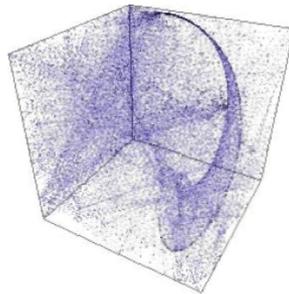


PRINCIPLES OF AUTONOMOUS NEURODYNAMICS 2017

Warsaw, Poland

July 10-11, 2017



SAND XIV

14th Annual Meeting of the

Society for Autonomous Neurodynamics (SAND)

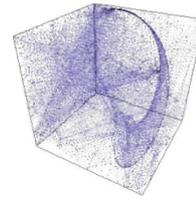
Location:

Faculty of Physics
University of Warsaw
5 Pasteur St., 02-093
Warsaw, Poland

http://www.fuw.edu.pl/~jarekz/SAND2017/SAND_2017.htm

SAND XIV - PRINCIPLES OF AUTONOMOUS NEURODYNAMICS 2017

Warsaw, Poland - July 10-11, 2017



DAY 1 - Monday, July 10
Room B1.44-46

Coffee and Registration

9:00 - 9:50

Piotr Suffczynski

Opening word

9:50 - 10:00

Session I

Chair: Piotr Suffczynski

Katarzyna Blinowska

Neurovascular coupling studied by means of EEG and functional Near Infrared Spectroscopy (fNIRS)

10:00 - 10:20

Marcin Szwed

Challenging the sensory division of labor in the brain. Lessons from the deafs' sense of rhythm and tactile braille reading in the sighted.

10:20 - 10:40

Maciej Kamiński

MVAR based functional connectivity analysis in biomedical data

10:40 - 11:00

Coffee break

11:00 - 11:20

Session II

Chair: Jarosław Żygierewicz

Maciej Łabęcki

Are the Steady State Visual Evoked Potentials real steady-state signals? Evidence from EEG experiments.

11:20 - 11:40

Ela Gajewska-Dendek

Mechanisms of SSVEP investigated with computational model

11:40 - 12:00

Piotr Suffczynski

On the origin of the harmonic frequencies in SSVEP signals

12:00 - 12:20

Group photo & Lunch

12:20 - 14:40

Session III

Chair: Berj Bardakjian

Piotr Durka

Brain-Computer Interfaces, Assistive Technologies and Disorders of Consciousness

14:40 - 15:00

Magda Zieleniewska

Sleep patterns in disorders of consciousness

15:00 - 15:20

Stiliyan Kalitzin

Multi-spectral optical flow technique for image sequence analysis.

15:20 - 15:40

Coffee break

15:40 - 16:00

Session IV

Chair: Maciej Łabęcki

Dominik Krzemiński

Temporal dynamics of ECoG signals during anesthesia

16:00 - 16:20

Daniel Wójcik

Source reconstruction from extracellular potentials, from single cells to the whole brains

16:20 - 16:40

Jarosław Żygierewicz

A note on the Blind Source Separation

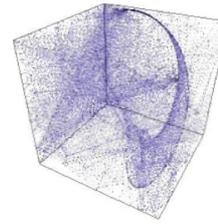
16:40 - 17:00

Dinner (Bazyliśzek at Old Town Square 1/3)
&
Night stroll along Vistula river

19:00 - till late

SAND XIV - PRINCIPLES OF AUTONOMOUS NEURODYNAMICS 2017

Warsaw, Poland - July 10-11, 2017



DAY 2 – Tuesday, July 11
Room B1.44-46

Session V

Chair: Stiliyan Kalitzin

Uzi Awret	Essential Differences, Symmetry, Noether's Theorem, and the Mind-Body Problem.	10:00 - 10:20
Elan Ohayon	The tile to be announced during the talk	10:20 - 10:40
Ernest Aleksy Bartnik	Toroidales algorithmicus: A simulated evolution (The what of what? said Pooh)	10:40 - 11:00
	Coffee break	11:00 - 11:20

