of the interictal and immediate preictal states

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Received 7 February 2013; received in revised form 10 May 2013; accepted 28 June 2013

KEYWORDS
Juvenile myoclonic epilepsy;
Network;
EEG;
Functional connectivity;
Graph analysis

Summary
Introduction: The neuronal mechanisms of enduring seizure propensity and seizure precipitation in juvenile myoclonic epilepsy (JME) are not known. We investigated these issues, within the framework of the ''network concept'' of epilepsy.
Methods: Design1: 19, unmedicated JME patients were compared with nineteen, age-, and sex-matched normal control persons (NC). A total of 120 s, artifact-free, paroxysm-free, eyes-closed, resting state EEG background activity was analyzed for each person. Design2: interictal and immediate preictal periods of the JME patients were compared in order to explore interictal–preictal network differences. For both comparison designs, statistically significant differences of EEG functional connectivity (EEGfC), nodal and global graph parameters were evaluated.
Main results: Design1: maximum abnormalities were: increased delta, theta, alpha1 EEGfC and decreased alpha2 and beta EEGfC in the JME group as compared to the NC group, mainly among

Seizure 23 (2014) 213–221

Contents lists available at ScienceDirect

Seizure

journal homepage: www.elsevier.com/locate/yseiz

Uppermost synchronized generators of spike–wave activity are localized in limbic cortical areas in late-onset absence status epilepticus

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ARTICLE INFO

Article history:
Received 14 April 2013
Received in revised form 25 November 2013
Accepted 27 November 2013

Keywords:
Absence status epilepticus
LORETA
LORETA Source Correlation

ABSTRACT

Purpose: Absence status (AS) epilepticus with generalized spike–wave pattern is frequently found in severely ill patients in whom several disease states co-exist. The cortical generators of the ictal EEG pattern and EEG functional connectivity (EEGfC) of this condition are unknown. The present study investigated the localization of the uppermost synchronized generators of spike–wave activity in AS.
Method: Seven patients with late-onset AS were investigated by EEG spectral analysis, LORETA (Low Resolution Electromagnetic Tomography) source imaging, and LSC (LORETA Source Correlation) analysis, which estimates cortico-cortical EEGfC among 23 ROIs (regions of interest) in each hemisphere.
Results: All the patients showed generalized ictal EEG activity. Maximum Z-scored spectral power was found in the 1–6 Hz and 12–14 Hz frequency bands. LORETA showed that the uppermost synchronized generators of 1–6 Hz band activity were localized in frontal and temporal cortical areas that are parts of the limbic system. For the 12–14 Hz band, abnormally synchronized generators were found in the antero-medial frontal cortex. Unlike the rather stereotyped spectral and LORETA findings, the individual EEGfC patterns were very dissimilar.
Conclusion: The findings are discussed in the context of nonconvulsive seizure types and the role of the underlying cortical areas in late-onset AS. The diversity of the EEGfC patterns remains an enigma. Localizing the cortical generators of the EEG patterns contributes to understanding the neurophysiology of the condition.

EREDETI KÖZLEMÉNY

AZ MDS-UPDRS MAGYAR VALIDÁCIÓJA: MIÉRT SZÜKSÉGES ÚJABB PARKINSON-PONTOZÓSKÁLA?

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VALIDATION OF THE HUNGARIAN MDS-UPDRS: WHY DO WE NEED A NEW PARKINSON SCALE?

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Ideggyogy Sz 2014;67(3–4):129–134.

Bevezetés – Az 1986-os Egységesített Parkinson Pontozóskála (UPDRS) utódjaként kifejlesztett, a Movement Disorder Society által fémjelzett Egységesített Parkinson Pontozóskálát (MDS-UPDRS) 2008-ban véglegesítették. A skála egyéb nyelvre történő hivatalos fordításához az MDS négy lépésből álló szigorú validálási módszert dolgozott ki: 1. fordítás/visszafordítás, 2. kognitív előtesztelés, 3. nagy beteganyagon történő tesztelés és 4. klinimetrikus analízis. Vizsgálatunk célja az MDS-UPDRS ismertetése és a magyar nyelvi validáció folyamatának és eredményeinek bemutatása.

