NeuroEng 2013
Melbourne Australia

6th Australian Workshop on Computational Neuroscience

The University of Melbourne
30-31 January 2013

Electrical & Electronic Engineering
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Welcome to NeuroEng 2013 Workshop

Welcome to the NeuroEng 2013 Workshop! NeuroEng 2013 is the sixth in the series of Australian Workshops on Computational Neuroscience. The aim of this on-going series of workshops is to bring together researchers in Australia and New Zealand who are working at the interface between neuroscience and engineering, including researchers working in mathematical and computational neuroscience, neural modelling, neuroimaging, EEG analysis, neuromorphic engineering, and neuroprostheses. More information can be found on the website of The Australian Association of Computational Neuroscientists and Neuromorphic Engineers, where you can also sign up for membership:

www.neuroeng.org.au

We have a strong focus upon providing an opportunity for new researchers to meet with those who are established in the field. Thus, there is a mix of talks and posters from eminent invited keynote speakers, senior researchers and new researchers. We have deliberately intermingled the talks and posters in order to encourage a cross fertilisation of ideas and approaches. We have also allocated ample time for the poster sessions and we hope that all participants will use this time to speak and engage with as many other participants of all levels across the interdisciplinary and geographic boundaries as possible.

While this meeting is predominantly a local workshop, we welcome and value the participation from our international visitors. In particular, we are pleased to have two outstanding research leaders in different aspects of neuroengineering as keynote speakers at the Workshop: Steven Schiff, Penn State Center for Neural Engineering, and Moira Steyn-Ross, University of Waikato.

The Workshop has been organized by a scientific committee and a local organising committee of volunteers. Submissions to the Workshop were accepted based upon a review process and all submissions were reviewed by at least two members of the scientific committee, and by the local organising committee. Feedback was provided to the authors and in many cases resulted in re-submission of modified abstracts. Thanks to all who so willingly contributed their time and skills in this process.

We are grateful to the sponsors of NeuroEng 2013, including: NeuroEngineering Laboratory, Dept. of Electrical & Electronic Engineering, University of Melbourne; Centre for Neural Engineering, University of Melbourne; Melbourne School of Engineering, University of Melbourne; Monash Biomedical Imaging, Monash University; Swinburne Research, Swinburne University of Technology; National ICT Australia (NICTA); and Bionic Vision Australia. In addition, the Florey Neuroscience Institutes has provided two prizes for the best student presentation or poster.

Finally, thank you to all who are presenting and attending the NeuroEng 2013. I hope that, over the course of the Workshop, you have many interesting discussions and forge new relationships with other researchers, both junior and more experienced, and that you encounter plenty of interesting new ideas to fire your imagination!

David Grayden,
Chair NeuroEng 2013

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*Prizes sponsored by the Florey Institute of Neuroscience and Mental Health will be awarded to the Best Student Oral Presentation and Best Student Poster Presentation.

WORKSHOP PROGRAM

Wednesday 30th January

08:00-09:00 **Registration**

09:00-09:30 **Introduction and Welcome**

09:00-09:15 Welcome by Chair

09:15-09:30 Opening Address by Prof. James McCluskey
Deputy Vice-Chancellor (Research), University of Melbourne

09:30-10:30 **Keynote Session 1: Prof. Steven Schiff**
Towards Model-based Observation and Control in the Brain: Seizures, Parkinson's Disease, and Migraines

10:30-11:00 **Morning tea**

11:00-12:30 **Oral Session 1**

11:00-11:30 Bruce Graham
Synaptic Learning Beyond STDP

11:30-12:00 Cliff Kerr
How Parkinson's Disease Affects Cortical Information Flow: A Multiscale Model

12:00-12:30 Pulin Gong
Associative Learning as an Emergent Property of Spatially Extended Spiking Neural Circuits with Spike-Timing-Dependent Plasticity

12:30-15:30 **Lunch / Poster Session 1 / Afternoon Tea**

15:30-17:30 **Oral Session 2**

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Auditory Temporal Processing

16:00-16:30 David Liley
The Mesoscopic Modeling of Burst Suppression During Anaesthesia

16:30-17:00 Mark McDonnell
Slow Bistability in Fluctuation Driven Models of Cortical Networks with Multiplex Neuronal Connectivity

17:00-17:30 Shaun Cloherty
Statistical Criteria for Assessing Phase Sensitivity of Visual Cortical Neurons

19:00-late **Dinner**

WORKSHOP PROGRAM

Thursday 31st January

09:00-10:00 **Keynote Session 2: Prof. Moira Steyn-Ross**
Modelling Symmetry-Breaking Transitions in the Cortex

10:00-10:30 **Morning tea**

10:30-12:30 **Oral Session 3**

10:30-11:00 Peter Robinson
Brain Networks: Roles of Stability and Geometry in Determining Connectivity

11:00-11:30 Hamish Meffin
Mean Field Formalism for Electrical Stimulation of Nerve Fibre Bundles

11:30-12:00 Tara Hamilton
The Dynamic Ripple Pond Recognition Network

12:00-12:30 Kiri Pullar
Functional Design of the Electrosensory System in Sharks

12:30-15:30 **Lunch / Poster Session 2 / Afternoon Tea**

15:30-17:00 **Oral Session 4**

15:30-16:00 Geoffrey Goodhill
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Effects of Electroconvulsive Therapy Stimulus Frequency

16:30-17:00 Tjeerd Boonstra
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17:00 **Finish**
Closing remarks and announcement of NeuroEng Australia and NeuroEng 2014

Keynote Speaker
Prof. Steven Schiff

Brush Chair Professor of Engineering and Director of the Penn State Center for Neural Engineering

Towards Model-based Observation and Control in the Brain: Seizures, Parkinson's Disease, and Migraines

Wednesday 30th January, 09:30-10:30

Since the 1950s, we have developed mature theories of modern control theory and computational neuroscience with surprisingly little interaction between these disciplines. With the advent of computationally efficient nonlinear Kalman filtering techniques, along with improved neuroscience models that provide increasingly accurate representation of dynamics in a variety of important normal and disease states in the brain, the prospects for a synergistic interaction between these fields are now strong. We will examine the reconstruction of seizure dynamics from experimental data, explore the prospects of using models of Parkinson's disease to observe and control motor system dynamics, and discuss ongoing work to perform model-based control of spreading depression – the underpinning of migraine auras. The broad potential impact of the rigorous application of observability and controllability to better understand the brain and treat dynamical neuronal disease systems will be discussed.

Steven J. Schiff, Brush Chair Professor of Engineering and Director of the Penn State Center for Neural Engineering, is a faculty member in the Departments of Neurosurgery, Engineering Science and Mechanics, and Physics. A Pediatric Neurosurgeon with particular interests in Epilepsy and Hydrocephalus, he holds a Ph.D. in Physiology, and an M.D., from Duke University School of Medicine. Dr. Schiff is a Fellow of the American Physical Society, the American College of Surgeons, and the American Association for the Advancement of Science. He has been listed in the Consumer's Research Council of America's guides to top physicians and surgeons. He plays the viola in the Nittany Valley Symphony in an out of tune manner.

Keynote Speaker
Prof. Moira Steyn-Ross

Professor of Physics, School of Engineering,
University of Waikato

Modelling Symmetry-Breaking Transitions in the Cortex

Thursday 31st January, 09:00-10:00

Imaging studies have revealed fluctuating patterns of neural activation which are believed to represent cognitive states of the brain. The origin of such patterning has not yet been unambiguously established. I will discuss a mean-field model of the cortex in which spatiotemporal patterns can arise spontaneously through a symmetry-breaking Turing (spatial) bifurcation, modulated by a temporal (Hopf) instability. In our model, populations of neurons are densely interlinked by both chemical synapses, including an idealized long-range axonal connection, and by direct electrical connections forming a continuous network of interneuronal gap junctions. We argue that normal functioning of the awake brain requires a delicate balance between Turing and Hopf instabilities, resulting in spatial patterns which allow information transfer through global rhythms. Reduction of gap-junction diffusivity disturbs the balance in favor of the Hopf instability, eventually predicting global seizure in the limit of severe imbalance. To demonstrate model behaviour over a wide range of distinct of brain states, we explore model dynamics in the vicinity of a general-anaesthetic induced transition from wake to coma. We model anaesthesia as a moderate reduction in inhibitory diffusion, paired with an increase in inhibitory postsynaptic response, producing a coma state that is characterized by emergent low-frequency oscillations whose dynamics is chaotic in time and space. We suggest that this spatiotemporally chaotic state, generated by the nonlinear Turing–Hopf interaction, may provide a mechanism for the slow oscillation observed in general anesthesia. Such a mechanism may also explain the slow oscillation of natural sleep and thus indicate a role of chaotic dynamics in memory processing and learning hypothesized to occur during deep sleep.

Moira Steyn-Ross is a professor of physics in the School of Engineering at the University of Waikato, NZ. She obtained a PhD in theoretical quantum optics and stochastic physics, and has also researched applications of satellite remote sensing. Since 1997, she has worked in theoretical modelling of the cerebral cortex, focusing on the anaesthetic phase transition, natural sleep, and has a general interest in nonlinear dynamics as manifest in the cortical system.