Session VI

Chair: Katarzyna Blinowska

Peter Carlen	Neocortical potassium dynamics in vivo: gap junctional coupling and spreading depression	11:20 – 11:40
Władysław Średniawa	Mechanisms of novel seizure pattern investigated with computational model	11:40 – 12:00
Damiano Gentiletti	Investigating epileptic seizures generation and evolution with a realistic computational network model	12:00 - 12.20
	Lunch	12:20 - 14.20

Session VII

Chair: Peter Carlen

Berj Bardakjian	Coupled Oscillator model of hyperexcitable neuro-glial networks	14:20 – 14:40
George Petkov	From susceptibility to seizures: Separation of endophenotype and phenotype using dynamic network models	14:40 – 15:00
Ernie Pulil	Metabotropic glutamate receptors in thalamus activated by isovaline – A New Antiepileptic?	15:00 – 15:20
	Retreat announcements	15:20 - 15.40
	Departure for SAND Scientific Retreat	18:00

Abstracts

Uziel Awret

Akko, Israel

Title: Essential Differences, Symmetry, Noether's Theorem, and the Mind-Body Problem.

Abstract: Noether's theorem relates conservation laws to symmetries of space, time and internal symmetries. The theorem furnishes profound connections between the fundamental constituents of reality and symmetry by combining the calculus of variations and quantum mechanics with Lie groups. More importantly for our purpose the unification program based on this theorem attributes the differences between elementary constituents of matter to "broken symmetries" prior to which the differences were indiscernible. Electrons and neutrinos seem very different as do photons and Z particles but electroweak unification shows that they are related because the present differences between them resulted from prior symmetry breaking.

If consciousness is physical we need to explain why it appears to be so different than standard elementary physical constituents. Unlike approaches that attempt to explain this difference by explaining away our anti-physicalist intuitions conceptually/psychologically, I will assume that consciousness is composed of some strange state of matter and ask whether symmetry principles in general and Noether's theorem in particular can help us get a handle on that difference. Failure to do so would strengthen the conclusion that even if consciousness is a "broadly physical" constituent of reality, it is still different from other physical constituents.

I will try to apply Noether's theorem to:

- Categorical Russellian monism in which the bearer of the microphysical properties can be physical, phenomenal, or any combination of the two that does not influence the physical behavior of the system. Here Noether's theorem may provide us with novel conserved quantities, perhaps even the associated "particle" mediating the interaction between the physical and the phenomenal.
- Is symmetry breaking immune to Chalmers' argument about structure and dynamics?
- Time-dependent spatial topology exhibiting novel continuous symmetry with timelike curves and temporally non-orientable manifolds as examples of hidden temporal symmetries with philosophically relevant conserved quantities.
- Restoration of hidden temporal symmetry and the "physics of consciousness": chronos, kiros and projective geometry.

Berj L. Bardakjian, Vasily Grigorovsky and Firas Farah.

University of Toronto

Title: Coupled Oscillator model of hyperexcitable neuro-glial networks

Abstract: This study deals with a 16 coupled oscillator model of neuro-glial network which incorporates pyramidal, interneuron, micro glia and astrocyte cell types. Local field potentials and individual cellular type outputs are obtained from this network and the contribution of each cell type to hyper excitability of the network is investigated. With those results, changes in excitability, average spontaneous electrical discharge (SED) duration and cross frequency coupling (CFC) features are measured. We found that (i) an increase in the SED duration was exponentially related to the level of excitability of the system, (ii) short duration SEDs showed CFC between high frequency oscillations and theta oscillations (4-8 Hz), but in longer duration SEDs the

low frequency changed to the delta range (1-4 Hz). The model was validated as it displayed CFC features similar to those reported by our team in MeCP-2 deficient rodent models and patients with epilepsy. This study sheds light on the importance of glial factors in hyper excitability of neuro-glial networks of the brain, as contributors to the genesis of seizure like activities in the brain.