Módszertan – Első lépésben az MDS-UPDRS-t magyarra lefordítottuk, amit független munkacsoport angolra visszafordított. Miután a visszafordított szöveget az MDS-UPDRS bizottsága elemezte, a magyar szöveg érthetőségét két kognitív előteszteléssel ellenőriztük. A validálási folyamat harmadik fázisában a magyar verziót 357 Parkinson-kóros betegen vettük fel. Ezt követően ellenőrző faktoranalízis segítségével megvizsgáltuk, hogy a magyar MDS-UPDRS faktorszerkezete mennyire illeszkedik az angol verzió faktorszerkezetéhez. Az általunk lefordított skálát akkor tekinthet-

Background – The Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS) has been published in 2008 as the successor of the original UPDRS. The MDS-UPDRS organizing team developed guidelines for the development of official non-English translations consisting of four steps: translation/back-translation, cognitive pretesting, large field testing, and clinimetric analysis. The aim of this paper was to introduce the new MDS-UPDRS and its validation process into Hungarian.

Methods – Two independent groups of neurologists translated the text of the MDS-UPDRS into Hungarian and subsequently back-translated into English. After the review of the back-translated English version by the MDS-UPDRS translation administration team, cognitive pretesting was conducted with ten patients. Based on the results of the initial cognitive pretesting, another round was conducted. For the large field testing phase, the Hungarian official working draft version of MDS-UPDRS was tested with 357 patients with Parkinson's disease (PD). Confirmatory factor analyses (CFA) determined whether the factor structure for the English-lan-

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Érkezett: 2014. február 1. Elfogadva: 2014. február 17.

www.elitmed.hu

Epilepsy Research (2013) 106, 128–135

journal homepage: www.elsevier.com/locate/epilepsyres

Remission of benign epilepsy with rolandic spikes: An EEG-based connectivity study at the onset of the disease and at remission

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Received 12 January 2013 ; received in revised form 8 April 2013; accepted 19 April 2013
Available online 19 May 2013

KEYWORDS
Epilepsy;
Remission;
EEG;
Network theory;
LORETA;
LORETA Source Correlation

Summary
Purpose: The neuronal mechanisms of remission of epilepsy are not known. Based on the principles of the ''network theory of epilepsy'' we postulated the existence of abnormal cortico-cortical interactions at the onset of epilepsy (Hypothesis-1), and postulated that remission is associated with the decrease or disappearance of the abnormal quantitative EEG findings (Hypothesis-2).
Methods: Four children with benign epilepsy with rolandic sharp waves (BERS) were investigated. 21-channel EEG was recorded at the onset of the disease (Setting No. 1) and in remission (Setting No. 2). Local EEG synchronization was estimated by LORETA (low resolution electromagnetic tomography). Remote EEG synchronization (intra-hemispheric, cortico-cortical EEG functional connectivity, EEGfC) was computed by the LSC (LORETA Source Correlation) method, among 23 regions of interest (ROI) in both hemispheres. Both local and remote EEG synchronization were evaluated in very narrow frequency bands of 1 Hz bandwidth (VNB), from 1 to 25 Hz.
Results: Individual results were presented. Abnormal but topographically very dissimilar LORETA and LSC findings were found at the onset of the disease. The disappearance of the initial abnormalities was found in Setting No. 2. An unforeseen finding was the presence of abnormal EEGfC results in Setting No. 2.
Discussion: The authors confirmed both hypotheses. The dissimilarity of the initial abnormalities is in accord with the network concept of epilepsy and the etiology of BERS. The disappearance

Epilepsy Research (2014) 108, 1896—1903

journal homepage: www.elsevier.com/locate/epilepsyres

Valproate treatment normalizes EEG functional connectivity in successfully treated idiopathic generalized epilepsy patients

