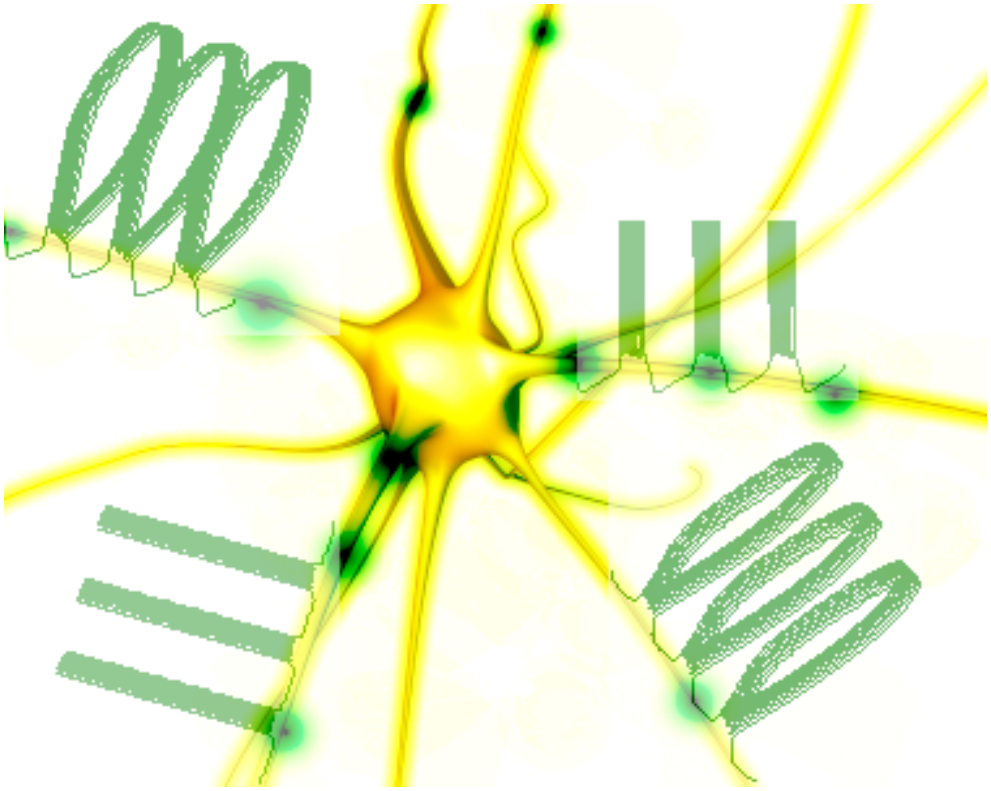
Georgia State University

2010

Dynamics of Bursting Activity of neurons

April 16- April 17



Center for BEHAVIORAL NEUROSCIENCE

Department of Physics and Astronomy

NEUROSCIENCE INSTITUTE

Sponsored by
The Center for Behavioral Neuroscience,
The Neuroscience Institute and
The Department of Physics and Astronomy,
GSU, Atlanta, GA

Dynamics of Bursting Activity of Neurons

Place: Brown Room on the 18th floor of The Commerce Club, 34 Broad St, Atlanta

Sponsored by The Center for Behavioral Neuroscience, The Neuroscience Institute and The Dept. of Physics and Astronomy,

Georgia State University

April 16

8:00 - 8:45 **Registration and Breakfast**
8:45 – 8:50 **Gennady Cymbalyuk:** ***Opening Remarks***
8:50 - 9:00 **Walter Wilczynski, Director of the Neuroscience Institute, GSU:** ***Welcoming Address***

Session 1 Chair: Andrey Shilnikov

9:00 - 9:45 **Jeff Smith:** ***Multiple modes and mechanisms of rhythmic burst pattern generation in brainstem respiratory networks***
9:45 - 10:15 **Illya Rybak:** ***Late-Expiratory Oscillations in RTN/pFRG: Emergence and Coupling with Respiratory CPG***
10:15 - 10:45 **Rhonda Dzakpasu:** ***Modulating the ratio between excitatory and inhibitory neurons in in vitro neural networks***
10:45 - 11:05 **Coffee Break**

Session 2 Chair: Gennady Cymbalyuk

11:05 – 11:50 **Nino Ramirez:** ***Neuromodulation and bursting gone mad: Insights gained from studying neurological disorders***
11: 50 - 12:20 **Andrew Tryba:** ***TRP-ing over your next breath and enhancing neocortical oscillations***
12:20 – 13:20 **Lunch**

Session 3 Chair: Robert Clewley

13:20 – 13:50 **Dieter Jaeger:** ***Pathological bursting in basal ganglia circuits in Parkinson's disease***
13:50 – 14:20 **Ari Berkowitz:** ***Multifunctional and specialized spinal interneurons for several kinds of limb movements***
14:20 – 14:50 **Donald Edwards:** ***Neuromechanical analysis of posture and locomotion in crayfish***
14:50 – 15:10 **Coffee Break**

Session 4 Chair: Mukesh Dhamala

15:10 – 15:40 **Joel Tabak:** ***The unusual bursting pattern produced by pituitary cells***
15:40 – 16:10 **Robert Butera:** ***Voltage-dependent and Voltage-independent pacemakers in the pre-Botzinger Complex: An Integrative Model***
16:10 – 16:30 **Andrey Shilnikov:** ***Polyrhythms of bursting patterns in deterministic models for central pattern generators***
16:30 – 16:45 **Coffee Break**

Session 5 Chair: Peter Rowat

16:45 – 17:15 **Michael Sorensen:** ***Accelerating simulations of bursting neurons with simEngine***
17:15 – 17:45 **David Mogul:** ***Multisite phase synchrony during chemically-induced seizures in rat brain***
17:45 – 18:05 **Igor Belykh:** ***Inhibitory synchronization in bursting networks***
18:05 - 20:00 **Reception-Dinner**
19:00 - 21:30 **Poster session**

Dynamics of Bursting Activity of Neurons

Place: Brown Room on the 18th floor of The Commerce Club, 34 Broad St, Atlanta

Sponsored by The Center for Behavioral Neuroscience, The Neuroscience Institute and The Dept. of Physics and Astronomy, Georgia State University

April 17

8:00 - 8:55 **Breakfast**

8:55 - 9:00 **Gennady Cymbalyuk:** ***Opening remarks***

Session 6 Chair: Sonya Bahar

9:00 - 9:45 **Roger Traub:** ***Some normal and abnormal collective phenomena in cortical circuits amongst bursting neurons***

9:45 - 10:15 **Daniel Wagenaar:** ***Bursting with desire: a two-timescale rhythm for mate exploration in the medicinal leech***

10:15 - 10:45 **Ronald Calabrese:** ***Given biological variation, how should we model small rhythmic networks?***

10:45 - 11:05 Coffee Break

Session 7 Chair: Ronald Calabrese

11:05 – 11:35 **Ayako Yamaguchi:** ***NMDAR-dependent control of call duration in Xenopus laevis***