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THE DYNAMIC RIPPLE POND RECOGNITION NETWORK

Saeed Afshar[1], Gregory Cohen[2], André van Schaik[2], Jonathan Tapson[2], Tara Julia Hamilton[1]

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Introduction

We present the Dynamic Ripple Pond Recognition Network (dRPRN), a neuromorphic visual recognition system that achieves rapid, view-invariant, multiple object recognition in a randomly structured spiking neural network. It connects two recently developed neuromorphic components: the Dynamic Vision Sensor (DVS) and the Spike-Timing Dependent Delay Plasticity (STDDP) circuit.

Methods

The DVS system is a biologically-inspired asynchronous time-based camera for signal acquisition [1]. The STDDP circuit is a hardware implementation of a polychronous network that is capable of learning and recalling spatio-temporal spike patterns [2]. The spatio-temporal spike train from the DVS camera forms the raw activation 'images' for the system. The dRPRN receives this input and operates as a bottom-up salience detector, simultaneously extracting any number of significant objects whilst rapidly converting the complex input images into multiple shift/rotation/scale and skew invariant time series signatures.

From a high-level functional viewpoint, the dRPRN uses a succession of localised Low-Pass Filter→Normalization→Threshold operators to create circular activation regions centered on the most salient objects in its field of view. The resultant activation is then projected on a locally inhibitory, randomly distributed and connected neural network. This creates vector fields onto which the original image is projected. The outputs are then directly read out from the most highly activated neurons in this vector field. This output is the view-invariant time-series signature that is well suited to training and recalling in the polychronous network.

Results

The resulting end-to-end system presented is a complete, biologically-inspired neuromorphic visual recognition system using time-based, decentralized processing deliverning robust, high-speed performance. The dRPRN's architecture also offers insights into how recognition is achieved in real biological systems where energy constraints and rapid speed are the paramount factors selected by evolution.

[1] Lichtsteiner, P., C. Posch, and T. Delbruck. A 128 X 128 120db 30mw asynchronous vision sensor that responds to relative intensity change. in Solid-State Circuits Conference, 2006. ISSCC 2006. Digest of Technical Papers. IEEE International. 2006. IEEE.

[2] Wang, R., et al. An aVLSI programmable axonal delay circuit with spike timing dependent delay adaptation. in Circuits and Systems (ISCAS), 2012 IEEE International Symposium on. 2012.

MODELLING POTASSIUM CURRENTS IN AII AMACRINE CELLS

N. Apollo[1,2], T. Kameneva [1,2,3]

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[2]Centre for Neural Engineering; [3]NICTA Victoria Research Lab; tkam@unimelb.edu.au.

Introduction

In patients who have lost their photoreceptors due to retinal degenerative diseases, it is possible to restore rudimentary vision by electrically stimulating surviving neurons. AII amacrine cells, which reside in the inner plexiform layer, split the signal from rod bipolar cells into ON and OFF cone pathways. As a result, it is of interest to develop a computational model to aid in the understanding of how these cells respond to the electrical stimulation delivered by a prosthetic implant.

Aims

The aim of this work is to develop a single-compartment model of an AII amacrine cell using data from whole-cell patch clamp recording. This model will be used to explore electrical response properties of AII amacrine cells.

Methods

Single-compartment Hodgkin-Huxley-type neural models were simulated in the NEURON environment. Morphological cell properties and passive membrane parameters, ionic reversal potentials and gating variables were adapted from published data. Three types of potassium and leak maximum conductance values were included in the parameter search. Whole cell patch clamp data were used to constrain the values of these conductances. Systematic parameter search was performed in NEURON-simulated voltage and current clamp experiments.

Results

Simulations lead to successful reproduction of resting membrane potential, potassium current-voltage relationships, and some spiking properties observed in vitro experimentally.

POPULATION CODES FOR TOPOGRAPHY IN THE ZEBRAFISH OPTIC TECTUM

Lilach Avitan[1], Zac Pujic[1], Ethan Scott[1,2], Geoffrey Goodhill[1,3].

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[3]School of Mathematics and Physics

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The visual system has been an attractive target for studying neural coding. However, this has so far been mostly in the context of problems such as decoding edge orientation from the activity of populations of edge-selective neurons in V1. Surprisingly, there has been little quantitative investigation of population coding in the topographic representation of visual space, perhaps because topography is traditionally thought of as a place code rather than a population code. To address this we perform functional imaging of topographic representations in the zebrafish optic tectum, a model system which permits non-invasive imaging of neural activity. Stimuli placed in different positions on an LCD screen, covering different areas of the zebrafish visual field, are presented to zebrafish larvae while performing confocal calcium imaging of tectal neurons loaded with fluorescent calcium indicator (OGB-AM1). Using a Bayesian framework we decode the visual topographic information from a large population of tectal cells, to examine the extent to which the spatial information in the stimulus is preserved in the tectum, and the role this plays in decoding.

EFFECTS OF ELECTROCONVULSIVE THERAPY STIMULUS FREQUENCY

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Introduction and Aim

It has been demonstrated by clinical research that variations in the electroconvulsive therapy (ECT) stimulus parameters can affect treatment efficacy and adverse effects. The objective of this study was to investigate the influence of stimulus frequency by using a high-resolution anatomically-accurate head model.

Methods

Image segmentation and finite element mesh generation were carried out using MRI scans of a healthy 35-year-old male subject. Regions representing passive volume conductors (skin, eyes, skull, and cerebrospinal fluid) were extracellularly coupled to an excitable neural continuum region representing the brain, which was used to simulate direct brain excitation induced by the ECT stimulus.

Results and Conclusions

Both 60 Hz and 90 Hz ECT stimuli managed to initiate an action potential (AP) at every cycle, whereas 120 Hz only initiated an AP at every other cycle; moreover, the subsequent AP generated with 90 Hz showed obvious fatigue (i.e., reduced overshoot) in comparison to the first two in the train.

In the 120-Hz model, of all the brain neurons that were able to be directly activated by a full cycle, none were activated by the second of two consecutive cycles. Hence for a stimulus of this frequency, the number of efficient pulses is likely to be only 50% of the total pulse number. With 90 Hz stimulus frequency, the efficiency of the second cycle is only around 10% of the first one in a two-cycle stimulus train. In addition, the slight reduction in the membrane potential overshoot for the third AP indicates that the activation pattern may be more complex if given a long stimulus train. In comparison, when a stimulus with 60 Hz frequency was delivered, the efficiency of the second cycle was almost 80% of the first. A similar trend was found in the regions of interest for all three frequencies.

Therefore, an ECT stimulus train with lower frequency appears the most efficient for cortical activation. This is probably due to the existence of the neuronal refractory period.

TRACKING PHYSIOLOGICAL CHANGES IN THE BRAIN USING A NEURAL MASS MODEL

Richard Balson[1,2,3], Dean R. Freestone[1,2,] David B. Grayden[1,2,4], Anthony N. Burkitt[1,2,4], Peter Blamey[4], Mark J. Cook[3,2]

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Introduction

At present, the mechanisms underlying seizures are not well understood. This has led to difficulties in developing effective treatments. However, new modelling techniques are being developed to provide insight into the mechanisms that cause seizures.

Aims

The aim of this research is to develop a method to track the changes that occur in the underlying physiology of the brain as it transitions between seizure and normal activity.

Methods

Artificial EEG is simulated using the neural mass model (Wendling et al. 2002). The model parameters are then assumed to be unknown for estimation purposes. An unscented Kalman filter (Voss et al. 2004) with the artificial EEG as the observations, is then used to estimate these model parameters.

Results and Discussion

The tracking of model parameters using the Kalman filter is achieved with model parameters initialised within a small range of their actual value. The tracking of model parameters with experimental and clinical data will provide insight into the changes that occur in the brain as a seizure is manifesting.

Reference

Wendling, F., Bartolomei, F., Bellanger, J., and Chauvel, P. (2002). Epileptic fast activity can be explained by a model of impaired GABAergic dendritic inhibition. European Journal of Neuroscience, 15(9):1499–1508.

Voss, H., Timmer, J., and Kurths, J. (2004). Nonlinear dynamical system identification from uncertain and indirect measurements. International Journal of Bifurcation and Chaos, 14(6):1905–1933.

Acknowledgements

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EFFECTS OF INTRINSIC NEURONAL PROPERTIES ON HIGH-FREQUENCY OSCILLATIONS IN INTERNEURONAL NETWORKS

F. Baroni[1,2], A.N. Burkitt[1,2], D.B. Grayden[1,2]

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Introduction

High-frequency oscillations have been observed in sensory and higher-order brain areas, and are believed to constitute a general hallmark of functional neuronal activation. Fast inhibition in densely coupled interneuronal networks has been suggested as a general mechanism for the generation of high-frequency oscillations. Certain classes of interneurons exhibit subthreshold oscillations, but the effect of this intrinsic neuronal property on the population rhythm is not completely understood.