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Received 25 June 2014; received in revised form 13 September 2014; accepted 29 September 2014
Available online 13 October 2014

KEYWORDS
Idiopathic generalized epilepsy;
Valproate;
EEG functional connectivity

Summary
Aim: To investigate the effect of chronic VPA treatment of EEG functional connectivity in successfully treated idiopathic generalized epilepsy (IGE) patients.
Patients and methods: 19-channel waking, resting-state EEG records of 26 IGE patients were analyzed before treatment (IGE) and after the 90th day of treatment (VPA), in seizure-free condition. Three minutes of artifact-free EEG background activity (without epileptiform potentials) was analyzed for each patient in both conditions. A group of 26 age-matched healthy normative control persons (NC) was analyzed in the same way. All the EEG samples were processed to LORETA (Low Resolution Electromagnetic Tomography) to localize multiple distributed sources of EEG activity. Current source density time series were generated for 33 regions of interest (ROI) in each hemisphere for four frequency bands. Pearson correlation coefficients (R) were computed between all ROIs in each hemisphere, for four bands across the investigated samples. R values corresponded to intrahemispheric, cortico-cortical functional EEG connectivity (EEGfC). Group and condition differences were analyzed by statistical parametric network method.

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http://dx.doi.org/10.1016/j.eplepsyres.2014.09.032

MIRTAZAPIN ÁLTAL PROVOKÁLT FOKÁLIS MOTOROS ROHAMOK ÉS STATUS EPILEPTICUS

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PRINT | Hungarian | http://dx.doi.org/10.18071/isz.68.0286 | www.elitmed.hu

Kapcsolódó cikk online

A mirtazapin gyakran használt tetraciklikus antidepresszívum, amelynek epilepsziás rohamot provokáló hatásáról alig van adat. A szerzők egy 39 éves, nem epilepsziás, depresszió miatt kezelt beteg esetét ismertetik, aki esetében az addig szedett mirtazapin dózisának növelésével jobb alsó végtagi fokális clonusos epilepsziás rohamok jelentkeztek. További dózisemelés mellett fokális motoros status epilepticus alakult ki. Ezt videó-EEG segítségével rögzítették, majd az intravénás benzodiazepin hatástalanságát tapasztalva, intravénás levetiracetam adásával megszüntették. A mirtazapin elhagyása és carbamazepin beállítása után újabb rohamok nem jelentkeztek, a beteg a carbamazepin elhagyása után is rohammentes maradt a 13 hónapos követés során. A klinikai adatok és a laboratóriumi, EEG- és képalkotó leletek a mirtazapin rohamprovokáló hatására utalnak.

Kulcsszavak: *status epilepticus, mirtazapin*

FOCAL MOTOR SEIZURES AND STATUS EPILEPTICUS PROVOKED BY MIRTAZAPINE

Dömötör J, MD; Clemens B, MD, PhD

Ideggyogy Sz 2015;68(7–8):286–288.

The seizure-provoking effect of the tetracyclic antidepressant mirtazapine is not a well-known adverse effect of the drug. The authors report on a 39-year-old non-epileptic patient who had been treated for depression with the usual daily dose of mirtazapine. Having increased the daily dose of the drug from 30 to 45 milligrams he experienced a few clonic seizures of the right lower limb. This symptom and insomnia erroneously intended the patient to further increase the daily dose of mirtazapine, which immediately resulted in the evolution of focal clonic status epilepticus in the same limb. After admission, this condition was recorded by video-EEG and abolished by intravenous administration of levetiracetam after the intravenous clonazepam had been ineffective. Discontinuation of mirtazapine and administration of carbamazepine resulted in completely seizure-free state that persisted even after carbamazepine treatment was terminated. The clinical and laboratory data indicate the seizure-provoking effect of mirtazapine in the reported case.

Keywords: *status epilepticus, mirtazapine*

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Érkezett: 2013. szeptember 7. Elfogadva: 2013. november 14.