11:35 - 12:05 **Paul Katz:** ***Biological Evolution of central pattern generators***

12:05 – 13:05 Lunch

Session 8 Chair: Vladimir Bondarenko

13:05 – 13:35 **Maxim Volgushev:** ***UP and DOWN states in neocortical neurons during slow wave oscillations***

13:35 – 14:05 **Maxim Bazhenov:** ***Network bistability mediates spontaneous transitions between normal and pathological brain states***

14:05 – 14:35 **Ernest Barreto:** ***Ion concentrations and seizure dynamics***

14:35 – 14:55 Coffee Break

Session 9 Chair: Igor Belykh

14:55 – 15:25 **Sonya Bahar:** ***Synchronization of Neural Activity during Focal Neocortical Seizures***

15:25 – 15:55 **Robert Gross:** ***'Filling-In-The-Blanks': Therapeutic Stimulation of the Nervous System for Disorders Involving Abnormal Burst Firing***

15:55 – 16:25 **Leonid Bunimovich:** ***Isospectral reduction of networks and spectral networks' equivalence***

16:25 – 16:40 Coffee Break

Session 10 Chair: Maxim Bazhenov

16:40 – 17:10 **Arnold Mandell:** ***Strudels: Intermittent MEG Sensor Field Correlates of the Inattentive Mind***

17:10 – 17:30 **Mukesh Dhamala:** ***Synchronization of Time-Delayed Coupled Bursting Neurons***

17:30 – 17:50 **Robert Clewley:** ***Reducing the fine structure of bursting dynamics***

17:50 – 18:10 **Gennady Cymbalyuk:** ***Co-existence of epileptiform bursting and silent regimes in neuronal dynamics***

18:10 – 19:00 Round table

Poster Session

P1	**William Barnett**, Martin Anquez, Gennady Cymbalyuk *Pattern scaling in neuronal dynamics*
P2	**Vladimir E. Bondarenko** and Andrey L. Shilnikov *Autonomous spiking and bursting in a mouse ventricular cell model*
P3	**Mirza Dobric** and Robert Clewley *Multi-modal optimization techniques for biophysical neuron models.*
P4	**Anca Doloc-Mihu** and Ronald Calabrese *Analyzing how varying neuronal parameters influence network activity using a database of computational models of a half-center oscillator*
P5	**Gregory Filatov** and Maxim Bazhenov *Role of Extracellular K^+ Dynamics in Network Synchronization.*
P6	**Sajiya Jalil**, Andrey Shilnikov, Igor Belykh *Synchronization in a bursting half-center oscillator with slow-to-fast reciprocal inhibition.*
P7	**Alexander N. Klishko** and Boris I. Prilutsky *Firing rates of cat muscle Ia, Ib, II and paw cutaneous afferents during walking computed using a musculoskeletal hindlimb model*
P8	**Alexey Kuznetsov** *Mechanisms of frequency control in the midbrain dopaminergic neuron.*
P9	**Giri P Krishnan** and Maxim Bazhenov *Modulation of bursting and tonic activity during seizure by ion concentrations explains spontaneous seizure termination.*
P10	**Tatiana Malaschenko** and Gennady Cymbalyuk *Good currents playing bad: propensity to bi-stability in neuronal dynamics*
P11	**Peter Rowat** *"Distribution of Burst Length and Inter-Burst-Interval of the stochastic Morris-Lecar Neuron"*
P12	**Wondimu Teka** *From Plateau to Pseudo-Plateau Bursting: Making the Transition.*
P13	**Wafa Soofi** *Covarying ionic conductances to emulate phase maintenance in stomatogastric neurons*
P14	**David Qian,** Gennady Cymbalyuk and Bill Walthall *Deviations from the straight and narrow: C. elegans uncoordinated mutants that Circle.*
P15	**Yaroslav I. Molkov,** Jonathan E. Rubin, Bartholomew J. Bacak, Natalia A. Shevtsova, Jeffrey C. Smith and Ilya A. Rybak *Modeling State-Dependent Interactions between BötC/pre-BötC and RTN/pFRG oscillations*
P16	**Jeremy Wojcik** and Andrey Shilnikov *Poincare Mappings for Models of Elliptic Bursters*
P17	**Xiaoli Zhang,** Junda Su, Ningren Cui, Hongyu Gai, Zhongying Wu and Chun Jiang *The breathing disorders of a mouse model of Rett syndrome are linked to defects in central CO2 chemosensitivity of locus coeruleus neurons*
P18	**Yang Yang,** Weiwei Shi, Ningren Cui, Xianfeng Chen, T. Trower, and Chun Jiang *Reactive oxygen species (ROS) inhibit KATP channel via S-glutathionylation.*
P19	**Cengiz Gunay,** Fred Sieling, Logesh Dharmar and Astrid Prinz *Characterizing change in activity patterns in a Drosophila motoneuron with different sodium channel splice variants.*

KeyNote Speakers

Jeffrey C. Smith

Senior Investigator
Cellular and Systems Neurobiology Section
Laboratory of Neural Control, NINDS

Jan Marino "Nino" Ramirez

Principal Investigator
Director of Center for Integrative Brain Research,
Seattle Children's Research Institute

Roger Traub

Dept. Physical Sciences
IBM T.J. Watson Research Center
Yorktown Heights, NY

Adjunct Professor of Neurology
Columbia University

Lectures

Synchronization of Neural Activity during Focal Neocortical Seizures

Daisuke Takeshita and **Sonya Bahar**

Department of Physics and Astronomy, University of Missouri at St Louis, MO

Rather than discuss neuronal bursting per se, I will address a closely related problem, that of neuronal synchronization. In an acute in vivo rat model of neocortical seizures, induced by injection of the potassium channel blocker 4-aminopyridine, D. Takeshita and I have investigated the spatial distribution of synchronized neocortical activity during seizures. Seizures were imaged using a voltage-sensitive dye (VSD). Following removal of heartbeat and respiratory artifacts, as well as artifacts caused by dye bleaching, the phase of the VSD signal was determined at each pixel using the Hilbert transform. Stochastic phase synchronization was used to determine the synchronization index between various pixel pairs in order to create synchronization maps illustrating the neocortical activity prior to, and during the seizure. We find a dramatic and statistically significant increase in synchronization during seizure events with respect to pre-seizure levels.

Ion concentrations and seizure dynamics

Ernest Barreto

Center for Neural Dynamics Krasnow Institute for Advanced Study,

George Mason University, VA

We develop models of individual neurons and of networks that include intra- and extra-cellular ion concentration dynamics. A reduction of the single neuron model is used to identify the bifurcation structure, leading to the identification of a novel mechanism for bursting and seizure-like events that are similar to those seen in experiments. We also examine possible mechanisms for excitatory-inhibitory interplay dynamics, also seen in experiments.

Network bistability mediates spontaneous transitions between normal and pathological brain states.

