MAKING A MARK IN EPILEPSY CARE: CAN WE DO MORE?

Chair: Torbjörn Tomson (Sweden)

This symposium will address some important issues in the care of people with epilepsy. Combining literature and data with patient cases and real-world advice, the experienced faculty will challenge some of our current practices and discuss ways to improve and individualise care for our patients with epilepsy.

The transition from child to adult epilepsy care is a phase requiring special consideration. Our patients experience the difficult transition from childhood to adulthood while also being transferred from a comprehensive paediatric care system to an adult neurology system that has different resources, competencies, management strategies, and perspectives.

For many adolescents with epilepsy, poor adherence to medical advice and prescribed medication is the reality. Are we as physicians aware of how our patients really take their medications? Or to what extent medication non-adherence affects seizure control and outcomes? We will discuss strategies to uncover and improve poor medication adherence among our patients.

One factor that impacts adherence is drug load, and monotherapy has been considered the gold standard in epilepsy treatment for nearly 40 years. The AED landscape has evolved considerably, and since the introduction of new-generation AEDs like perampanel, which has a different mode of action from all other AEDs, it may be timely to re-evaluate our attitudes to polytherapy to strive for the best possible outcomes for our patients.

These fundamental issues of transition, adherence, and personalising polytherapy, will be discussed in formal lectures and illustrative case studies with clinical practice points and the opportunity for audience questions and panel discussion.

BUILDING BRIDGES: MANAGING THE TRANSITION FROM CHILD TO ADULT EPILEPSY CARE

Marina Nikanorova (Denmark)
**Satellite Symposia**

**Tuesday, 1st July 2014**  
**Satellite Symposium: Cyberonics**  
07:30–09:00, Hall K2

**ADVANCING PATIENT CARE THROUGH INNOVATIVE CLOSED LOOP NEUROSTIMULATION**

**Chair:** Paul Boon (Belgium)

**INTRODUCTION: REFRACTORY EPILEPSY – MORE THAN SEIZURES**  
*Paul Boon (Belgium)*

**REDUCING THE BURDEN OF REFRACTORY EPILEPSY**  
*Philippe Ryvlin (France)*

**SEIZURE DETECTION USING AUTONOMIC SIGNATURES OF EPILEPSY: CLOSING THE LOOP**  
*Shivkumar Sabesan (USA)*

**THE IMPACT AND POTENTIAL OF CLOSED LOOP NEUROSTIMULATION**  
*Kristl Vonck (Belgium)*

**DEBATE: ARE WE ADVANCING PATIENT CARE?**  
*Paul Boon (Belgium)*

**Tuesday, 1st July 2014**  
**Satellite Symposium: NeuroSigma, Inc**  
18:00–19:30, Hall K2

**TRIGEMINAL NERVE STIMULATION: NEUROMODULATION FOR THE 21ST CENTURY**

**Chair:** Christianne Heck (USA)

**INTRODUCTION TO TRIGEMINAL NERVE STIMULATION: BACKGROUND INFO, ANATOMY, BASIC SCIENCE**  
*Christianne Heck (USA)*

**ACUTE AND LONG TERM RESULTS IN US CLINICAL TRIALS FOR EPILEPSY AND DEPRESSION**  
*Christianne Heck (USA)*

**TNS AND CORTICAL EXCITABILITY: TRANSCRANIAL MAGNETIC STIMULATION STUDIES**  
*Adam Pawley (UK)*

**CLINICAL TRIAL RESULTS AT EUROPEAN EPILEPSY CENTRES**  
*Lina Nashef (UK)*  
*Jose M. Serratosa (Spain)*  
*Seán J. Slaght (UK)*

**Tuesday, 1st July 2014**  
**Satellite Symposium: ViroPharma**  
16:30–18:00, Hall A2

**MANAGEMENT OF PROLONGED, ACUTE, CONVULSIVE SEIZURES FOR CHILDREN IN THE COMMUNITY – FAR FROM PERFECT™?**

**Chair:** Lieven Lagae (Belgium)

**MANAGEMENT OF PROLONGED, ACUTE, CONVULSIVE SEIZURES IN THE COMMUNITY – WHAT HAVE WE LEARNT SO FAR?**  
*Lieven Lagae (Belgium)*

**UNDERSTANDING THE MANAGEMENT OF PROLONGED, ACUTE, CONVULSIVE SEIZURES – PERSPECTIVES FROM HCPS, CARERS AND PATIENTS**  
*Richard Appleton (UK)*

**THE PERFECT™ INITIATIVE – EMERGING DATA FROM EUROPEAN-WIDE SURVEY**  
*Federico Vigevano (Italy)*

**FUTURE MANAGEMENT OF CHILDREN WITH PROLONGED, ACUTE, CONVULSIVE SEIZURES IN THE COMMUNITY – DISCUSSION**  
*Richard Chin (UK) and Rima Nabbout (France)*

**SUMMARY AND CLOSE**  
*Lieven Lagae (Belgium)*

**Epilepsia, 55(Suppl. 2):1–3, 2014**  
doi: 10.1111/epi.12674
Wednesday, 2nd July 2014
Satellite Symposium: Elekta
16:30–18:00, Hall K2

WHAT CAN EPILEPTOLOGISTS EXPECT FROM MEG?