Ernest Aleksy Bartnik

University of Warsaw

Title: Toroidales algorithmicus: A simulated evolution (The what of what? said Pooh)

Abstract: I present a computer simulation of a population of simple virtual organisms living in the Cyber Ocean. They are subjected to Darwinian evolution, which exhibits a remarkable feature: punctuated equilibria.

Katarzyna J, Blinowska

Institute of Biocybernetics and Biomedical Engineering of Polish Academy of Sciences & University of Warsaw

Title: Neurovascular coupling studied by means of EEG and functional Near Infrared Spectroscopy (fNIRS)

Abstract: Nowadays large bulk of evidence concerning topographical and functional aspects of neural activity comes from the neuroimaging methods such as fMRI and fNIRS. However, the fundamental relation between cerebral changes of hemodynamics and brain activity is hardly known. Therefore the integration of electrophysiological and hemodynamic signals (fNIRS, BOLD) with the aim of understanding of the neurovascular coupling is in the center of attention of the scientific community. The information about neurovascular coupling may be derived from the multimodal measurements of hemodynamic and electric activity of the brain by means of fNIRS combined with EEG.

The relationship between brain rhythmic activity and hemodynamic response was studied by means of simultaneous measurement of electroencephalogram (EEG) and functional near-infrared spectroscopy (fNIRS) during motor task (self-paced right finger movements) for 10 subjects. EEG was recorded by a 32-electrode system and the hemodynamic response by means of 8 optodes placed over sensorimotor cortex on both hemispheres. During the task an increase of oxyhemoglobine (HbO) was accompanied by a decrease of deoxyhemoglobine (HbR) concentration and a decrease of amplitudes (desynchronization) of alpha (8-13 Hz) and beta (13-30 Hz) EEG rhythms. These phenomena were prominent in the hemisphere contralateral to the moving finger. Highly significant ($p < 0.001$) negative Pearson correlations were found between HbO and alpha ($r^2 = -0.69$) and HbO and beta ($r^2 = -0.54$) rhythms envelopes and positive correlations $r^2 = 0.5$ between these rhythms and HbR were found. Usually an increase of HbO is connected with an increase of brain activity. Our findings may be explained by the fact that unlike EEG, which shows synchronised brain activity, fNIRS detects brain activity requiring increased metabolic rate independently of the neural synchronisation and thus might be sensitive to less synchronized high frequency gamma rhythms.

Ela Gajewska-Dendek

University of Warsaw

Title: Mechanisms of Steady State Evoked Potentials investigated with computational model

Abstract: Steady State Evoked Potentials (SSEP) are emerging in EEG signal in response to periodically changing stimulus. Their frequencies correspond to stimulus frequency, its harmonics and subharmonics. The SSEP can be observed in visual, auditory and somatosensory modalities. The aim of this work is to investigate the mechanisms of SSEP generation with a computational model.

We have constructed a model of neuronal network comprising single compartment excitatory and inhibitory cells with extended Hodgkin-Huxley dynamics. The network consists of multiple domains representing cortical columns. Connectivity is based on anatomical data from cat's primary visual cortex. The modelled neurons receive three kinds of Poisson inputs, which represent: (i) background sensory input from the thalamus, (ii) background top-down input from higher order cortical regions and (iii) periodic stimulus from the thalamus to all of the domains, representing sensory stimulation. The sensory stimulus was modelled by Poisson process, with mean rate modulated periodically in time by square or sinusoidal function at frequency in 7 to 50 Hz range. The EEG signal was modelled as a sum of synaptic currents of pyramidal neurons. We compare the simulation data with experimental EEG recordings obtained in somatosensory cortex area during vibrotactile stimulation.

The spectra of modeled SSEP signals exhibit fundamental and higher harmonic frequencies similarly to experimental observations. The first harmonic is stronger than fundamental response for the driving frequencies smaller than network's natural frequency (15 – 20 Hz) as in the case of square stimulation in the EEG experiment. The neurons firing rates are approximately constant with stimulus frequency and the network oscillation emerges from irregular and sparse firing of individual neurons but in phase with the population rhythm.