Maxim Bazhenov

Department of Cell Biology and Neuroscience, University of California, Riverside, CA

Little is known about how cortical networks support the emergence of remarkably different activity patterns. Physiological activity interspersed with epochs of pathological hyperactivity in the epileptic brain represents a clinically relevant yet poorly understood case of such rich dynamic repertoire. Using a realistic computational model, we propose that physiological sparse and pathological tonic-clonic activity may co-exist in the same cortical network for identical afferent input level. Transient perturbations in the afferent input are sufficient to switch the network between these two stable states. The effectiveness of the potassium regulatory apparatus determines the stability of the physiological state and the threshold for seizure initiation. These findings contrast with the common notions of (1) pathological brain activity representing dynamic instabilities and (2) necessary adjustments of experimental conditions to elicit different network states. Rather, we propose that the rich dynamic repertoire of cortical networks may be based on multistabilities intrinsic to the network.

Inhibitory synchronization in bursting networks

Igor Belykh

Department of mathematics and statistics and Neuroscience Institute, GSU, Atlanta, GA

Isospectral reduction of networks and spectral networks' equivalence

Leonid Bunimovich,

Georgia Institute of Technology, School of Mathematics, Atlanta,GA

It is tempting when dealing with large networks try to reduce it to a smaller network. However a danger is to lose in process of reduction some important features of a network we are dealing with. So one needs to know which features/properties of a network must be preserved in a process of such reduction. One of the most important characteristics of a dynamical network is the spectrum of its weighted adjacency matrix. We developed a procedure which allows to reduce networks to smaller (with less nodes) networks while keeping the spectra of their weighted adjacency matrices. Moreover this procedure allows to determine equivalence classes of networks. Namely two networks are spectrally equivalent if they can be isospectrally reduced to the same (smaller) network. Various examples of the isospectral reduction of networks will be demonstrated.

Multifunctional and specialized spinal interneurons for several kinds of limb movements

Ari Berkowitz

Department of Zoology, University of Oklahoma, Norman, OK

The turtle spinal cord, even without brain input and movement-related sensory feedback, can appropriately generate the fictive motor patterns underlying forward swimming, three forms of scratching, and flexion reflex (limb withdrawal). Does the spinal cord have a separate circuit to generate each or does a single, multifunctional circuit generate all of them? Our provisional answer is intermediate. Many spinal interneurons are multifunctional: they increase their activity for both scratching and swimming and usually during withdrawal as well. Most of these multifunctional neurons are rhythmically active during the same phase of the hip cycle during all forms of scratching and during forward swimming. This group includes transverse interneurons (T neurons), which were recently defined by morphological (somatodendritic) criteria.

Other interneurons, however, are specialized. One such group is activated during scratching but not swimming; some receive hyperpolarizing inhibition during swimming. A second group is activated strongly and at short latency during withdrawal, but is not activated during scratching or swimming; they can receive hyperpolarizing inhibition during scratching and swimming. This inhibition can be maximal during the hip flexor bursts, even though these neurons are activated during the hip flexor burst of limb withdrawal. Thus, these neurons are specialized for a particular behavior, not for a muscle or phase.

In summary, the spinal cord of an adult, limbed vertebrate appears to use a combination of multifunctional and specialized interneurons to generate locomotion, scratching, and limb withdrawal. Future research will focus on differences between multifunctional and specialized interneurons in morphology, neurotransmitters, and synaptic targets.

Voltage-dependent and Voltage-independent pacemakers in the pre-Botzinger Complex: An Integrative Model

Robert Butera

Laboratory for Neuroengineering, Georgia Institute of Technology, Atlanta, GA

Inspiratory neurons in the pre-Bötzinger complex (pBC) generate a regular rhythm that persists under highly variable neuronal input. The mechanism of pBC rhythm generation at the level of the network is an active area of debate. However, fractions of inspiratory pBC neurons generate a stable bursting rhythm even when pharmacologically isolated from the network, and likely contribute to the rhythm. Experiments indicate that the intrinsic bursting mechanism of these pacemaker neurons depends on either persistent sodium current or changes in intracellular Ca^{2+}. Motivated by experimental evidence obtained from these subpopulations of bursting neurons, we developed a two-compartment mathematical model of a pBC pacemaker neuron with two independent bursting mechanisms based on both of these mechanisms. The model explains a number of contradictory experimental results and generates a robust bursting rhythm over a large range of parameters, with a frequency adjusted by neuromodulators. The model predicts that in synaptically isolated cells, the bursting mechanism depends on neuromodulators, endogenously released within the pBC. The neuromodulatory tone can bias the neuron to a somatic (I_{NaP}) or dendritic (Ca^{2+} and I_{CaN}) mode of bursting, or a hybrid of the two. In the dendritic mode, the period of bursting is largely modulated by the IP_3 concentration, whereas in the somatic mode the burst duration is modulated by the persistent sodium current. This model displays changes in burst duration and period that are consistent with experimentally published pharmacological manipulations, such as the application of ion channel blockers (FFA and Riluzole) as well as neuromodulatory manipulations.

Given biological variation, how should we model small rhythmic networks?

Ronald L. Calabrese

Department of Biology, Emory University, Atlanta, GA

Variability in intrinsic membrane and synaptic parameters and their implication for network activity has received considerable theoretical attention (Prinz et al., 2004; Prinz, 2007). It is now increasingly clear that many biological neuronal networks, and CPGs in particular, can display a 2-5 fold range of intrinsic membrane currents and synaptic strengths while still producing stereotypical output (Bucher et al., 2005; Marder and Goaillard, 2006; Marder et al., 2007; Goaillard et al., 2009). Moreover, there are indications that average values for such strength parameters may be misleading in that models constructed from average values may not produce stereotypical output.

One of the long-range aims of my lab has been a complete model of how a CPG controls intersegmental motor outflow. In our system, the leech heartbeat CPG, output from premotor interneurons of the well-characterized CPG onto motor neurons is all inhibitory. We began by choosing an exemplar recording of spiking activity in all the intersegmental premotor interneurons as the temporal input for our model of motor neuron coordination into a fictive motor pattern (Norris et al., 2006). We then focused on a quantitative assessment of inhibitory synaptic strength and dynamics and associated conduction delays of premotor interneurons to be used as parameters in the model (Norris et al., 2007a, b). Using values averaged across animals for these synaptic strengths allowed us to construct a model that captured the gross intersegmental coordination but fell well short of quantitative verisimilitude with phase data for the fictive pattern averaged across animals (Garcia et al., 2008). This led us to reassess not only animal-to-animal variability in the pattern of synaptic strengths but also in the temporal pattern of interneuronal input and of motor neuron output. We found that all of these patterns, while on average true to the general conception of the fictive pattern, showed uncorrelated variability.

Given that we did not even consider variability in motor neuron intrinsic properties then it becomes clear that we are faced with nearly impossible task in arriving at a quantitatively accurate model of even the simplest neuronal networks: parameter and characteristic measurement must all be made in the same animal. Perhaps then there is merit in using average data for models and sacrificing quantitative accuracy for mechanistic understanding. On the other hand it may be desirable to tune a model based on average data to a desired output using automated search algorithms (with as many parameters set free as computational feasible) and then determine whether the parameters found fall within the biological range.