A mirtazapin tetraciklikus, a noradrenerg és szerotoninerg rendszerre ható antidepresszívum. Erőteljes antidepresszív hatása a triciklikus és a szerotonin-újrafelvételt gátló (SSRI) típusú antidepresszívumokhoz viszonyítva kedvező mellékhatásprofillal társul. Leggyakoribb mellékhatásai: álmosság (54%), szájszáradás (25%), megnövekedett étvágy (17%), testsúlynövekedés (12%), szédülés (7%)[1]. Mirtazapin által provokált epilepsziás rohamra egyetlen megbízható utalás van az irodalomban: egy, phenytoinkezelés mellett rohammentes epilepsziás beteg esetében az első mirtazapindózis bevételét követő éjszakán epilepsziás roham jelentkezett, majd a rákövetkező éjszakán megis-

EREDETI KÖZLEMÉNY

EGYEDI LORETA-ABNORMALITÁSOK VIZSGÁLATA IDIOPATHIÁS GENERALIZÁLT EPILEPSZIÁKBAN

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PRINT | Hungarian | http://dx.doi.org/10.18071/isz.69.0001 | www.elitmed.hu

Háttér és cél – Idiopathiás generalizált epilepsziában (IGE) gyűlnek az agykérgi ictogen állapotra vonatkozó strukturális és funkcionális képalkotó vizsgálatok eredményei, de a kvantitatív EEG (qEEG) -jellemzőket csak csoportszinten vizsgálták. Munkánkban az EEG-háttértevékenység egyedi vonásait vizsgáltuk IGE-betegekben, gyógyszermentes és antiepileptikummal kezelt állapotban.
Módszerek – Prospektív vizsgálatban IGE-betegek nyugalmi-ébrenléti EEG-aktivitását elemeztük gyógyszermentes állapotban és a gyógyszeres kezelés 3–6. hónapja között. Emellett retrospektíve gyűjtött IGE-betegekben a qEEG-lelet állandóságát is vizsgáltuk. Tizenkilenc csatornás EEG-felvételekből betegenként összesen kétpercnyi háttértevékenységet elemeztük LORETA (Low Resolution Electromagnetic Tomography) módszerrel. A LORETA-aktivitás abszolút értékeit Z-transzformációnak vetettük alá, majd színkódoltan, MRI-templáton jelenítettük meg. A [+3Z] és [–3Z] közti intervallumon kívül eső értékeket kórosnak minősítettük.
Eredmények – 1. Gyógyszermentes állapotban a LORETA-lelet kóros volt a betegek 41–50%-ában. 2. A kóros esetekben nagy interindividuális változatosság jelentkezett. 3. IGE-betegek többségében a kóros aktivitást mutató agykérgi terület kétoldali és szimmetrikus volt. 4. A maximális eltérés a legtöbb IGE-betegben a frontális vagy temporalis kéregben jelentkezett. 5. A rohammentesség többnyire LORETA-normalizációval járt, a perzisztáló rohamok perzisztáló LORETA-abnormalitással.

INDIVIDUAL EVALUATION OF LORETA ABNORMALITIES IN IDIOPATHIC GENERALIZED EPILEPSY

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Ideggyogy Sz 2016;69(1–2):000–000.