Aims

This study aims to assess the influence of intrinsic subthreshold oscillations in the emergence of collective high-frequency oscillations, and to elucidate the dynamical mechanisms that underlie this phenomenon.

Methods

We simulate neuronal networks composed of either Integrate-and-Fire (IF) or Generalised Integrate-and-Fire (GIF) neuron models. While the IF model displays a purely passive subthreshold dynamics, the GIF model exhibits subthreshold damped oscillations. Individual neurons receive inhibitory synaptic currents mediated by spiking activity in their peers as well as noisy synaptic bombardment.

Results

We show that GIF networks display more prominent oscillations in a broad range of parameter space. We identify three factors that affect the influence of single neuron properties on synchronisation mediated by inhibition: i) the firing rate response, ii) the membrane potential distribution, and iii) the shape of Inhibitory Post-Synaptic Potentials (IPSPs). The presence of a restorative current in the GIF neuron results in lower firing rates and smaller deviations from rest in the membrane potential distribution, which would predict a lesser tendency towards synchronisation in GIF networks. However, the effect of IPSP shape typically dominates the dynamics: the presence of a depolarising component in the IPSP profile induces a coherent spike-mediated depolarisation across cells, and greatly fosters synchronous oscillations. Importantly, if inhibition is shunting instead of hyperpolarising, the effects of firing rate response and membrane potential distribution dominate, highlighting the context-dependence of the observed phenomenon.

Acknowledgements

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Neural spike sorting framework using selected wavelets coefficients and k-means clustering

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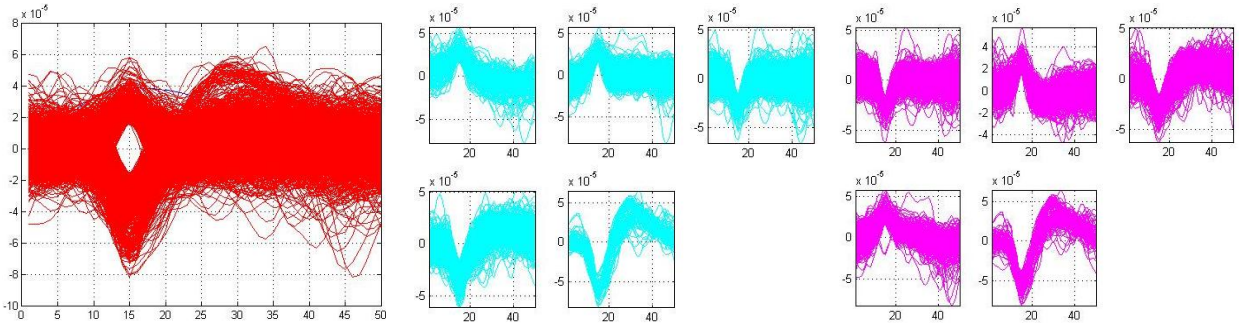
Introduction

In vitro multichannel recordings from neurons have been used as important evidence in neuroscientific studies to understand the fundamentals of neural network mechanisms in the brain. Accurate detection and sorting of neural activity waveforms becomes a key requirement for creating meaningful machine brain interfaces and to understand the working principles of neural networks. In this work we propose a unified framework for unsupervised neural spike clustering. Proposed framework exploits the features of wavelets scale-space representation and time-frequency localisation as well as multiscale principle component analysis to minimise the dimensionality of the raw data at different scales prior to clustering.

Method

Influenced by the technological and fabrication limitations, the number of microelectrodes is always significantly less than the number of cultured neurons on a micro electrode array (MEA) chip. Based on the commonly used MEAs, consisting of 60 micro electrodes; the neurons to microelectrodes ratio could be 60:10,000+, as shown in Figure 2 D. This leads to a unique communication paradigm, where a single microelectrode could detect and record signals from many surrounding neurons. Depending on the objectives of the investigation, neuroscientists are required to detect and sort these signals by assigning detected spikes to putative neurons with high degree of reliability, precision and robustness. As the neurons in a localised formation often produce action potentials of similar shape and size therefore in the presence of inherent background noise it becomes a challenge to map the waveform to individual neuron with high accuracy.

In this work we propose a unified framework for unsupervised neural spike clustering. Proposed framework exploits the features of wavelets scale-space representation and time-frequency localisation as well as multiscale principle component analysis to minimise the dimensionality of the raw data at different scales prior to clustering. The number of clusters are estimated using principle component analysis by selecting high variance components that cover certain percentage of total variance of the data. In 1, variance of 98% is selected. Clustering algorithms are applied on the selected wavelets coefficients that are selected globally based on their significance through the approximation and detail spaces.



(a) (b) (c)

Figure 1: (a) Raw neural spikes data; Clustering of neural waveforms with 98% variance using (b) Hierarchical clustering (c) K-Means

NOISE DRIVEN OSCILLATIONS: A COMPUTATIONAL MODEL OF BIMANUAL TAPPING

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Introduction

Rhythmic bimanual tapping is a paradigmatic example where close agreement has been established between theory and experiment. Both iso- and multi-frequency tapping has been modelled by two coupled oscillators operating at the movement frequency. These oscillators are thought to be instantiated by neuronal circuits but their biophysical implementation has not been established. Electrophysiological studies on tapping indicate that cortical activity operates at much faster time-scales. In particular, many studies found beta activity (15-30Hz) to be functionally relevant in motor performance. A central question is therefore how n:m coupling at the movement frequencies (1-3Hz) can arise from neural activity at timescales an order of magnitude faster.

Methods

In this study a model of coupled phase oscillators is investigated in its ability to yield multi-frequency coordination. In broad regions of parameter space, such systems robustly exhibit a form of winnerless competition known as heteroclinic cycles, characterized by transitions between partially synchronized cluster states. Whereas the individual phase oscillators operate at a fast time-scale, the switching between different cluster states evolves much slower. In a noise-free system a typical trajectory would stay for increasingly longer period of time near each state. In contrast, stochastic systems will settle on a periodic orbit with noise-dependent period lengths.

Results

Examples of rhythmic bimanual tapping are discussed showing n:m frequency coupling at the movement frequency that are linked to oscillatory neural activity at faster time-scales. The model agrees with empirical findings on bimanual tapping and provides an explicit implementation for nested cortical oscillations. On a physiological level, the phase dispersion of coupled oscillators can be interpreted as partially synchronized local inhibition.

A COMPUTATIONAL MODEL OF INTRINSIC SENSORY NEURONS OF THE GUT

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Introduction

Intrinsic sensory neurons (ISNs) of the enteric nervous system respond to stimuli such as muscle contractions, distortion of the mucosa and chemical changes in the lumen. ISNs form a recurrent network that probably drives many intestinal motor patterns and reflexes. ISNs express a large number of voltage and calcium gated ion channels. However, it remains unclear how the interactions between the different ionic currents can produce both normal and pathological behaviours.

Aims

To construct a detailed computer model of ISNs to allow a systematic investigation into how different ionic currents can influence the excitability of ISNs.

Methods

Our model was constructed using the NEURON simulation environment. The voltage and calcium dependent currents were modelled using a Hodgkin-Huxley-type formalism. The model includes several voltage-gated sodium and potassium channels, an N-type calcium channel, a big conductance potassium (BK) channel, a calcium dependent non-specific cation channel (I_{can}), intermediate conductance potassium (IK) channel, hyperpolarisation activated cation (I_h) channels and internal calcium dynamics. The model was based on data from the literature and our electrophysiological studies.

Results

The model reproduced the physiological observations of firing in response to multiple current pulses (250 pA 10 ms duration at 50 Hz, n = 8) or prolonged depolarising current pulses (50-350 pA for 500 ms), and responses to prolonged hyperpolarising current pulses. A sensitivity analysis for each conductance showed that I_h, IK, I_{can} and BK had the largest influence on the number of action potentials observed during a prolonged depolarisation. The model also predicts that changes to the voltage of activation for I_h have a large influence on the number of action potentials, but that changes to the time constant of activation for I_h have a minor effect. In conclusion, our model identifies how interactions between different ionic currents can influence the excitability of ISNs and indicates an important role for I_h and IK in disease states, such as inflammatory bowel.

STATISTICAL CRITERIA FOR ASSESSING PHASE SENSITIVITY OF VISUAL CORTICAL NEURONS

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Introduction

Neurons in primary visual cortex are often classified as either *simple* or *complex* based on the linearity of their response to spatial luminance contrast. In practice, classification is typically based on Fourier analysis of a cells response to an optimal drifting sine-wave grating. Cells are easily and objectively classified based on the *relative modulation* of their responses -- the ratio of the phase-sensitive response at the fundamental frequency of the stimulus (F_1) to the phase-invariant sustained response (F_0). Cells are classified as *simple* if $F_1/F_0 > 1$ and *complex* if $F_1/F_0 < 1$. However, Fourier analysis of spiking responses is sensitive to the number of spikes available – F_1/F_0 increases as the number of spikes is reduced. Here we describe a statistical basis for objectively assessing whether modulation of spiking responses is reliable, thereby adding a level of statistical certainty to established measures of phase sensitivity.