Reducing the fine structure of bursting dynamics

Robert Clewley

Neuroscience Institute, GSU, Atlanta, GA

We hypothesize that the key elements of a half-center bursting oscillator CPG can be represented in low dimensional form using a "dominant scales" reduction and simulation of the resulting hybrid dynamics. The model consists of a one compartment leech heart interneuron with 19 differential equations per cell. The hybrid (piecewise-reduced) dynamical system involves four sets of 6-7 equations each. This approximation will be numerically demonstrated along with the consequences for understanding the roles of the ion channels and synaptic coupling in the circuit's function.

Co-existence of epileptiform bursting and silent regimes in neuronal dynamics

Gennady Cymbalyuk, William Barnett and Tatiana Malaschenko

Neuroscience Institute and Department of Physics and Astronomy, GSU, Atlanta, GA

We modeled single neurons of leech ganglia under specific pharmacological conditions inducing epileptiform bursting (Opdyke & Calabrese 1994). The Ca^{2+} currents and the inhibitory coupling were blocked by Co^{2+}. The K^{+} currents, apart from the non-inactivating current, I_{K2}, were effectively blocked by 4-aminopyridine. Model consisted of I_{Na}, I_{K2}, I_h, and I_{NaP}. It demonstrates co-existence of silence and epileptiform bursting. We show that the co-existence can be explicated by the Rinzel scenario with the unstable sub-threshold oscillations (USTO) separating silence and an oscillatory regime and setting the threshold between them. The range of parameters, where the co-existence is observed, is determined by the critical values at which the USTO appear and disappear. More precisely, the USTO occur through the sub-critical Andronov-Hopf bifurcation, where the rest state loses stability. Then, the USTO disappear on the homoclinic bifurcation near which the oscillatory regime disappears as a regime. We investigate how modulations of different ionic currents affect the range of co-existence.

Synchronization of Time-Delayed Coupled Bursting Neurons

Mukesh Dhamala

Department of Physics and Astronomy and Neuroscience Institute, Georgia State University, Atlanta, GA

Time delays in signal propagation are unavoidable in spatially distributed coupled oscillator systems such as neurons in the brain. The interplay of coupling strengths and time delays can generate various interesting effects such as phase-flip transitions, cessation of oscillations and emergence of coherent patterns. In this talk, we will discuss the effects of time-delay coupling in neuronal oscillations and synchronization of synaptically coupled bursting neurons.

Modulating the ratio between excitatory and inhibitory neurons in in vitro neural networks

Rhonda Dzakpasu

Department of Physics, Georgetown University, Washington, DC

How is the network temporal structure altered when the balance between excitation and inhibition is changed? Proper balance is essential for normal brain function, including cognitive processing, the representation of sensory information and motor control. When the balance is compromised, neurological disorders may result. We use a simple reduced experimental system to investigate how manipulating the number of inhibitory neurons in a network of cultured hippocampal neurons affects synchronized bursting activity, the most prominent temporal signature of cultured hippocampal networks. Inhibitory neurons are thought to control spike timing and modulate network excitability and their absence may lead to widespread synchronization.

We culture dissociated hippocampal neurons with varying quantities of inhibitory neurons on an 8x8 grid of extracellular electrodes and study how inhibitory neurons modulate network temporal dynamics. We show that as the proportion of inhibitory neurons increase, there is a dramatic transition in the temporal pattern.

Neuromechanical analysis of posture and locomotion in crayfish

Donald H. Edwards

Neuroscience Institute, Georgia State University, Atlanta, GA

Crayfish locomote by walking while underwater and on land, and they use their legs to adopt a variety of stationary postures in both environments. Much of the circuitry responsible for control of posture or walking has been described in in vitro electrophysiological experiments on anesthetized, restrained and dissected animals, and so it is unclear whether these descriptions can account for the freely behaving animal's postural and locomotor behavior. To address this question, we have first analyzed the patterns of movement and EMG activity of the major leg muscle groups during postural adjustments and locomotion in freely behaving crayfish. Second, we are attempting to reconstruct the neuromechanical system controlling posture and locomotion in the crayfish with a computational model of the circuitry, proprioceptors, exoskeletal framework, and muscles of the animal, situated in a virtual physical world. The model was developed using AnimatLab (www.animatlab.com), a free, open-source, general purpose neuromechanical simulator. This talk will describe progress to date.

'Filling-In-The-Blanks': Therapeutic Stimulation of the Nervous System for Disorders Involving Abnormal Burst Firing

Robert E. Gross

Center for Neurodegenerative Disease, Emory University School of Medicine, Atlanta, GA

Involving Abnormal Burst Firing Electrical stimulation (ES) of the nervous system, such as 'deep brain stimulation'(DBS), has already replaced or will soon replace ablative procedures for a number of neurological disorders (e.g. Parkinson's Disease, depression), and moreover has expanded the palette of conditions that may be treated surgically. Remarkably, the mechanism(s) of action of ES remain poorly understood. Reconciling the similar effects of ablation and ES led some to early posit a depolarization blockade, but much recent evidence supports axonal activation as the primary mechanism. Many neurological disorders are characterized by abnormal patterns of neural output including increased 'burstiness' (e.g. PD, dystonia) if not outright burst-firing (e.g. epilepsy, pain). Does ES negate output ('electrical ablation'), or does it normalize the pattern of output by 'filling-in-the-blanks'? Preliminary data from intraoperative single unit recordings in a patient with PD will be presented that support the latter, and relevant literature reviewed. The pattern of discharges in models of epilepsy, and from human recordings will be reviewed, and attempts to translate a strategy to counter burst-firing akin to epileptic activity into a rodent model reviewed. The pattern of thalamic recordings in deafferentation pain, characterized by burst-firing, will also be reviewed, towards a mechanism of action in DBS for pain involving 'filling-in-the-blanks'.

Pathological bursting in basal ganglia circuits in Parkinson's disease.

Dieter Jaeger

Department of Biology, Emory University, Atlanta, GA

A relatively recent set of findings from multiple labs shows that neuronal activity in Parkinson's Disease (PD) is characterized by excessive bursting activity both in the basal ganglia and in cortex. This bursting activity is synchronized across neurons and across brain structures and in some cases is organized in an oscillatory fashion. These oscillations predominantly take place in the beta band (ca 12-30 Hz) and have been found to be correlated with the severity of PD symptoms. I will review this literature of pathological bursting and oscillations and show some initial results from our own lab comparing neural activity between normal mice to mice rendered parkinsonian with MPTP. I will also compare and contrast this activity with slow wave oscillations and bursting induced by anesthesia. An important questions remain whether the synchronized activity patterns during anesthesia in any way are mechanistically related to PD oscillations and bursting and could be used as a research tool to study pathological synchronization.