Background – Contemporary neuroimaging methods disclosed structural and functional cerebral abnormalities in idiopathic generalized epilepsies (IGEs). However, individual electrical (EEG) abnormalities have not been evaluated yet in IGE patients.
Methods – IGE patients were investigated in the drug-free condition and after 3-6 month of antiepileptic treatment. To estimate the reproducibility of qEEG variables a retrospective recruited cohort of IGE patients was investigated. 19-channel resting state EEG activity was recorded. For each patient a total of 2 minutes EEG activity was analyzed by LORETA (Low Resolution Electromagnetic Tomography). Raw LORETA values were Z-transformed and projected to a MRI template. Z-values outside within the [+3Z] to [-3Z] range were labelled as statistically abnormal.
Results – 1. In drug-free condition, 41-50% of IGE patients showed abnormal LORETA values. 2. Abnormal LORETA findings showed great inter-individual variability. 3. Most abnormal LORETA-findings were symmetrical. 4. Most maximum Z-values were localized to frontal or temporal cortex. 5. Succesfull treatment was mostly coupled with disappearence of LORETA-abnormality, persistent seizures were accompanied by persistent LORETA abnormality.
Discussion – 1. LORETA abnormalities detected in the untreated condition reflect seizure-generating property of the cortex in IGE patients. 2. Maximum LORETA-Z abnormalities were topographically congruent with structural

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Érkezett: 2014. augusztus 8. Elfogadva: 2014. november 27.

Seizure 35 (2016) 50–55

Contents lists available at ScienceDirect

Seizure

journal homepage: www.elsevier.com/locate/yseiz

Increased resting-state EEG functional connectivity in benign childhood epilepsy with centro-temporal spikes

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ARTICLE INFO

Article history:
Received 9 December 2014
Received in revised form 15 November 2015
Accepted 3 January 2016

Keywords:
EEG functional connectivity
Benign childhood epilepsy with centro-temporal spikes

ABSTRACT

Purpose: To explore intrahemispheric, cortico-cortical EEG functional connectivity (EEGfC) in benign childhood epilepsy with rolandic spikes (BECTS).
Methods: 21-channel EEG was recorded in 17 non-medicated BECTS children and 19 healthy controls. 180 s of spike- and artifact-free activity was selected for EEGfC analysis. Correlation of Low Resolution Electromagnetic Tomography- (LORETA-) defined current source density time series were computed between two cortical areas (region of interest, ROI). Analyses were based on broad-band EEGfC results. Groups were compared by statistical parametric network (SPN) method. Statistically significant differences between group EEGfC values were emphasized at $p < 0.05$ corrected for multiple comparison by local false discovery rate (FDR).
Results: (1) Bilaterally increased beta EEGfC occurred in the BECTS group as compared to the controls. Greatest beta abnormality emerged between frontal and frontal, as well as frontal and temporal ROIs. (2) Locally increased EEGfC emerged in all frequency bands in the right parietal area.
Conclusions: Areas of increased EEGfC topographically correspond to cortical areas that, based on relevant literature, are related to speech and attention deficit in BECTS children.

1. Introduction

Benign epilepsy of childhood with centro-temporal spikes (BECTS) is a well-known epilepsy syndrome. Typical BECTS patients have very rare focal seizures and do not show neurological abnormalities in the interictal state. Their EEG records show centro-temporal interictal epileptiform discharges. Prognosis is excellent, terminal remission occurs in all cases by the age of 16 years. Therefore, BECTS has been classified as idiopathic focal epilepsy [1,2]. The term "idiopathic" traditionally implies lack of "demonstrable anatomic lesions" [1]. However, this notion has not been valid any longer. Structural and functional abnormalities were described in typical BECTS children: bilaterally increased gray matter volume in the frontal lobes and insula [3], extensive cortical thinning in frontal, central, parietal and temporal areas [4]. Abnormal white matter was found in the frontal and temporal lobes [5]. Decreased functional MRI (fMRI) connectivity was demonstrated between Broca's area and the sensorimotor network [6]. Subtle cognitive and language difficulties that occur in 28–53 per cent of BECTS children [7] further suggest the presence of

Abbreviations: BECTS, benign childhood epilepsy with centro-temporal spikes; EEGfC, EEG functional connectivity; CSD, current source density; FD, false discovery rate; NC, normal (healthy) control; LORETA, Low Resolution Electromagnetic Tomography; LSC, LORETA Source Correlation; ROI, region of interest; SPN, statistical parametric network.

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http://dx.doi.org/10.1016/j.seizure.2016.01.001
1059-1311/