Methods

We developed a stochastic model of the spiking responses of visual cortical neurons presented with optimal moving sine-wave gratings. Spike arrival times are assumed to be independent and identically distributed random variables, we make no attempt to explicitly model the biophysics of spike generation (refractory period etc.). Nevertheless, the model is informative in demonstrating the importance of the number of spikes collected when using Fourier analysis. Moreover, the model provides a useful basis for objectively assessing the statistical reliability of modulated responses observed in real cortical neurons.

Results

Using our model of spiking responses we estimate, for a given number of spikes, the probability of observing a given level of response modulation and derive a statistical criterion for assessing the significance of modulation in observed spiking responses of real cortical neurons. This criterion accounts for the number of spikes recorded and allows differentiation of reliable phase-sensitive responses from those that appear modulated simply due to the limited data available. Our criterion adds a measure of statistical significance to the established metric of *relative modulation* (F_1/F_0) for assessing phase sensitivity. This is particularly powerful when fewer spikes are available, either due to low spike rates observed in some neurons even for optimal stimuli, or in experiments that specifically employ stimulus manipulations that alter the spike rate.

RETINAL GANGLION CELL PARAMETERS PREDICTING HUMAN PERFORMANCE IN A TWO-STAGE NEURAL SPIKING MODEL OF LUMINANCE INCREMENT DETECTION

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Introduction

Perimetry is a clinical test that measures non-central vision loss using circular luminance increment stimuli. Gardiner et al (Vision Res, 2008) developed a two-stage neural spiking model of perimetric stimulus detection that used parameters from animal neurophysiology to model signal propagation through the retina and visual cortex. Signal detection was based on firing rates in the visual cortex.

Aims

To determine parameters for modelled retinal ganglion cells (RGCs) that allow the model to predict human psychometric functions at two retinal eccentricities, accounting for changing size of perimetric stimuli.

Methods

Psychometric functions for stimulus detection were measured for two observers, at eight visual field locations (± 9, ± 9 and ± 15, ± 15) in a temporal two-alternative forced choice task. Stimuli were circular luminance increments of 0.43° and 1.70° diameter. Model psychometric functions were also computed using the same stimuli and task. RGC receptive field characteristics (difference-of-Gaussians model), maximum firing rate and centre-to-centre spacing were varied, and the psychometric functions produced were compared to the empirical data. The effect of RGC dysfunction was also modelled, and compared to data from glaucoma patients in the literature.

Results

Model RGC parameters were found that matched empirical detection thresholds across locations and stimulus sizes (mean difference [95% CI] for smaller stimulus -0.1dB [-0.5 to +0.3], for larger stimulus +0.3dB [0 to +0.6]). Similar to the study of Gardiner et al, empirical psychometric functions were steeper than those of the model, possibly due to unmodelled feedback factors (mean difference [95% CI] for smaller stimulus 1.0dB [0.9 to 1.1], for larger stimulus 1.9dB [1.7 to 2.1]). The RGC parameters were within the range found in electrophysiological studies of monkeys. Increasing RGC dysfunction caused modelled psychometric functions to flatten and thresholds to increase, consistent with the literature on glaucoma patients. The model will be useful in predicting the effects of RGC disease on perimetric thresholds.

CURRENT STEERING IN RETINAL PROSTHESES: TOWARDS ARBITRARY RESOLUTION

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Background

In Australia and other developed nations, Retinitis Pigmentosa, Age-related Macular Degeneration, and other retinal degenerative disorders have caused legal blindness in approximately 1 in 1115 individuals, with the potential to affect many more as our populations age. While there has been extensive research into finding pharmacological or surgical treatments for these conditions, to date none of these approaches have successfully reversed the progression of these degenerative disorders. However, while in these diseases the photoreceptors of the outer retina are lost, some electrically active neurons survive in the inner retina. It was shown in the early 1990s that these neurons can be driven by extracellular electrical stimuli to create a visual sensation in a blind individual – this prompted the development of retinal prostheses in numerous groups worldwide. Numerous different prostheses have been implanted in clinical trials with generally positive patient outcomes; however, the resolution of the restored sense of vision is not yet sufficient to realize a significant gain of function for blind individuals.

Aims

The field of retinal prostheses has largely been following the general trajectory of most electronics towards miniaturization, which tends to increase the resolution of the achieved sense of vision; however, there are several physical and electrochemical limitations on the resolution of the electrode-tissue interface. Advanced stimulation strategies may be able to achieve effective resolutions greater than the resolution of the electrode-tissue interface.

Methods & Results

Using a continuum computational model of the retina which expresses the Hodgkin–Huxley dynamics of the retinal ganglion cells (RGCs) (the ultimate targets of prosthetic stimulation), the authors demonstrate evidence that electrical stimuli which are individually too weak to induce activation of RGCs, when presented on adjacent electrodes simultaneously, may be able to selectively activate RGCs intermediate between the electrodes, effectively predicting that a “virtual electrode” stimulation strategy may provide higher resolution prosthetic vision.

PITCH RECOGNITION USING AUDITORY MODEL INPUT

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Introduction

Pitch has an important status among different aspects of sound stimuli because of the prominent role it plays in speech prosody and music melody. According to the rate-place theory of pitch perception, pitch is coded by the specific positions on the basilar membrane and auditory nerve that have maximum vibration or neuronal firing rate, respectively.

Aims

The aim of this research is to train rate-based perceptrons to assign a pitch label to input sound stimuli. Input features are extracted from an auditory model and the results are compared with measures of absolute pitch perception in humans.

Methods

A) *Database*: The database includes: (1) 1392 vowels generated with 29 fundamental frequencies (from G2 to B4 on the western musical scale) by the KLATT speech synthesiser; (2) 26 real sung vowels with 13 fundamental frequencies that are included in (1); and (3) 29 notes recorded from piano keys identical to the fundamental frequencies in (1).

B) *Feature extraction*: An auditory periphery model that converts sounds into inner hair cell activity is used to calculate the firing rates at 200 locations along the basilar membrane for sound stimuli of 100 ms duration. The rates are then averaged over time to generate a vector of 200 features for each sound.

C) *The model*: A single layer of perceptrons with a continuous log-sigmoid transfer function receives the 200 features and is trained by back propagation to assign a note (out of 29) to each input. 75% of the KLATT data is used during the training process.

Results

The network error reaches zero after 183 iterations of training. A correct pitch classification rate of 93.1% is achieved when the network is tested on the remaining KLATT test data. However, when tested on the sung vowel and piano data, pitch classification rates drop down to 11.5% and 24.1%, respectively. Classification errors are generally associated with octave errors and errors within one tone; these errors are also the most common errors among absolute pitch possessors when performing pitch identification tasks. When discounting octave errors and errors within one tone, the performances on sung vowels and piano notes are 65.4% and 74.4%, respectively.

Acknowledgements

Supported by ARC Discovery Grant DP1094830 and the Victorian Life Sciences Computation Initiative (VLSCI). The Bionics Institute acknowledges the support it receives from the Victorian Government through its Operational Infrastructure Support Program.

INFORMATION THEORETIC OPTIMISATION OF COCHLEAR IMPLANT ELECTRODE POSITIONS AND USAGE PROBABILITIES

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Introduction

Cochlear implants are neural prostheses that can restore hearing. An open question for the design of future cochlear implants is whether improved electrode designs that enable less current spread could be exploited to achieve improved hearing performance through the use of more electrodes than presently, or more precise positioning.

Aims

The overarching goal is to find the information theoretic channel capacity in a stochastic model of the electro-neural interface in cochlear implants from [1], under varying assumptions about the number of electrodes and their current spread. Channel capacity is defined as the maximum mutual information between an input random variable (choice of electrode) and an output random variable (defined as a function of active nerve fibres).

Methods

First we compare how many electrodes provide maximum mutual information when the electrode array is specified rather than optimised, under varying conditions. Channel capacity can be found for any channel model using either the Blahut-Arimoto algorithm or convex optimization, when the input positions are fixed. When the positions can vary, channel capacity can increase, and the optimisation problem in standard form is non-convex. However, the problem can be converted to a convex one, and using this approach allows us to relate the capacity-achieving input positions to the optimal positions of electrodes.

Results

For uniformly spaced electrodes, we show how the mutual information changes in the model of [1] as the distribution of nerve fibres change, and also study the variance in the optimal number of electrodes when nerve fibres have parameters that are randomly chosen. We also find the channel capacity-achieving input distribution for some specific cases, and relate this to the optimal electrode positions and the optimal probabilities of using them. Both the Blahut-Arimoto algorithm and convex optimisation lead to same channel capacity. Convex optimization shows more efficiency in finding capacity-achieving input distribution and further is useful in finding optimal placements of electrodes.

[1] M. D. McDonnell *et. al*. "A channel model for inferring the optimal number of electrodes for future cochlear implants," IEEE Transactions on Information Theory 56:928-940, 2010.