Biological Evolution of central pattern generators

Paul Katz

Neuroscience Institute, GSU, Atlanta, GA

Strudels: Intermittent MEG Sensor Field Correlates of the Inattentive Mind

Arnold Mandell

University of California San Diego, Can Diego, CA and Cielo Institute, Asheville, NC

Our research characterizes *symmetric sensor difference sequences, ssds(i),* in eyes closed, resting human brain magnetic flux fluctuations that: (1) Are intrinsic and spontaneous, not evoked; (2) Are treated as global dynamical magnetic scalar fields, not the reflections of vectorial current sources; (3) Are examined in the similarity and scaling regime with the modal ($\neq \Delta, \Theta, \alpha, \beta, \gamma\ldots$) content minimized (along with movement artifacts) via the *ssds(i)* computation.

Three minutes of eyes closed resting MEGs on ten schizophrenic Probands and ten sex and age matched Controls were recorded at 600 Hz with 150 Hz cut-off [1] from frontal (F14), central (C!6), parietal (P57) and temporal (T44) sensor pairs with high pass, 0.06Hz, and notch filters at 60,120,180 and 240 Hz before their transformation into *ssds(i).*

Borrowing from turbulence mathematic and our premise of conservation of brain entropy, the *ssds(i)* demonstrated, a statistically significant ~ 50% decrease in the *measureable entropy manifold volume, memv=*$\pi[\Lambda \times D_C \times h_T]$ in the Probands in all region's *ssds(i)* and increases in indices of emergent dynamical structure, X^4 , -α, and U_S. Note these findings, though statistically significant, indicate only suggestive trends in light of relatively small sample size. Similar intermittent dynamics are seen in taskless monkey pyramidal cell LFP in cortical layers 2 and 3 (data from D. Plenz).

In an effort to further explore what is meant by entropy loss and emergent dynamical structure in ssds(i)'s we compute the leading Broomhead/King leading eigenfunction of the t lagged auto-covariance matrices *of ssds(i),* Ψ(*ssds(i),* the lag obtained from the autocorrelation decay length, $R_{ssds}(t)$). Morelet wavelet transformation of W[Ψ(*ssds(i)*] revealed a unique pattern of intermittent, hierarchical scaling helices, we call *strudels* (whirlpools, eddies). Their duration and hierarchical scaling range varies from .006 to several seconds. Preliminary findings suggest a smaller number of strudels in the Probands versus Controls. Magnetic dynamo theory positing magnetic field stretching, lypounov exponent > 0, to form magnetic turbulent eddies (strudels) make consistent the decrease in *memv* and reduction in magnetic field strudels in Probands. Current fMRI and psychological research on the eyes closed, resting brain reveals intermittent, intrinsic activity called variously "default activation", "task unrelated thoughts" TUTs, "unrest at rest" "stimulus independent thoughts", SITs, daydreaming, fantasy and "mind wanders" manifest density and duration parameters not inconsistent with *strudels*. A "dropped" strudel in proband is seen in the 66.6 seconds W[Ψ(*ssds(i)*] below which may reflect "thought blocking" a primary Bleulerian sign of schizophrenia.

Multisite phase synchrony during chemically-induced seizures in rat brain

David J. Mogul

Department of Biomedical Engineering, Illinois Institute of Technology, Chicago, IL

A study of instantaneous spatiotemporal phase synchrony during chemically-induced seizures in rat limbic cortex was undertaken. Depth electrodes were placed in bilateral hippocampi and anteromedial thalamus for deep brain local field potential (LFP) recordings. A bone-screw pair were placed over the hemisphere contralateral to focal epileptogenic chemical placement. Micro-injection in the left hippocampus of either penicillin or kainic acid was used to induce seizures. Continuous recordings were made. Analysis of the LFP consisted of decomposition of each channel's signal to component oscillators using empirical mode decomposition, followed by calculation of phase using the Hilbert analysis. Mean phase coherence (a bivariate measure) was used to compare phase values among oscillators from all channels. This was then extended to a multivariate measure using eigenvalue decomposition yielding clusters of mean fields composed of two or more oscillators from one or more anatomical locations. Finally, the phase difference between oscillators belonging to each cluster was calculated and used to define instantaneous frequency over time. Frequencies of synchronization were found to be higher after exposure to either epileptogenic chemical. The numbers of synchronized events were found to be higher and more variable in both induction methods; however, the variability in frequency was much higher in kainic acid than in penicillin. In addition, synchronization frequencies within a given experiment but between different anatomical locations were found to be significantly different although some patterns were detected. Hence, these methods of assessing instantaneous spatiotemporal synchrony may be especially critical for better understanding such highly variable seizure dynamics.

Neuromodulation and bursting gone mad: Insights gained from studying neurological disorders

Jan-Marino Ramirez, Sebastien Zanella, Alfredo Garcia, Frank Elsen, Henner

Koch and Michael Carroll

Center for Integrative Brain Research, Seattle Children's Research

Institute, Seattle, WA.

Multiarray electrode recordings from the pre-Bötzinger complex, an area critical for the generation of the respiratory rhythm in mammals, reveal that respiratory neurons exhibit significant cycle-by-cycle variation. Pacemaker and non-pacemaker neurons could lead a respiratory burst during one cycle, but could be "followers" during subsequent cycles. Computational simulations aimed at modeling these electrophysiological findings suggest that the mammalian respiratory network is sparsely connected. In this network configuration, norepinephrine induces bursting properties which contributes to an increased regularity of the respiratory rhythm. Interestingly, the same stabilizing neuromodulator causes dramatic irregularities if the network was previously exposed to intermittent hypoxia. We find dramatic irregularities in the generation of burst frequency and amplitude. Thus the relationship between a neuromodulator and the modulatory response of the network is not fixed. We hypothesize that this switch in the modulatory response is caused by subtle changes in the configuration of the respiratory network. This finding has important clinical implications: Neuronal networks that are normally stabilized by neuromodulators become disrupted, which could result in a clinical phenotype that is driven by its own neuromodulators. Pharmacological interventions aimed at simply compensating for a deficiency in neuromodulation could worsen a clinical phenotype.

Late-Expiratory Oscillations in RTN/pFRG: Emergence and Coupling with Respiratory CPG

Ilya A. Rybak[1], Ana P. L. Abdala[2], Yaroslav I. Molkov[1], Bartholomew J. Bacak[1], Julian F. R. Paton[2], and Jeffrey C. Smith[3]

[1]Drexel University College of Medicine, Philadelphia, PA, USA; [2]University of Bristol, Bristol, UK; [3]NINDS, NIH, Bethesda, MD, USA.