The modeling results suggest that the emergence of oscillatory synchrony patterns is mediated by inhibitory interneurons driven by periodic excitation. The observed SSEP oscillations are caused by firing-rate synchrony.

Piotr Durka, Anna Chabuda, Anna Duszyk, Magdalena Zieleniewska, Marian Dvgialo, Marcin Pietrzak and Piotr Rózański

University of Warsaw

Title: Brain-Computer Interfaces, Assistive Technologies and Disorders of Consciousness

Abstract: Starting from the definition of a brain-computer interface (BCI) we will briefly:

1. recall the principles of operation of EEG-based BCIs,
2. summarize the state of the art in the field of BCI,
3. discuss (the limits of) applications of BCI in the field of Assisstive Technologies, explaining why prof. Stephen Hawking does not use a BCI.

Finally, we will mention a new field where basically the same technology finds an important application, which is assessment of the state of the patients suffering severe disorders of consciousness, commonly called "coma". We will outline the recent project undertaken in cooperation with the Warsaw's Alarm Clock Clinic -- the first model hospital for children with severe brain damage.

Damiano Gentiletti¹, Piotr Suffczynski¹, Vadym Gnatkovski², Marco De Curtis²

¹ Department of Experimental Physics, University of Warsaw, Warsaw, 02-093

² Istituto Neurologico Carlo Besta, Milan, 20133

Title: Investigating epileptic seizures generation and evolution with a realistic computational network model

Abstract: Epilepsy and seizures are traditionally associated with an imbalance between excitatory and inhibitory forces in the brain. This classic view is challenged by the *in vitro* isolated guinea pig brain model of focal seizures. Based on experimental data recorded from the entorhinal cortex (EC), it appears that inhibitory neurons are active at the very beginning of a focal seizure, whereas excitatory cells are quiescent. This is accompanied by an increase of the extracellular potassium concentration. Within a few seconds from seizure onset, the principal cells display excessive firing associated with the seizure discharge. Neuronal firing of principal neurons subsequently decreases, and further evolves into rhythmic bursting activity that terminates the seizure.

In order to gain a deeper understanding of the link between ionic dynamics and neuronal activity during seizures we developed a realistic computational model of the entorhinal cortex circuit. The model consists of a small neuronal network made up of five hippocampal cells – an inhibitory interneuron and four pyramidal cells – each one surrounded by an extracellular space. Each extracellular environment incorporates dynamics of Na^+ , K^+ , Cl^- and Ca^{2+} ions, the glial buffering system and diffusion mechanisms. Different extracellular spaces communicate with each other by diffusive exchange of K^+ and Na^+ ions. Intra- and extracellular dynamics of volume changes is also implemented.

Simulations performed with our *in silico* model show that ion concentration changes have significant impact on the network behaviour and determine the different phases of a focal seizure. In particular, the model is able to reproduce the membrane potential and potassium concentration traces recorded experimentally, and the pathological sequence taking place in the pyramidal cells: quiescent period – seizure onset – excessive pyramidal firing – late bursting phase. Our simulations confirm the experimentally-driven hypothesis that strong discharge of inhibitory interneurons may result in long lasting accumulation of extracellular K^+ , which in turn is responsible for seizure progression in principal cells.

Our study also shows that a reduced model with fixed ionic concentrations is not able to reproduce the seizure patterns observed experimentally, pointing to the importance of the role played by non-synaptic mechanisms in modeling focal epileptic activity.

Stiliyan Kalitzin^{1,2}, Evelien Geertsema¹

¹ Stichting Epilepsie Instellingen Nederland (SEIN), Heemstede, The Netherlands

² Image Sciences Institute, University Medical Center Utrecht, The Netherlands

Title: Multi-spectral optical flow technique for image sequence analysis.