SENSITIVITY TO STATISTICAL STRUCTURE IN THE HUMAN BRAIN

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Introduction

We constantly look for patterns in the environment that allow us to learn its key regularities. These regularities are fundamental in enabling us to make predictions about what is likely to happen next. The physiological study of regularity extraction has primarily focused on repetitive sequence-based rules within the sensory environment.

Aims

Here we ask whether we implicitly encode non-sequential stochastic regularities, and detect violations therein.

Methods

We addressed this question using a novel experimental design and both behaviour and magnetoencephalographic (MEG) metrics associated with a response to pure tone sounds sampled from a Gaussian distribution.

Results

We observed that sounds in the tail of the distribution evoked a larger response than those that fell at the centre. Crucially, responses to physically identical outliers were greater when the distribution was narrower. Source reconstruction revealed a temporo-parietal network that encompassed areas associated with attention orientation to unexpected events.

These results show that humans implicitly keep tract of the mean and the uncertainty present in apparent random distributions of sensory events. We suggest that this sensitivity provides a computational basis for our ability to make perceptual inferences in noisy environments and to make decisions in an uncertain world.

INTERMITTENCY EXPLAINS VARIABILITY IN HUMAN MOTOR CONTROL

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Introduction:

Human behaviour is variable. A useful computational theory seeks to explain such variability in a parsimonious way. In the context of human motion control, variability has been explained by adding a specially tailored coloured noise process to an otherwise deterministic model. The experimentally well-established psychological refractory period (PRP) leads to a second form of variability: the variation of second-reaction time. The PRP has been explained by the concept of intermittency and a corresponding mathematical description of intermittent control has been proposed by the authors.

Aims:

We argue that the process of adding noise to a model does not, by itself, add any explanatory power to a theory of human motion-control variability. On the contrary, variation in central reaction times suggests a neuro-processing source and we argue that this variability is a necessary concomitant of the well-established psychological refractory period as expressed in our computational theory of intermittent human control. In particular, the hypothesis addressed in this paper is that the observed variability is due to intermittent control in which the intermittent control interval is variable, rather than to additive noise.

Methods:

Variability is most clearly shown when the response to periodic stimuli is non-periodic. For this reason, each subject has the task of controlling a precisely defined single-input-single-output system disturbed by a periodic signal with period T with some of the harmonic frequencies $f_i = i/T$ deleted. The system output is displayed on a screen and the system is controlled by means of a sensitive hand-held joystick. To make the task non-trivial, the controlled system is second order and unstable thus doing nothing is not an option for the subject. If both system and controller were linear, time invariant and noise-free, the measured response signals would be periodic with period T and the measured frequency spectrum would have Fourier components only at the excited frequencies. It follows that signals detected at non-excited frequencies are a consequence of variability.

Results and Discussion:

The frequency components derived from the experimental results could be fitted by either hypothesis. But the intermittent hypothesis provides a more parsimonious explanation. In contrast to explaining variability by additive random noise, explaining variability by random intermittency has three advantages: it has a natural, and potentially testable, physiological basis; it adds to the evidence for intermittency already accumulated and it provides a simple explanation for the observed increase of variability with signal amplitude.

We suggest that the variation in intermittent interval is due to event-triggering via a threshold. Future work will examine how variation in intermittent interval is related to threshold, signal and system properties and the insights of stochastic resonance theory may be relevant here.

EXPLORING AND VISUALISING THE FUNCTIONAL ROLES OF INTERNEURONS

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Introduction and aims

Interneurons are very diverse and are believed to stabilise and synchronise other neurons. Little is known, however, regarding their involvement in behavioural changes. This study aimed at investigating and answering the question "Do interneurons play a role in behavioural transitions?"

Methods

By performing three case studies, one *in silico* and two *in vivo*, and using visualisation techniques, this research studied the effect of interneurons on the network dynamics and their firing patterns during rat's behavioural changes. The *in silico* component was to study the complex spiking model [1], which is a complex network based on the "small world" theory, while the *in vivo* studies involved investigating datasets from awake rats performing navigational tasks and object recognition tasks.

Results

The *in silico* study showed that less inhibitory synaptic strength resulted in simpler dynamics, while increasing the strength caused chaotic behaviour. One *in vivo* study found that some interneurons fired less frequently when the rat was preforming the trained navigational task (fig. 1), while the other showed that there was an inverse change to the paired firing pattern of an interneuron and an excitatory cell when the rat switched behaviours.

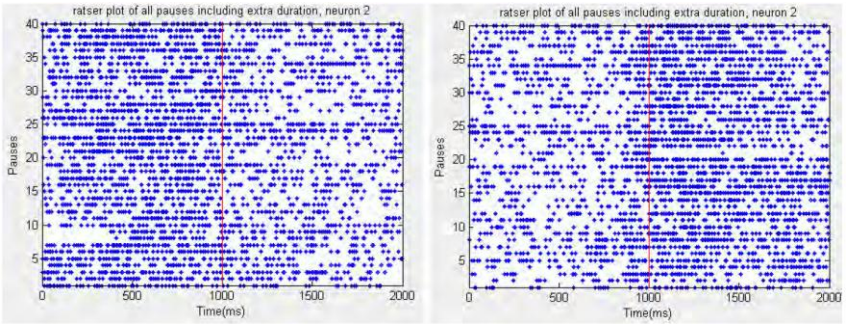

Fig. 1 The raster plot of spikes from an interneuron during the time that a rat experienced a transition of behaviours (marked by the red lines). Left: from the period before pausing to after having paused. Right: from the end of the pause duration to the period afterwards. This particular neuron showed a less dense firing pattern during pauses.

[1] P. Stratton, and J. Wiles. "Complex spiking models: A role for diffuse thalamic projections in complex cortical activity." *Neural Information Processing. Theory and Algorithms*: vol.6443, pp. 41-48. 2010

ASSOCIATIVE LEARNING AS AN EMERGENT PROPERTY OF SPATIALLY EXTENDED SPIKING NEURAL CIRCUITS WITH SPIKE-TIMING-DEPENDENT PLASTICITY

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Association of sequential events happening at different time moments is of fundamental importance for perceptual and cognitive functions. One of the important paradigmatic forms of such association is classical conditioning, in which the pairing of two subsequent stimuli is learned such that the presentation of the first stimulus (conditioned stimulus) is taken as a predictor of the second one (unconditioned stimulus). Most of the theoretical models proposed to account for classical conditioning have focused on individual neurons or synapses by assuming the presence of slowly decaying firing activity of neurons, which along with some synaptic plasticity such as spike timing dependent plasticity (STDP), enables associative learning between temporally separated events. However, the experimental evidence of such slowing decaying firing activity for associative learning is still inconclusive. Here, we present a novel account for the association based on the emergent properties of spiking neural circuits instead of individual neurons. Our proposal relies on two basic, known neurophysiological features of neuronal circuits: (1) lateral inhibitory coupling for neural circuits, and (2) spike timing dependent plasticity. In a two-dimensional, spatially extended spiking neural circuit with these features, we find that each event can be represented by an emergent spiking sequence in terms of a propagating pattern in the network, and that the interactions of the sequences are timing dependent. When the time interval between the two stimuli is small there is no association happening between the two sequences; this is mainly due to the lateral inhibitory coupling, which results in a repulsive interaction between two spiking patterns when they propagate and approach each other. However, when the interval is increased, association can occur; namely, the presence of the first spiking sequence is able to regenerate the second one spontaneously without the second stimulus present. Interestingly, the reversed case is not true, i.e., the presence of the second sequence along is unable to regenerate the first sequence. Note that the same temporal causality holds in the classical conditioning paradigm. Furthermore, we show that our network model with noise can reproduce and then account for the contiguity of classical conditioning as found in behavioral studies, which states that the successful association rate is a non-monotonic function of the time interval of conditioned and unconditioned stimuli.

INTRINSIC VERSUS EXTRINSIC INFLUENCES ON GROWTH CONE BEHAVIOUR

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Introduction

The guidance of axons to their targets during neural development depends critically on the growth cone at the axonal tip. However, little is known about the relative importance of intrinsic versus extrinsic cues for determining the trajectories and dynamic morphology of growth cones.

Aims

By analysing growth cone dynamics under a variety of different conditions, we aimed to determine quantitatively the relative importance of external factors (such as molecular gradients) in determining growth cone behaviour.

Methods

We performed timelapse imaging of over 200 growth cones in a variety of conditions. Images were captured at 1 minute intervals, giving a dataset of over 12,000 frames in total. Growth cone outlines were automatically traced and parameterised by 500 spatial coordinates. Principal Components Analysis was then performed on this space of shapes (eigenshape analysis), giving modes representing the principal directions of variation. How the projections of growth cones onto these modes varied with time and with external conditions was then examined.

Results

The four principal shape modes capture over 80% of the variance in shape, and split into modes representing turning without changes in area and modes representing changes in area without turning. Projections of these modes over time show characteristic oscillations independent of external conditions, indicating intrinsic dynamics of the cytoskeleton. However mode projections varied systematically with the parameters of an externally applied molecular gradient, indicating strong extrinsic effects. Thus growth cone behaviour represents a dynamic interplay between intrinsic and extrinsic influences.