The respiratory rhythm and motor pattern are generated by a spatially organized brain stem respiratory network with a rhythmogenic core comprising interacting neural populations within the pre-Bötzinger (pre-BötC) and Bötzinger (BötC) complexes controlled by drives from other brain stem compartments. Our multi-compartmental computational model of the brainstem respiratory network (Rybak et al. 2007; Smith et al. 2007) was extended by incorporating the retrotrapezoid nucleus/parafacial respiratory group (RTN/pFRG) compartment to investigate neural oscillations emerging within this region and driving late-expiratory (late-E) bursting in the abdominal motor output. In the extended model, the late-E oscillations origin within a late-E population of RTN/pFRG consisting of excitatory neurons with persistent sodium current – dependent endogenous bursting propertirs and mutual excitatory interconnections within the population. The model suggests that under normal conditions the late-E oscillations are inhibited by the BötC/pre-BötC network, but may be released by either hypercapnia-evoked activation of RTN/pFRG or hypoxia-dependent suppression of RTN/pFRG inhibition by BötC/pre-BötC. The proposed interactions between the BötC/pre-BötC and RTN/pFRG allow the model to reproduce multiple experimentally observed behaviors, including (a) quantal acceleration of late-E (and abdominal nerve) oscillations with development of hypercapnia and (b) quantal slowing of BötC/pre-BötC (and phrenic nerve) oscillations with progressive suppression of pre-BötC excitability, as well as to predict a release of late-E oscillations by disinhibition of RTN/pFRG under normal conditions. The model proposes mechanistic explanations for the emergence of RTN/pFRG oscillations and their interaction/coupling with the rerspiratory oscilations generated by the respiratory CPG.

Accelerating simulations of bursting neurons with simEngine

Michael Sorensen

Simatra Modeling Technologies, Atlanta, GA

The study of rhythmic systems, and computational neuroscience in general, often requires complex biophysical models and extensive simulation. Although these simulations can provide invaluable insights into the theory and function of biological systems, they are often computationally intensive, requiring significant computing power to solve the simulations in a reasonable time frame. Modern computer hardware architectures, such as multi-core processors, GPUs, and cloud resources, have driven the cost of computing down, but the parallel programming required to take advantage of these architectures is a difficult and time-consuming distraction for the researcher. To solve this problem, Simatra has developed simEngine, a lightweight compiler and Matlab toolbox for simulating nonlinear dynamical systems that automatically builds optimized simulation executables for low-cost, high-performance hardware. We will demonstrate how simEngine can be used to examine the bursting properties of a both single cell and rhythmic network

---

Polyrhythms of bursting patterns in deterministic models for central pattern generators

Andrey Shilnikov, Jeremy Wojcik, Robert Clewley and Igor Belykh

Neuroscience Institute and Department of Mathematics, GSU, Atlanta, GA

We study the emergence of bursting dynamics in inhibitory-excitatory networks, such as central pattern generators (CPG) controlling various locomotive behaviors of animals. We show that the pacemaker determining the specific rhythm of a CPG comprised of realistic Hodgkin–Huxley-type interneurons is identified through the order parameter, which is the duty cycle of bursting. We discuss the origin of multistability in inhibitory networks giving raise to polyrhythmicity of synchronous bursting patterns in multifunctional CPGs.

Multiple modes and mechanisms of rhythmic burst pattern generation in brainstem respiratory networks

Jeffrey C. Smith[1], Ana P. L. Abdala[2], Julian F. R. Paton[2], Jonathan Rubin[3], and Ilya A. Rybak[4]

[1]NINDS, NIH, Bethesda, MD, USA; [2]University of Bristol, Bristol, UK; [3]University of Pittsburgh, PA, USA; [4]Drexel University College of Medicine, Philadelphia, PA, USA.

Neural circuits controlling breathing in mammals are structurally and functionally organized within hierarchically arranged brainstem compartments arrayed rostro-caudally from the pons to the lower medulla. Core circuit components of the neural machinery generating respiratory rhythm and shaping inspiratory and expiratory motor patterns are distributed among two adjacent structural–functional compartments in the ventrolateral medulla— the Bötzinger complex (BötC) and pre-Bötzinger complex (pre-BötC). Recent experimental and modeling studies to be described in this talk indicate that inhibitory network interactions within these compartments along with intrinsic rhythmogenic properties of pre-BötC excitatory circuits engender multiple mechanistic modes for rhythmic burst pattern generation. Expression of these mechanisms is controlled by multiple input excitatory drives, including from pontine, retrotrapezoid, and raphe nuclei, which regulate dynamic behaviour of the core BötC–pre-BötC circuitry. The hierarchical organization of the system has been revealed by progressively reducing the respiratory network to uncover circuit architectural building blocks in several experimental preparations including the in situ perfused brainstem and in vitro brainstem slice preparations from rodents. We propose that this organization enables state-dependent expression of different rhythmic burst pattern generation mechanisms that underlie respiratory motor behaviours emerging under various physiological and pathophysiological conditions.

The unusual bursting pattern produced by pituitary cells

Joel Tabak

Department of Biological Science, Florida State University, FL

Endocrine cells of the anterior pituitary are electrically excitable. Their electrical activity pattern affects intracellular calcium level and in turn the level of hormone secretion. Pituitary bursts are characterized by small amplitude oscillations on top of a plateau depolarization, unlike the high amplitude spiking that characterizes most neuronal bursting. This talk will highlight some differences between models of pituitary bursting and the more common square-wave bursters. I will then show that we can switch between pituitary and square-wave bursting by changing only two model parameters. Finally, I will show why the large conductance K^+ (BK) channels can have a stimulatory effect on bursting and demonstrate this effect in pituitary cells using the dynamic clamp.

Some normal and abnormal collective phenomena in cortical circuits amongst bursting neurons

Roger Traub

Dept. Physical Sciences, IBM T.J. Watson Research Center, Yorktown Heights, NY

One major type of intrinsic bursting (IB) in pyramidal neurons depends on the interaction between dendritic calcium currents and perisomatic action potential currents. IB pyramidal neurons can participate in collective phenomena of two broad types: in one, the actual burst generating properties matter, while in the other these properties seem to be irrelevant. An example of the first type would be synchronized epileptiform bursts in the hippocampus, which depend on the ability of intrinsic bursting in one pyramidal cell to induce bursting in other pyramidal cells. An example of the second type of phenomenon is a ~25 Hz "beta2" oscillation in layer 5 in association cortex. Beta2 proceeds even after surgical separation of the apical dendrites, where a major portion of calcium conductances are located. Interestingly, epileptiform bursts are suppressed by blockade of chemical synapses, while beta2 does not depend on chemical synapses and instead depends on gap junctional coupling.

TRP-ing over your next breath and enhancing neocortical oscillations

Andrew Tryba

Department of Physiology, Medical College of Wisconsin, WI

Intrinsic bursting properties are proposed to underlie respiratory and neocortical rhythms. A neural network, called the pre-Bötzinger Complex, is critical to respiratory rhythm generation. Respiratory network and pacemaker bursting depends on two rhythmogenic currents: 1) the persistent sodium current (I_{NaP}); and 2) the non-specific cation current (I_{CAN}). neuromodulators, such as serotonin (5HT) and Substance P (SubP), play key roles in conditional respiratory network and pacemaker bursting. 5HT acts via 5HT2a receptors to trigger I_{NaP}-dependent bursting, whereas SubP acts via NK1R to trigger I_{CAN}. We recently found that I_{CAN}-dependent bursting relies on activation of TRPC3/7 channels, suggesting a novel mechanism for bursting rhythmogenesis. We also found that activation of TRPC channels plays an important role in breath-by-breath regularity. In contrast to the respiratory network, neocortical oscillations in several mamalian species, including humans, are proposed to depend on I_{NaP}-dependent intrinsically bursting neurons. Indeed, our data suggests that human neocortical intrinsic bursting neurons are I_{NaP}-dependent. We also found that excitatory neuromodulation of TRPC channels can enhance I_{NaP}-dependent intrinsic bursting properties in the neocortex and promote neocortical oscillations.