Chair: Hermann Stefan (Germany)

MEG IN THE PRESURGICAL EVALUATION OF EPILEPSY PATIENTS: PERSPECTIVE OF THE REFERRING CENTRE
Paul Boon (Belgium)

IMPLEMENTATION, ORGANIZATION, AND ROLE OF A MEG LABORATORY IN A LARGE EPILEPSY SURGERY CENTRE
Richard C. Burgess (USA)

FUTURE OF MEG IN TREATMENT OF EPILEPSIES
Stefan Rampp (Germany)
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METALLOME OF SCLEROTIC HIPPOCAMPUS IN PATIENTS WITH DRUG-RESISTANT MESIAL TEMPORAL LOBE EPILEPSY

Ristić AJ1, Sokić D1, Bašcarević V2, Spasić S1, Vojvodić N3, Savić S2, Raščević Š1, Kovacević M1, Savić D3, Spasojević D1
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Purpose: Altered hippocampal metallome is strongly implicated in the pathology of mesial temporal lobe epilepsy with hippocampal sclerosis (mTLE-HS). We aimed to determine sodium, potassium, calcium, magnesium, iron, copper, manganese, and zinc concentration in epileptic human hippocampi.

Method: Hippocampi of 24 drug-resistant mTLE-HS patients (age: 35.6 ± 9.4 years) that underwent anterior temporal lobe resection and amygdalectomy were obtained by autopsy from 13 controls (age: 40.5 ± 12.9 years) were analyzed using inductively coupled plasma optical emission spectrometry.

Results: Epileptic hippocampi showed significantly lower concentrations (µg/g of tissue) of copper (HS: 2.34 ± 0.12; control: C): 3.57 ± 0.33, p < 0.001), manganese (HS: 0.205 ± 0.030; C: 0.409 ± 0.064; p = 0.004), and potassium (HS: 2011 ± 59; C: 2322 ± 61; p < 0.001), and increased sodium level (HS: 1131 ± 22; C: 1040 ± 25; p = 0.010). Zinc concentration was slightly higher in HS (13.97 ± 1.51 µg/g) compared to controls (10.97 ± 1.03 µg/g), whereas iron, calcium, and magnesium levels did not differ.

Conclusion: Our results provide a relevant prerequisite for understanding the potential involvement of different metals in the pathology of HS, emphasizing general deregulation of metallome, copper and manganese deficiency, and the absence of iron accumulation.

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RETINAL NERVE FIBER LAYER THICKNESS: A POSSIBLE BIOMARKER OF DRUG RESISTANCE IN EPILEPSY

Balestrini S1,2, Bartman AP1, Clayton LM1, Chinthapalli KV1, Novy J3, Coppola A1, Wandschneider B1, Koepf MJ1, Stern W1, Acheson J1, Bell GS1, Sander JW1, Sisodiya SM1
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Purpose: Retinal nerve fiber layer thickness is related to the axonal anterior visual pathway and is considered a marker of overall white matter "integrity". Retinal nerve fiber layer thickness was previously assessed in a cohort of people with epilepsy and a history of vigabatrin exposure, showing significant thinning compared to healthy controls. We hypothesised that retinal nerve fiber layer changes would occur in people with chronic epilepsy, independently of previous vigabatrin treatment, related to clinical characteristics of epilepsy.

Method: Three hundred subjects with chronic epilepsy and 90 healthy controls were included. People with previous exposure to vigabatrin or known ocular disease were excluded from the analysis. Retinal nerve fiber layer imaging was performed using spectral-domain Optical Coherence Tomography.

Results: People with epilepsy had significantly lower average retinal nerve fiber layer thickness and lower thickness of each of the 90 degrees quadrants than healthy controls (p = 0.001, Wilcoxon rank-sum test). In a multivariate logistic regression model, drug resistance was the only significant predictor of abnormal retinal nerve fiber layer thinning (OR 2.09, CI 95% 1.09–4.01, p = 0.03). Duration of epilepsy and the presence of intellectual disability also showed a significant relationship with retinal nerve fiber layer thinning in a multivariate linear regression model (coefficients −0.16, p = 0.004 and −4.0, p = 0.044, respectively).

Conclusion: This suggests that drug-resistant epilepsy is associated with thinning of the retinal nerve fiber layer. As this is easily assessed by optical coherence tomography, retinal nerve fiber layer thickness is a candidate biomarker of drug resistance and, by extension, of epilepsy severity. Longitudinal studies are now needed. The underlying mechanisms are unknown and may be diverse.

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ACONITUM COCHLEARE WOROSCHIN-OIL ATTENUATES THE MOLECULAR MARKERS OF EPILEPTOGENESIS IN PENTYLENETETRAZOLE-INDUCED KINDLED MICE WITH SAFE TOXICITY PROFILE

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Purpose: Epilepsy is a chronic neurological disorder, characterized by recurrent seizures occurring as a result of synchronized discharges of neurons in brain. As 33% of patients develop resistance against therapy while others are not without side effects, therefore, need for better and safer drugs is crucial. Neurtrophic factors and Oxidative stress are emerging as mechanisms that may play an important role in the etiology of seizure-induced neuronal death. In the present study, Aconitum cochleare Woroschin-oil (ACR-oil) was tested for its ability (i) to suppress the convulsive and lethal effects of Pentylenetetrazole (PTZ) in kindled mice, (ii) to attenuate the PTZ-induced oxidative injury in the brain tissue and (iii) to modulate the gene expression BDNF and its receptor Trk-B when given as a pretreatment prior to each PTZ injection during kindling acquisition. Diazepam and valproic acid, major antiepileptic drugs, were also tested for comparison.

Methods: Once acute screening was done, all groups except for control group were kindled by injections of PTZ with an interval of 48 h (n = 12). In the 18th injection, all groups were sacrificed and the brain samples were collected and used for determination of oxidative stress parameters and targeted gene expressions by PCR.

Results: Our results suggest that ACR-oil treatment (100 mg/kg, 200 mg/kg) significantly inhibit, both acute and chronic PTZ induced seizures (p < 0.05). Toxicity studies demonstrate that the test oil is devoid of major toxic effects on suggested doses. Our test oil not only produced antiepileptic effect but also diminished the PTZ induced oxidative stress (p < 0.05, p < 0.001).