SYNAPTIC LEARNING BEYOND STDP

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Introduction

Spike time dependent plasticity (STDP) describes only one aspect of the signalling requirements for learning (long lasting plasticity) at synapses. A fundamental signal for plasticity at excitatory synapses is the time course of the calcium concentration in the postsynaptic spine head. Differential timings of presynaptic and postsynaptic spikes do influence spine calcium concentrations and hence lead to STDP. More generally, spine calcium is the result of ongoing synaptic inputs and local cell excitability, which may or may not involve postsynaptic spiking. Further, plasticity induction is not instantaneous and proceeds over seconds to minutes. Biochemical pathways leading to long term potentiation (LTP) or depression (LTD) are separate and operate with different dynamics. Intense input over tens of milliseconds can initiate LTP, which is then induced over a period of seconds. LTD initiation requires seconds of moderately intense input. So both the intensity and duration of synaptic inputs determines plasticity outcomes.

Aims

Current experimental data and resultant models of synaptic plasticity based on spine head calcium provide a starting point from which to explore learning in neurons being driven by spatio-temporal patterns of synaptic input that are ultimately the product of animal behaviour. The hypothesis is that LTP enables the remembering of short, intense (important or novel) stimuli, while LTD leads to forgetting of stored patterns that are consistently being recalled out of context (no longer relevant). Recall within context or occasional spontaneous recall are protected from forgetting by being either above or below the threshold for LTD.

Methods and Results

A detailed computational model of synaptic input onto spines on a reconstructed hippocampal CA1 pyramidal cell is used to explore the hypothesis. An extension of the calcium-based plasticity model of Graupner and Brunel (PNAS 109:3991-3996, 2012) is used to induce synaptic changes on realistic time scales in response to spine head calcium levels.

Results show that synchronously-active groups of synapses undergo LTP or LTD or do not change depending of the number of coactive synapses and their frequency of stimulation. LTD induction requires longer than LTP, such that no change occurs even for many seconds of stimulation that does eventually lead to LTD if sustained for long enough (tens of seconds). Brief, intense stimuli to one group of synapses leads to LTP while coactive synapses on the same cell, receiving less intense activity, undergo LTD at the same time.

INFLUENCE OF DENDRITIC STRUCTURE ON RETINAL GANGLION CELL FIRING PATTERN: A MODELLING STUDY

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Introduction and Methods

Active dendrites play a critical role in shaping retinal ganglion cell (RGC) firing patterns. Morphologically-realistic models can quantitatively integrate anatomical information and biophysical principles and focus on how dendritic structures contribute to neural behaviour, which is quite difficult to achieve using experimental technique. In this study, realistic three-dimensional reconstructions of mice OFF RGCs were digitised and imported into NEURON. An extension of the Fohlmeister and Miller conductance-based model was used to simulate RGC biophysics. To isolate the contribution of dendritic tree, a set of models shared the common distribution and kinetics of ionic channels and differed only in their dendritic structures.

Results and Conclusion

We gradually disabled the dendritic tree by disconnecting the corresponding branch from soma in the computer-reconstructed RGC geometry, and then recorded somatic membrane potentials in response to various somatic current injections. The simulation results indicated that spiking frequency and first spike latency was progressively increased with less dendritic branching. Considering the dendrites have larger overall membrane surface area and far less active conductance compared with that in the soma, the high frequency ionic currents can be effectively filtered by dendrites, revealing their low-pass filtering properties. To isolate the contribution of dendritic bifurcation on action potential (AP) propagation, we recorded the AP backpropagation along one entire dendritic branch and one 'bald' branch without bifurcation respectively. The two results both indicated that when the soma was stimulated, APs were also evident in the distal dendrite. As APs propagated far from the soma, increasingly broadening and slowing kinetics occurred, as well as lower peak amplitude due to lower dendritic Na^+ and K^+ channel distributions. However, AP amplitude decreasing along the dendrite was significantly eliminated when less dendritic bifurcations were involved, indicating the influence of dendritic bifurcation on shaping the AP propagation. Our results indicate that in addition to their inherent biophysical properties, RGC dendritic morphology, including dendritic diameter, density and bifurcation can significantly influence the responses elicited, revealing their important contribution in neural encoding.

GAUSSIAN PROCESS METHODS FOR EVALUATING VISUAL MAP CHANGES FOLLOWING ABNORMAL VISUAL INPUT

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Introduction

Topographic maps of features such as orientation preference in the mammalian primary visual cortex provide a paradigm model system for examining the influence of the environment on brain structure. However experimental techniques to visualise these maps, such as optical imaging, suffer from high levels of noise. To improve upon the simple averaging procedures used up to now Macke and colleagues recently proposed a Gaussian process method for more accurately reconstructing maps. However in its original form this could not be applied to stripe rearing, one of the paradigm models of visual plasticity.

Aims

We aimed to generalise the Gaussian process method of Macke et al so that it could be applied to our data from stripe-reared cats, in order to provide a more rigorous assessment of the effects of stripe rearing on the structure of orientation maps.

Methods

Generalising the model required relaxing assumptions of the prior distribution used, taking into account changes in the covariance function and mean.

Results

The changes in different aspects of the fitted covariance and mean lead to multiple measures of orientation overrepresentation in maps from animals reared in stripes, which can be taken together to give an overall measure of overrepresentation, or analysed separately to tease apart the different contributions to the changes in map structure. Together these results expand the domain of applicability of Gaussian process methods for estimating cortical maps, and provide new insights into the nature of visual cortical plasticity.

A CABLE-INSPIRED FRAMEWORK FOR INVESTIGATING CALCIUM-BASED SYNAPTIC PLASTICITY IN NEURONAL DENDRITES

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Introduction

How the axons of cortical neurons learn to create neural circuits and adapt their corresponding synaptic strengths in an activity-dependent manner is still an intensely debated topic. Our current understanding has highlighted the important roles that both internal calcium concentration and the precise timing of pre-and post-synaptic action potentials play in altering, either by strengthening or weakening the strength of a connection between two neurons via a synapse located on the post-synaptic cells' spatially extended dendrite. Specifically, landmark studies have shown that synapses are strengthened when the generation of a post-synaptic spike proceeds the arrival of a pre-synaptic action potential, but if the temporal order is reversed then synaptic strength is weakened; where the degree of synaptic alterations can be quantified via an asymmetric temporal window of finite extent [6]. While other studies have illustrated distance-dependent changes in such a temporal window, including the sign of plastic change itself [1, 5]. Specifically, one study has made an attempt to quantify such location dependent changes of the temporal window by demonstrating that in distal regions of the apical dendrite, the extent of the temporal windows' depression component was broader when compared to observations taken from a proximal region of the dendrite (close to the cells' soma) [1]. Currently, the potential mechanism of such a distance-dependent effect is unknown and quantitative theoretical approaches to explore plasticity within dendrites is under developed.

Aims

This study aims to lay the foundation for a mathematical framework better equipped to handle spatial effects in neuronal dendrites and use it to investigate synaptic plasticity model outcome and investigate how the influence space alters such outcomes. Our aim is to develop such a framework, based upon cable theory, which also incorporates the spatial diffusion of calcium. Our framework relies upon extending a novel cable-based approach called ionic cable theory to include the reaction and diffusion of calcium [4, 2, 3]. Using this framework, in conjunction with the standard calcium based plasticity model, we investigate how the spatially extended nature of dendrites influences plasticity outcomes.

Results

Simulations show how the dissipation and low-pass filtering of signals in neuronal dendrites naturally leads to alterations in synaptic plasticity windows as those seen in experiments, specifically being able to reproduce the distance-dependent broadening of the depression component of STDP window.

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SYMMETRY-BREAKING INDUCED BY REFRACTORINESS IN BALANCED SPIKING NEURAL CIRCUITS

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Introduction

If neurons receive a sufficiently large current within a certain time window, they emit stereotyped action potentials; this is followed by a refractory period during which they are unable to fire again. It has been found that neurons in many regions of the brain have centre-surround coupling, i.e. short-range excitatory connections and longer-range inhibitory connections. It has recently been shown that a simple neural circuit with centre-surround coupling can form localised patterns [1]. These localised patterns can propagate if the refractory period is large enough [2]. We investigate whether this kind of pattern-formation could occur in a biologically more plausible model.

Methods

We use a uniform array of excitatory and inhibitory model neurons in a 4:1 ratio. The connection between any two neurons is given by distance-dependent exponential functions, the constants of which depend on whether they are excitatory or inhibitory. Each neuron is governed by a conductance-based leaky integrate-and-fire equation. The conductances are determined by a homogeneous, constant external input and the sum of postsynaptic potentials (PSPs) from afferent neurons. PSPs are represented by a dual exponential function with characteristic rise- and decay-times.