Abstract: Development of **autonomous** remote sensing systems, based on video camera's for example, can be advantageous in certain situations for home and institutional use as safety and security assets. One application currently investigated by our group is detection and real-time alerting of motor epileptic seizures, fall detection and situation awareness. Video modality offers the advantage of assessing the scene as a whole as opposed to limited information from dedicated attached sensors. It also avoids problems with patient stigmatization and burden from extra devices. Major challenge in video processing is the detection of movements and the

extraction of the classifying information from those movements. Existing optic flow algorithms are based on intensity changes of the images and do not use the full potential of complimentary imaging modalities. We have developed a modular concept to use synergistically multiple channels of image sequences, in the most common case consisting of the three spectral RGB components. This way we can decrease and eventually remove the degeneracy of the inverse optical flow solution. Validation of the reconstructive algorithm is proposed using natural and synthetic images subjected to pre-defined transformation patterns.

Dominik Krzemiński

Title: Temporal Dynamics of ECoG signals during anesthesia

Abstract: Consciousness is identified with binding and integration of information, both on the phenomenological and neuronal level. Whereas the neuronal basis of features' binding and multi-sensory integration has been extensively investigated, not much is known about the mechanisms of temporal integration and their role in maintaining consciousness. In the present study spontaneous brain activity was recorded in different states of consciousness and analyzed in terms of long-range temporal correlations (LRTCs), which indicate that signals possess long "memory" and are modulated across multiple time-scales. We hypothesized that loss of consciousness during general anesthesia will be related to weaker LRTCs in brain activity. Resting-state electrocorticography (ECoG) was recorded from four macaque monkeys during wakefulness and general anesthesia. We estimated amplitude envelopes of brain oscillations and used Detrended Fluctuation Analysis to estimate LRTCs of amplitude modulations. Our results complement and extend previous studies, which demonstrated LRTCs in non-invasive M/EEG recordings. Moreover, we revealed that during consciousness brain activity is modulated across a wide range of time-scales, which is indicative of a long temporal memory and might reflect the process of temporal integration.

George Petkov

College of Engineering, Mathematics & Physical Sciences, University of Exeter, Exeter, EX4 4QF, UK; Wellcome Trust Centre for Biomedical Modelling and Analysis, University of Exeter, EX2 4DW, UK.

Title: From susceptibility to seizures: Separation of endophenotype and phenotype using dynamic network models.

Abstract: In the present paper, we use a mathematical representation of large-scale brain networks to explore the critical features that define the presence of seizures (the phenotype) versus those that contribute to an increased risk (the endophenotype). We introduce a methodological framework that reveals the topological and spectral properties critical to each. Our main assumption is that all information is encoded in the network properties that constrain the dynamics that emerge from them. We introduce two mathematical models to explore how the emergent likelihood of seizures (which we term Brain Network Ictogenicity (BNI)) is influenced by properties of the network.

We test the resulted methodological framework over two different EEG resting state datasets: (40 controls/ 35 people with epilepsy/ 42 first-degree relatives) and (38 controls/ 21 drug-naive people with epilepsy). A functional network for every subject was reconstructed from EEG data using maximum correlation synchrony model (Schmidt et al. 2014). Statistical analysis of the results revealed that using model-based dynamic descriptors; one may obtain a "seizure based" data classification

showing statistically significant differences between controls and relatives on the one hand because they do not get seizures and people with epilepsy on the other.

Ernie Puil and Khalid Asseri

Department of Anesthesiology, Pharmacology and Therapeutics, Faculty of Medicine, University of British Columbia, Vancouver B.C., Canada

Title: Metabotropic glutamate receptors in thalamus activated by isovaline – A New Antiepileptic?