UP and DOWN states in neocortical neurons during slow wave oscillations

Maxim Volgushev

Department of Psychology, University of Connecticut, Storrs, CT

Slow wave sleep and some types of anaesthesia are characterised by slow (~1 Hz) large amplitude waves in the EEG, which reflect spontaneous alternations of active and silent states in thalamocortical networks. In the neurons, active states are associated with depolarization, vigorous synaptic activity reflected in high-frequency oscillations of the membrane potential and cell firing. During silent states, cells are hyperpolarized and fire no action potentials.

In my talk I will discuss experimental results showing that all neurons in the neocortex and most neurons in their target structures are involved in the slow rhythm and oscillate in-phase, being simultaneously active or simultaneously silent. This generalized nature of the slow oscillation excludes a "canonical oscillator" scheme in which mutually inhibiting populations of neurons oscillate in contra-phase. Instead, mechanism(s) mediating a synchronized origin of activity in a completely silent cortical network should exist. So far, three hypotheses on the origin of active states were proposed. The first hypothesis suggests that spontaneous release of transmitter occasionally depolarises some neurons to the firing threshold, which then trigger activity in the whole network. The second hypothesis maintains that active states are initiated by large layer 5 pyramids, which remain more depolarized due to their intrinsic or synaptic properties and continue to generate some spikes when other neurons are silent. The third hypothesis attributes origin of active states to the selective synchronization of small neuronal ensembles. The three hypotheses make distinct predictions as to the pattern and origin of activity during slow oscillation. I will argue that predictions of the "spontaneous release" hypothesis are best compatible with existing experimental data.

Bursting with desire: a two-timescale rhythm for mate exploration in the medicinal leech

Daniel Wagenaar

Division of Biology, CalTech, Pasadena,CA

NMDAR-dependent control of call duration in Xenopus laevis

Ayako Yamaguchi

Department of Biology, Boston University, Boston, MA

Many rhythmic behaviors are composed of temporally dynamic patterns. How does the brain organize temporal complexity of rhythmic behavior? We use the vocal central pattern generator (CPG) of Xenopus laevis to address this question. Isolated Xenopus brains can elicit fictive vocal nerve activity allowing us to study the CPG in vitro. The Xenopus advertisement call is temporally-modulated; calls consist of rhythmic click trills that alternate between fast and slow rates. We investigated the role of two CPG nuclei—the laryngeal motor nucleus (n.IX-X), and the dorsal tegmental area of the medulla (DTAM)—in setting rhythm frequency and call/trill durations. We discovered a local field potential (LFP) with two components; a baseline wave that coincides with fast trill and a phasic activity that coincides each fictive click. The wave persists after disrupting n.IX-X connections, whereas the phasic activity disappears after disrupting the connections, indicating that the wave is endogenous to DTAM while the phasic activity relies on the intact connection between DTAM and n.IX-X. A series of experiments were conducted to demonstrate that the DTAM wave determines the timing of fast trills in an NMDAR-dependent manner. Preliminary intracellular recordings indicate that the DTAM waves may reflect bursts of postsynaptic potentials received simultaneously by DTAM neurons. These findings indicate that at least two functionally distinct CPG circuits exist—a pattern generator in DTAM that determines trill and call duration, and a rhythm generator (spanning DTAM and n.IX-X) that determines click rates.

Posters

P1 *Pattern scaling in neuronal dynamics*

[1]**William Barnett**, [2]Martin Anquez, [1,2]Gennady Cymbalyuk

[2]Department of Physics and Astronomy and [1]Neuroscience Institute, GSU, Atlanta, GA

P2 *Autonomous spiking and bursting in a mouse ventricular cell model.*

Vladimir E. Bondarenko and Andrey L. Shilnikov

Department of Mathematics and Statistics, GSU, Atlanta, GA

P3 *Multi-modal optimization techniques for biophysical neuron models.*

Mirza Dobric and Robert Clewley

Department of Mathematics and Statistics, GSU, Atlanta, GA

P4 *Characterizing change in activity patterns in a Drosophila motoneuron with different sodium channel splice variants.*

Anca Doloc-Mihu and Ronald Calabrese

Department of Biology, Emory University, Atlanta, GA

P5 *Role of Extracellular K^+ Dynamics in Network Synchronization.*

Gregory Filatov and Maxim Bazhenov

Department of Cell Biology and Neuroscience, University of California, Riverside, CA

P6 *Synchronization in a bursting half-center oscillator with slow-to-fast reciprocal inhibition.*

Sajiya Jalil, Andrey Shilnikov, Igor Belykh

Department of Mathematics and Statistics and Neuroscience Institute, GSU, Atlanta, GA

P7 *Firing rates of cat muscle Ia, Ib, II and paw cutaneous afferents during walking computed using a musculoskeletal hindlimb model*

Alexander N. Klishko and Boris I. Prilutsky

School of Applied Physiology, Georgia Institute of Technology, Atlanta, GA, USA

It has been recognized for a long time that during locomotion feedforward (central pattern generator, CPG) and feedback (proprioceptive afferent) signals operate together to control movements and adapt locomotion to conditions of the environment and the musculoskeletal system. However, the structure and function of the mammalian CPG and the role of afferent feedback in mammalian locomotor control are not well understood. Our long-term goal is to develop a comprehensive neuromusculoskeletal model of the spinal control of locomotion by combining the model of the CPG and basic spinal reflexes (Rybak, McCrea, 2008) with a musculoskeletal model of cat hindlimb generating realistic motion-dependent afferent signals. Such a neuromusculoskeletal model will be used to study the mechanisms of spinal locomotion in mammals. The aim of this presentation is to demonstrate the recent developments of our cat musculoskeletal hindlimb model that computes the activity of hindlimb spindle, tendon organ and paw skin afferents during walking from simulated muscle fascicle length and velocity, tendon forces and loads on plantar surface of the paw using known relationships between these mechanical variables and the firing rate of muscle Ia, Ib, II and skin afferents (Prochazka, Gorassini, 1998). The developed hindlimb musculoskeletal model with its identified parameters allowed for a close match between the simulated and experimental joint angles, joint moments and ground reaction forces during walking. The computed firing rates of hindlimb Ia, Ib, II and paw skin afferents were compared with the corresponding in vivo recordings reported in the literature and demonstrated reasonable qualitative match.