Results

Localised patterns occur for a refractory period $2 \leq \tau_{ref} \leq 6$ ms, with internal chaotic activity. For $\tau_{ref} \geq 6$ ms, the localised patterns may propagate over long distances and exhibit behaviour such as repulsive collisions and annihilation, similar to that found in [1]. Propagating localised patterns can also be induced for lower conductance decay times $\tau_{decay} \leq 2$ ms. The type of wave interactions which occur depend on the number of propagating patterns and how they approach one another, providing testable predictions. Time series of PSP variability are constructed. Our results suggest that symmetry-breaking induced by characteristic times is the mechanism underlying pattern propagation. This may explain the generation of similar cortical waves that have been observed experimentally during spontaneous activity [3].

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GENERALISED REWARD-MODULATED SPIKE-TIMING-DEPENDENT PLASTICITY: A MODEL OF OPERANT CONDITIONING OF CORTICAL NEURONS

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Operant conditioning refers to an individual modifying its behaviour based on some consequence of that behaviour, such as reward or punishment. Experiments have shown that changes in the firing rates of individual neurons in the motor cortex of rhesus monkeys can be elicited through operant conditioning. In these experiments, the monkeys were presented with feedback based on the firing rate of a neuron (measured from an implanted electrode) and rewarded for increasing that rate. Underlying this behavioural learning is plasticity at the synaptic level. Reward-modulated spike-timing-dependent plasticity (RSTDP) has been proposed as such a model of synaptic learning and has previously been used to analytically explore the results of these biofeedback experiments. In RSTDP, neuromodulatory signals (such as dopamine) modulate the amplitude of the learning window.

We introduce a generalisation of RSTDP where, unlike classical RSTDP, the long term potentiation and depression parts of the learning window (LTP and LTD) are modulated separately by a neuromodulatory signal. Our model is based upon the way that neuromodulators have recently been experimentally observed to modify STDP.

Using the Poisson neuron model, we analytically investigate the conditions under which generalised RSTDP generates the results seen in the biofeedback experiments. We compare it to classical RSTDP and use numerical simulations with leaky integrate-and-fire (LIF) neuron models to support our findings.

We show that, for both uncorrelated and correlated inputs and with both additive and multiplicative weight dependencies, the generalised RSTDP model is able to account for the change in the firing rate of a neuron in a way that classical RSTDP is not. We also show that this is only possible when the reinforced neuron is able to exhibit short inter-spike-intervals. For a LIF neuron, this corresponds to being in a fluctuation-driven regime where it receives a balance of excitatory and inhibitory input. Our results suggest that for RSTDP to be able to change the firing rate of neurons through a biofeedback or reinforcement-learning scenario, the potentiation and depression parts of its learning window must be modulated separately.

HOW PARKINSON'S DISEASE AFFECTS CORTICAL INFORMATION FLOW: A MULTISCALE MODEL

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Introduction and Aims

The basal ganglia play a crucial role in the execution of movements, as demonstrated by the severe motor deficits that accompany neuronal degeneration in Parkinson's disease (PD). Since motor commands originate from the cortex, an important functional question is how the basal ganglia influence cortical information flow, and how this influence becomes pathological in PD.

Methods

We developed a hybrid neuronal network/neural field model. The neuronal network consisted of 9900 event-driven rule-based neurons, divided into 15 excitatory and inhibitory cell populations in the thalamus and cortex. This model was then embedded in a neural field model of the basal ganglia-thalamocortical system, including the cortex, thalamus, striatum, subthalamic nucleus, and globus pallidus. Both network and field models have been separately validated in previous work, with both shown to produce realistic firing rates and spectra. Two field models were explored: one with parameters based on data from healthy individuals, and one based on data from individuals with PD. Spikes generated by these field models were then used to drive the network model. We then explored the effects that these drives had on information flow in the network.

Results and Discussion

Compared to the network driven by the healthy field model, the PD-driven network had significantly lower mutual information between neurons. It also had a significantly fewer spike bursts, indicating reduced neuronal assembly formation. Networks driven by both healthy and PD field models showed peaks in coherence at 20 Hz and its harmonic at 40 Hz, but these features were much more pronounced with drive from the healthy model. The PD-driven network showed widespread reductions in Granger causality. In particular, the reduction in Granger causality from the main "input" layer of the cortex (layer 4) to the main "output" layer (layer 5) may explain some features of Parkinsonism, especially bradykinesia. We then replaced the field drive with uniform white noise, and found major changes in information flow, as well as in more basic measures such as power spectra. These results demonstrate that the brain's large-scale oscillatory environment, represented here by the field model, strongly influences the information processing that occurs within its subnetworks. We conclude that spiking network models need to be driven by realistic inputs, not simply white noise.

FAST LEARNING IN BAYESIAN SPIKING NEURONS

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Introduction

Bayesian Spiking Neurons (BSNs) provide a probabilistic interpretation of how neurons perform inference and learning. Online learning in BSNs typically involves maximum-likelihood expectation-maximisation (ML-EM) based parameter estimation, which is slow and limits the potential of studying networks of BSNs.

Aims

To improve the computational efficiency of online learning in BSNs in order to make the study of networks of BSNs more tractable.

Methods

A new online learning algorithm, Fast Learning (FL), is presented and compared to the benchmark online ML-EM algorithm typically used for learning in BSNs. Large numbers of simulations with different true parameter combinations are performed to assess the robustness of the FL and ML-EM algorithms to uncertainty in the initial parameter estimates. Simulation run times are also compared.

Results

FL runs increasingly faster than the benchmark ML-EM as the number of inputs to a BSN increases (e.g. 22.6 times faster for 20 inputs), and also provides reasonable convergence performance that is robust to initialization of parameter estimates that are far from the true parameter values.

ESTIMATING AXONAL MYELINATION USING MAGNETIC RESONANCE IMAGING

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Introduction

There is increasing interest in quantifying the distribution of magnetic relaxation times in each voxel of the brain, underpinning emerging techniques such as myelin water imaging used to study multiple sclerosis [1].

Aims

The purpose of this work is to extend the Bayesian algorithm in [2] to model stimulated echoes, which will improve the accuracy of the estimated multicomponent T_2 distributions and the corresponding myelin water fraction.

Methods

Experiments were performed on a 4.7T Bruker BioSpec small bore MRI scanner fitted with a cryogenically cooled surface coil. A multi-echo sequence with 24 echoes was run on a sample with agar gel and a sheep optic nerve fixed parallel to the transverse plane. The slice thickness was 1mm with a field-of-view of 6.4mm×12.8mm and a 64×128 matrix. Data was processed with the Bayesian algorithm using both the multi-exponential model (assuming ideal 180° flip angles) and the multicomponent model with stimulated echo compensation.

Results

The myelin water fraction from the Bayesian algorithm exhibits similar values along the length of the nerve despite the presence of stimulated echoes (Fig. 1 left). Conversely, the simple multi-exponential model only produces reasonable results in the region where the flip angle is close to ideal (Fig. 1 right). The relatively large myelin water fraction in the nerve region compared to the surrounding gel is expected, and suggests that the proposed technique is suitable to study neurodegenerative diseases.

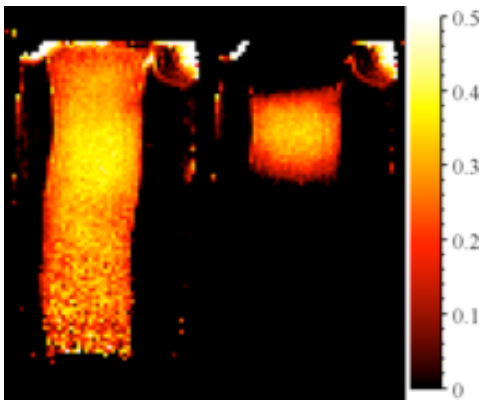

Fig. 1 The myelin water fraction of an optic nerve (left) with and (right) without stimulated echo correction.

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MULTIVARIATE CONSTRUCTION OF EFFECTIVE COMPUTATIONAL NETWORKS FROM OBSERVATIONAL DATA

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Introduction

Effective network inference from multivariate time-series data seeks to find a minimal circuit model that can reconstruct the activity patterns contained in this data. Ideally, effective network inference would be: made using model-free techniques; capture non-linear, multivariate, directional relationships; handle small amounts of data, and be statistically robust. The information-theoretic measure *transfer entropy* is becoming widely used for this purpose [2], however it is focussed only on *univariate* source-destination interactions.

Aims

We aim to extend transfer entropy based inference of effective networks using *multivariate* techniques. Specifically, we aim to: *capture* collective interactions where results in a target are due to more than one source variable (synergies); *eliminate* spurious connections for correlated sources (redundancies); and *avoid* combinatorial explosions in the number of source combinations evaluated.

Methods

We introduce a new method [1] for effective network inference which appropriately considers multivariate source interactions, addressing the above requirements. For each destination node in the network, the method identifies the set of source nodes which can be used to provide the most statistically significant information regarding outcomes of the destination, and are thus inferred as those source information nodes from which the destination is *computed*. This is done using incrementally conditioned transfer entropy measurements, gradually building the set of source nodes for a destination conditioned on the previously identified sources.

Results

We apply our method to various synthetic models of dynamics on networks (e.g. coupled Gaussian and random Boolean dynamics), and demonstrate the utility of the method in revealing significant proportions of the underlying structural network given only short time-series of the network dynamics, particularly in comparison to other methods (i.e. univariate transfer entropy analysis).