Abstract: Metabotropic glutamate receptors (mGluRs) are therapeutic drug targets for antinociception and brain disorders such as epilepsy. Previously we found that a non-proteinogenic amino acid analgesic, isovaline, inhibits thalamic neurons by activating GABA_B receptors. Isovaline may additionally activate group II mGluRs since these receptor subtypes belong to the same family C of G-protein-coupled receptors. Activation of either receptor inhibits neuronal firing by increasing postsynaptic K⁺-conductance and decreasing Ca²⁺-dependent transmitter release. Since group II mGluRs are prevalent on nerve terminals in thalamus, we postulated that isovaline would activate group II mGluRs, decreasing transmitter release. Whole-cell patch clamp recordings were performed on thalamocortical neurons of ventrobasal nuclei in rat brain slices. Application of a group II agonist (LY354740) had no effects on postsynaptic membrane properties which were blocked with intracellular Cs⁺ in subsequent experiments. The medial lemniscus was stimulated electrically to evoke inhibitory and excitatory postsynaptic currents (IPSCs, EPSCs). We identified the neurotransmitters by applying receptor antagonists. LY354740 reduced frequency of spontaneous IPSCs and miniature IPSCs (during TTX application). Application of isovaline or LY354740 depressed peak amplitudes of GABA_Aergic IPSCs and glutamatergic EPSCs. Co-application of isovaline or LY354740 with a selective antagonist of group II mGluRs prevented the depressions of IPSCs and EPSCs. The studies demonstrate that activation of group II mGluRs by isovaline decreases GABA and glutamate release evoked by medial lemniscal stimulation. Presynaptic group II auto- and heteroreceptors may regulate transmitter release on thalamic neurons, providing target

Piotr Suffczynski

University of Warsaw

Title: On the origin of the harmonic frequencies in SSVEP signals

Abstract: In this study, we investigated the origin of the harmonic and subharmonic components of steady state visual evoked potentials (SSVEP), which are not well understood. We applied both sine and square wave visual stimulation at 5 and 15 Hz to human subjects and analyzed the properties of the fundamental responses and harmonically related components. In order to interpret the results, we used the well-established neural mass model that consists of interacting populations of excitatory and inhibitory cortical neurons. In our study, this model provided a simple explanation for the origin of SSVEP spectra, and showed that their harmonic and subharmonic components are a natural consequence of the nonlinear properties of neuronal populations and the resonant properties of the modeled network. The model also predicted multiples of subharmonic responses, which were subsequently confirmed using experimental data.

Marcin Szwed

Jagiellonian University, Cracow

Title: Challenging the sensory division of labor in the brain. Lessons from the deaf's sense of rhythm and tactile braille reading in the sighted.

Abstract: Introduction

It is well established that the brain is capable of large-scale reorganization following sensory deprivation, injury or intensive training (1-3). What remains unclear is what organizational principles guide this process. In the blind, many visual regions preserve their task specificity despite being recruited for different sensory input; ventral visual areas, for example, become engaged in auditory and tactile object-recognition tasks (4, 5). It remains open whether task-specific reorganization is unique to the visual cortex, or alternatively, whether it is a general principle applying to other cortical areas.

Several areas in the auditory cortex are known to be recruited for visual and tactile input in the deaf (6-8). Although non-human data suggest that this reorganization might be task specific (6, 7), human evidence has been lacking. Here we enrolled deaf and hearing adults into an fMRI experiment, during which they discriminated between rhythms, i.e., temporally complex sequences of stimuli. In hearing individuals, rhythm processing is performed mostly in the auditory domain (9). Thus, if task-specific reorganization applies to the human auditory cortex, performing this function visually should recruit the auditory cortex in the deaf. Moreover, auditory areas activated by visual rhythm processing in the deaf should be also particularly engaged in auditory rhythm processing in the hearing.

Relative to visual control, visual rhythms enhanced bilateral activations in the auditory cortex of deaf subjects but no activations in the auditory cortex of hearing subjects. Anatomically-guided ROI analysis showed that in deaf subjects activations for visual rhythms were confined to high-level auditory areas. Contrasted with auditory control, auditory rhythms in hearing subjects enhanced activations similar to those observed for visual rhythms in deaf subjects. Moreover, activations for visual rhythms in the deaf and auditory rhythms in the hearing peaked in the same auditory area, namely the posterior-lateral part of the high-level auditory cortex.