P8 *Mechanisms of frequency control in the midbrain dopaminergic neuron.*

Alexey Kuznetsov

Department of Mathematical Sciences, IUPUI, Indianapolis, IN

P9 *Modulation of bursting and tonic activity during seizure by ion concentrations explains spontaneous seizure termination.*

Giri P Krishnan and Maxim Bazhenov

Department of Cell Biology and Neuroscience, University of California, Riverside, CA

P10 *Good currents playing bad: propensity to bi-stability in neuronal dynamic.*

[1,2]**Tatiana Malaschenko** and [1,2]Gennady Cymbalyuk

[1]Department of Physics and Astronomy and [2]Neuroscience Institute, GSU, Atlanta, GA

P11 *Distribution of Burst Length and Inter-Burst-Interval of the stochastic Morris-Lecar Neuron*

Peter Rowat

Institute for Neural Computation, UCSD, La Jolla, CA

We demonstrate that the length of a spike sequence of the stochastic Morris-Lecar neuron is geometrically distributed. The parameter of this geometric distribution turns out to be a sigmoidal function of applied current. A long portion of the inter-spike interval (ISI) distribution of this model is exponential, with parameter depending on applied current, again according to a sigmoidal function. We obtain these results by a combination of analysis of the stochastic dynamics of the model with estimation based on simulations.

P12 *From Plateau to Pseudo-Plateau Bursting: Making the Transition.*

Wondimu Teka

Department of Mathematics, Florida State University, Tallahassee, FL.

Bursting electrical activity is ubiquitous in excitable cells such as neurons and many endocrine cells. The technique of fast/slow analysis, which takes advantage of time scale differences, is typically used to analyze the dynamics of bursting in mathematical models. Two classes of bursting oscillations that have been identified with this technique, plateau and pseudo- plateau bursting, are often observed in neurons and endocrine cells, respectively. Using fast/slow analysis, we show that models for one type can produce the other, and we provide a procedure for achieving this transition. This suggests that the design principles for bursting in endocrine cells are just quantitative variations of those for bursting in neurons.

P13 *Covarying ionic conductances to emulate phase maintenance in stomatogastric neurons*

Wafa Soofi

Department of Biology, Emory University, Atlanta, GA

P14 *Deviations from the straight and narrow: C. elegans uncoordinated mutants that Circle*

David Qian, Gennady Cymbalyuk and Bill Walthall

Department of Biology and Neuroscience Institute, Georgia State University, Atlanta, GA

P15 *Modeling State-Dependent Interactions between BötC/pre-BötC and RTN/pFRG oscillations*

[1]Yaroslav I. Molkov, [2]Jonathan E. Rubin, [1]Bartholomew J. Bacak, [1]Natalia A. Shevtsova, [3]Jeffrey C. Smith and [1]Ilya A. Rybak

[1]Department of Neurobiology and Anatomy, Drexel University College of Medicine, Philadelphia, PA

[2]Department of Mathematics, University of Pittsburgh, Pittsburgh, PA

[3]Cellular and Systems Neurobiology Section, Porter Neuroscience Research Center, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD

Late-expiratory (late-E, or pre-inspiratory) oscillations emerge in abdominal motor output with increasing metabolic demands (e.g., during hypercapnia, hypoxia, etc.). These oscillations originate in the retrotrapezoid nucleus / parafacial respiratory group (RTN/pFRG) and couple with the respiratory oscillations generated by the interacting neural populations within and between the Bötzinger (BötC) and pre-Bötzinger (pre-BötC) complexes, representing the kernel of the respiratory central pattern generator. Based on experimental data on the generation of late-E oscillations we have developed a model that simulates the possible interactions between the BötC/pre-BötC and RTN/pFRG oscillations under different conditions. Three physiologically relevant behaviors have been analyzed: emergence and quantal acceleration of late-E oscillations during hypercapnia, transformation of the late-E activity into a biphasic-E activity with the development of hypoxia, and quantal slowing of BötC/pre-BötC oscillations with the reduction of pre-BötC excitability. Each behavior is elicited by gradual changes in excitatory drives or other model parameters, reflecting specific changes in metabolic and/or physiological conditions. Our results provide important theoretical insights into interactions between RTN/pFRG and BötC/pre-BötC oscillations and the role of these interactions in the control of breathing under different metabolic conditions.

P16 *Poincare Mappings for Models of Elliptic Bursters*

Jeremy Wojcik and Andrey Shilnikov

Department of mathematics and statistics and Neuroscience Institute, GSU, Atlanta, GA

Using a computer-assisted reduction to an 1D Poincar'e mapping for an voltage interval, we provide with an in-depth examination of global bifurcations of periodic orbits. These bifurcations underly complex transitions between tonic spiking, bursting, and mixed-mode oscillations and as well as quiescence in three representatives of elliptic bursters: a mathematical FitzHugh-Rinzel model, a bursting implementation of the Hodgkin-Huxley model, and a realistic Rubin-Terman model for the external segment of the Globus Pallidus.

P17 *The breathing disorders of a mouse model of Rett syndrome are linked to defects in central CO2 chemosensitivity of locus coeruleus neurons*

Xiaoli Zhang, Junda Su, Ningren Cui, Hongyu Gai, Zhongying Wu and Chun Jiang

Department of Biology, Georgia State University, Atlanta, GA

P18 *Reactive oxygen species (ROS) inhibit KATP channel via S-glutathionylation*

Yang Yang, Weiwei Shi, Ningren Cui, Xianfeng Chen, Timothy C. Trower, and Chun Jiang

Department of Biology, Georgia State University, Atlanta, GA

P19 *Characterizing change in activity patterns in a Drosophila motoneuron with different sodium channel splice variants.*

Cengiz Gunay, Fred Sieling, Logesh Dharmar and Astrid Prinz

Department of Biology, Emory University, Atlanta, GA

Participating Institutions

Georgia State University, Atlanta, GA

Emory University, Atlanta, GA

Georgia Institute of Technology, Atlanta, GA

Simatra Modeling Technologies, Atlanta, GA

Florida State University, Tallahassee, FL

Krasnow Institute for Advanced Study, George Mason University, VA

University of Missouri at St Louis, MO

University of California, Riverside, CA

University of Oklahoma, Norman, OK

California Institute of Technology, Pasadena, CA

Georgetown University, Washington, DC

Boston University, Boston, MA

Seattle Children's Research Institute, Seattle, WA

Porter Neuroscience Research Center, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD

Drexel University College of Medicine, Philadelphia, PA, USA

Medical College of Wisconsin, WI

IUPUI, Indianapolis, IN

IBM T.J. Watson Research Center, Yorktown Heights, NY

Illinois Institute of Technology, Chicago, IL

University of Connecticut, Storrs, CT

UCSD, La Jolla, CA

Scientific and Organizing Committee

Gennady Cymbalyuk
Ronald Calabrese
Andrey Shilnikov
Robert Clewley
Sonya Bahar
Igor Belykh
Vladimir Bondarenko
Mukesh Dhamala

Student Core Organizing Committee:

Tatiana Malaschenko
Sajiya Jalil
Bhim Adhikari
William Barnett
Jeremy Wojcik
Michael Mooney

Notes

Notes