Conclusions

In our study, the same auditory areas were recruited for visual rhythm processing in deaf humans and auditory rhythm processing in hearing humans. This directly confirms the prediction of task-specific reorganization hypothesis. Task-specific, sensory-independent reorganization has so far been documented in the human visual cortex and in the cat auditory cortex. Our study shows that similar rules might guide plasticity in the human auditory cortex. Thus, switching the sensory but not the functional role of recruited areas might be a general principle that guides large-scale reorganization of the brain.

References

1. Rauschecker, J.,P. (1995), 'Compensatory plasticity and sensory substitution in the cerebral cortex', *Trends in Neurosciences*, vol. 18, no. 1, pp. 36–43.
2. Siuda-Krzywicka, K. (2016), 'Massive cortical reorganization in sighted Braille readers', *eLife*, 5:e10762.
3. Sur, M. (1988), 'Experimentally induced visual projections into auditory thalamus and cortex', *Science*, vol. 242, no. 4884, pp. 1437-1441.
4. Heimler, B. (2015), 'Origins of task-specific sensory-independent organization in the visual and auditory brain: neuroscience evidence, open questions and clinical implications', *Current Opinion in Neurobiology*, vol. 35, pp. 169–177.
5. Renier, L. (2014), 'Cortical plasticity and preserved function in early blindness', *Neuroscience & Biobehavioral Reviews*, vol. 41, pp. 53-63.
6. Lomber, S.G. (2010), 'Cross-modal plasticity in specific auditory cortices underlies

- visual compensations in the deaf', *Nature Neuroscience*, vol. 13, no. 11, pp. 1421–1427.
7. Meredith, M.A. (2011), 'Crossmodal reorganization in the early deaf switches sensory, but not behavioral roles of auditory cortex', *Proceedings of the National Academy of Sciences*, vol. 108, no. 21, pp. 8856–8861.
 8. Pavani, F. (2012), 'Cross-modal plasticity as a consequence of sensory loss: insights from blindness and deafness', *The New Handbook of Multisensory Processes*, pp. 737–759.
 9. Glenberg, A.M. (1989), 'Modality effects in the coding reproduction of rhythms', *Memory & Cognition*, vol. 17, no. 4, pp. 373–383.
 10. Pascual-Leone, A. (2001), 'The metamodal organization of the brain', *Progress in Brain Research*, vol. 134, pp. 427–445.

Władysław Średniawa

University of Warsaw

Title: Mechanisms of novel seizure pattern investigated with computational model

Abstract: Epilepsy progression can be caused by diverse ethologies and still basic pathogenic mechanisms remain unknown. Recent experimental and theoretical studies identify significant impact of the extracellular potassium ions concentration on initiating and spreading seizure discharges. Recent EEG and ECoG studies detected a novel type of focal seizure pattern in a sub-population of patients. Similar pattern has been also recorded in the isolated guinea pig brain with the potassium channel blocker 4-aminopyridine. This pattern is region specific and occurs only in cortical layers characterized by unmyelinated axons. To check the hypothesis that reduced glial potassium clearance in unmyelinated axons may play a role in the generation of the observed seizure pattern we developed a computational model. The results of biophysical modelling reproduced the main stages of the seizure discharges and confirmed the proposed pathological mechanism.

Iliya Weisspapir, Azin Amini, Paolo Bazzigaluppi, Peter L. Carlen

Krembil Research Institute, UHN and University of Toronto, Canada

Title: Neocortical potassium dynamics in vivo: gap junctional coupling and spreading depression

Abstract: Neocortical spatiotemporal potassium dynamics and buffering are key factors in the spread of focal seizure activity and spreading depression (SD). We demonstrate that inter-astrocytic gap junctional coupling plays a key and powerful role in enabling the spatial buffering of focally raised potassium. Initially we demonstrated that blocking gap junctional communication between astrocytes, by applying the Gap27 peptide, markedly increased extracellular potassium without causing seizures in the mouse neocortex. To elucidate the role of $[K^+]_e$ on seizure generation without the confounding effect of gap junctional blockade, we injected 50 mM $[K^+]_o$ solution into the parenchyma. The local increase in $[K^+]_o$ (12.1 ± 2.3 mM, $n = 2$) was transient and failed to generate persistently raised levels of $[K^+]_o$, demonstrating powerful local spatial $[K^+]_o$ buffering mechanisms. We then applied solutions of increasing $[K^+]_o$ onto the exposed dura which did generate steady levels of raised $[K^+]_e$, but seizures were not elicited. Focal metabolic inhibition raises $[K^+]_e$. We focally applied the metabolic inhibitor, DNP, to the mouse neocortex in vivo, and noted a marked DC shift occurring initially remotely from the focal point of DNP application, followed later by a DC shift at the focal point of DNP application. Similarly when hypoglycemic seizures were

generated in thick hippocampal slices, usually seizure activity originated in the CA3 region, whereas a DC shift suggesting spreading depression, originated in the CA1 area. Hence we suggest that local $[K^+]_o$ spatial buffering is very powerful such that higher levels of $[K^+]_o$ can occur peripherally from the initial source of raised $[K^+]_o$ generation, resulting in the origination of SD phenomenon remote from the source site, particularly in the presence of metabolic inhibition.

Supported by CIHR and the OBI

Daniel Wójcik

Nencki Institute of Experimental Biology

Title: Source reconstruction from extracellular potentials, from single cells to the whole brains

Abstract: Extracellular recordings of electric potential remain a popular tool for investigations of brain activity on all scales in animals and humans, from single cells (spikes) to systems studied with depth electrodes (LFP, SEEG), subdural recordings (ECoG), and on the scalp (EEG). They are relatively easy to record but difficult to interpret: since electric field is long range one can observe neural activity several millimeters from its source. As a consequence, every recording reflects activity of many cells, populations and regions, depending on which level we focus. One way to overcome this problem is to reconstruct the distribution of current sources (CSD) underlying the measurement. We recently proposed a kernel-based method of CSD estimation from multiple extracellular recordings from arbitrarily placed probes (i.e. not necessarily on a grid) which we called kernel Current Source Density method (kCSD). In my presentation, I will present this method and explain why it works. I will also show two recent developments, skCSD (single cell kCSD) and kESI (kernel Electrophysiological Source Imaging). skCSD assumes that we know which part of the recorded signal comes from a given cell and we have access to the morphology of the cell. This could be achieved by patching a cell, driving it externally while recording the potential on a multielectrode array, injecting a dye, and reconstructing the morphology. In this case we know that the sources must be located on the cell and this information can be successfully used in source estimation. In kESI we consider simultaneous recordings with subdural ECoG (strip and grid electrodes) and with depth electrodes (SEEG). Such recordings are taken on some epileptic patients prepared for surgical removal of epileptogenic zone. When MR scan of the patient head is taken and the positions of the electrodes are known as well as the brain's shape, the idea of kCSD can be applied to constrain the possible distribution of sources facilitating localization of the foci.

Magda Zieleniewska, Krzysztof Piwoński

University of Warsaw

Title: Sleep patterns in disorders of consciousness

Abstract: Previous research on disorders of consciousness (DOC) phenomenon indicated significant changes in circadian activity and sleep architecture that correlated with a behavioral diagnosis of patient's consciousness level. The aim of our study was to identify potential quantitative indices of polysomnographic recordings characterizing the process of regaining consciousness of pediatric patients with DOC. In the presentation the automatic construction of EEG sleep profiles based on the matching pursuit algorithm will be shown. This methodology provides a reliable parametrization of sleep microstructures and allows for monitoring changes in sleep patterns during rehabilitation process.

Jarosław Żygierewicz
University of Warsaw

Title: A note on the Blind Source Separation

Abstract: EEG signals are a linear mixture of the activity of the underlying cortical sources. The knowledge of the activity of the sources is of interest in many applications. I will focus on the family of techniques known as Blind Source Separation. I will show the principals, the usefulness of these techniques in the context of construction of brain-computer interfaces, and their limitations.