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MODELLING GLAUCOMOUS RETINAL GANGLION CELLS

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Introduction

Glaucoma is the second leading cause of irreversible blindness worldwide. Histology shows that the morphology of the retinal ganglion cells (RGCs) alter prior to cell death due to glaucoma. Currently, it is not clear how these morphological changes affect human vision. It is critically important to the future development of neuroprotective strategies for glaucoma to understand the effects of cellular alterations prior to cell death.

Aims

This project aims to understand how RGC morphological changes affect electrophysiological properties of the cells, and ultimately vision, – a key step in the development of tests for cellular abnormalities in human early glaucoma.

Methods

The membrane dynamics are described using the Hodgkin–Huxley equations. Simulations are conducted in NEURON environment using morphologically realistic multicompartment models. Soma and dendrites diameters in healthy cells are reduced as described in Weber and Hartman 2005 to replicate glaucomous RGCS. The effect of the changed morphology in glaucomous cells was investigated on the following passive and active electrophysiological cells properties: resting membrane potential, input resistance, threshold for spiking, spike widths and maximum spiking rate. Published experimental data from a primate retina was used to validate model predictions.

Results

Our study shows that to replicated published electrophysiological properties of glaucomous RGCs, ion channel concentrations have with changes in morphology. In particular, sodium and leak channel concentrations have to decrease in different proportions in the soma and in the dendrites.

ELECTROPHYSIOLOGICAL VARIATIONS IN RETINAL GANGLION CELLS: EFFECT OF MORPHOLOGY

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Introduction

Retinal ganglion cells (RGCs) are the sole output neurons of the retina. They convert synaptic input from inner retinal neurons into signals that carry visual information to the brain. RGCs display differences in their morphology and intrinsic electrophysiology. We extend our previous single compartment cell models of ON and OFF RGCs to more biophysically realistic multicompartmental cell models, and investigate the effect of cell morphology on intrinsic electrophysiological properties.

Aims

The aims of this study are to characterise the ionic currents and to distinguish the morphological differences that explain the behaviours of ON and OFF RGCs.

Methods

The membrane dynamics are described using Hodgkin–Huxley equations. Three currents proposed to shape the responses of RGCs are included in the model: the persistent sodium, hyperpolization activated, and low voltage activated (LVA) calcium currents. A subset of published patch-clamp data from isolated intact mouse retina is used to constrain the model, and another subset that characterises OFF RGCs is used to validate the model. 200 morphologically distinct, biologically realistic ON and OFF RGCs are simulated with various densities of ionic channels in different morphological neuron segments. The results are compared with the experimentally known properties of RGCs.

Results

Our study shows, through simulation, that not all morphological types of RGCs exhibit the phenomena described in recent experiments. Comparisons of outputs from different cells indicate that the RGC morphologies that best describe recent experimental results are ones that have a larger soma and a larger ratio of soma to total surface area. We show the presence of subthreshold oscillations, burst firing, and spontaneous activity in the absence of synaptic input in OFF RGCs, and the absence of such in ON RGCs. Our model predicts that the differences between ON and OFF cells intrinsic electrophysiology are based on the presence of LVA calcium current in OFF cells and absence of such in ON cells.

SLOW BISTABILITY IN FLUCTUATION DRIVEN MODELS OF CORTICAL NETWORKS WITH MULTIPLEX NEURONAL CONNECTIVITY

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Introduction

Although directed random graph models are frequently used successfully in modeling the population dynamics of networks of cortical neurons connected by chemical synapses, experimental results consistently reveal that actual network topologies are complex, and vary for different classes of neurons. This suggests that the random network assumption is unrealistic and that when simulating population dynamics in networks with multiple classes of neurons, it is necessary to employ directed network models that are the union of subnetworks with distinct topological structure—such networks are called *multiplex networks*.

Aims

We aimed to establish whether the balanced excitation and inhibition frequently observed in small cortical regions, and in models of them, can disappear in otherwise standard neuronal-scale models of fluctuation driven dynamics, solely due to changing the network topology whilst not changing any in-degrees. We also introduce *structurally-defined neuron classes*, where neurons with the same physiology and in-degrees are sub-classified according to the abundance of two- and three-edge directed motifs in which they participate.

Methods

We simulated a network of 4000 excitatory and 1000 inhibitory neurons, each modelled as a leaky integrate-and-fire cell with conductance synapses based on the difference of exponentials model. Each neuron was driven by an independent generated external Poisson spike rate of 3.0 spikes/ms. Realistic parameter values lead to neurons that fire sparsely with high ISI variance, and thus *cannot be modelled accurately as oscillators*.

Four network topologies were compared, each with fixed in-degrees (1000 total inputs per cell, 800 from excitatory cells, 200 from inhibitory cells): (i) random, with fixed in-degree; (ii) deterministic forwards-backwards ring-lattice; (iii) randomly rewired forwards-backwards ring-lattice (7.5% of cells had the originating neuron for their incoming inhibitory synapses replaced by a randomly chosen inhibitory cell); (iv) a modular excitatory network where each neuron had a higher probability of connecting to neurons in its own module then outside it; all synapses involving inhibitory neurons as for the random network.

Results

Our simulations revealed that the multiplex connectivity in the rewired ring lattice gives rise to bistable population dynamics reminiscent of so called cortical up and down states (see, e.g. Haider & McCormick, Neuron, 2009). The bistability is due to neurons in a single structurally-defined neuron class switching slowly between sparsely firing and highly active states. Our results suggest that models and simulations should take into account both multiplex complex structure and that it may be important to look for structurally-defined neuron classes within the same physiological class in connectome data obtained in future experiments.

AUDITORY TEMPORAL PROCESSING

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Introduction and Aims

Electrical stimulation of the auditory system is now a clinically accepted method of providing sound perception for people with severe to profound deafness. The electrical stimulus is usually applied to the auditory nerve (cochlear implants - CIs), but in a subgroup of patients where this is not feasible the stimulus is applied to the cochlear nucleus (auditory brainstem implants - ABIs) or the inferior colliculus (auditory midbrain implants - AMIs). Since the perception of temporal information in a speech signal is crucial for speech understanding, differences in the central auditory processing of temporal information may underlie the poorer outcomes for ABIs and AMIs compared to CIs. The aim of this study was to apply a model of central temporal processing to psychophysical data from CI and AMI subjects to understand the effect of stimulation site on transmission of temporal information.

Methods

Four experiments were conducted with AMI and CI subjects to measure the following: effect of interpulse intervals on detection thresholds and loudness; temporal modulation transfer functions; effect of duration on detection thresholds; and forward masking decay.

Results

The CI data were consistent with a phenomenological model that based detection or loudness decisions on the output of a temporal integration window, the input to which was the auditory nerve response to each stimulus pulse. AMI data were consistent with a neural response that decreased more steeply compared to CI stimulation as the pulse rate increased. The AMI model required an integration window that was significantly wider (decreased temporal resolution) than that for CI data, the latter being well-fit using the same integration window shape as derived from normal-hearing data. These models provide a useful way to conceptualise how stimulation of central auditory structures differs from stimulation of the auditory nerve and to better understand why AMI users have difficulty processing temporal cues important for speech understanding.

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Recognizing Pitch and Roughness

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Introduction and Aims

Roughness is perceved at amplitude modulation (AM) frequencies between about 20 and 150 Hz. The upper end of this range is close to the maximum possible AM frequency that could be detected by loudness integration mechanisms, since the minimum loudness integration window for human hearing is about three ms. Recent neurocognitive modelling and behavioural research points to a role for recognition mechanisms based on templates of specific loudness in the early stages of auditory processing. In particular, a spectral recognition mechanism based on template matching has been described and implicated in pitch processing. Roughness due to the co-modulation of unresolved high order harmonics overlaps with the lower frequency range for pitch perception, and so may also provide pitch cues. This paper presents a recognition mechanism for roughness based on spectrotemporal template matching. The paper also presents pilot behavioural data that validates the model's predictions for the pitch perception of unresolved harmonic complexes in the absence of spectral cues.

Methods

The computational model was implemented in Matlab. Musicians and non-musicians traced the perceived pitch contour for a series of harmonic complexes with fundamental frequencies that increased in 1 Hz steps from 80 to 160 Hz, and were band pass filtered between 1,500 and 3,000 Hz to remove spectral cues from lower order resolved harmonics. These contours were compared to predictions of the temporal recognition model.

Results

The temporal recognition model with memory templates at semitone intervals predicted that pitch would oscillate by around 1/3 of an octave while steadily increasing as the stimulus fundamental frequency increased. Since only musicians could be expected to have this density of templates, the model was also run with templates at four semitone intervals. In this case the model predicted that pitch perception would increase in a few large steps. Pitch contours drawn by musicians and non-musicians for the same stimulus qualitatively matched these predictions, with musicians producing substantially more oscillations in their pitch contours. These results suggest that learning music involves the formation of spectrotemporal memory templates for commonly occurring musical sounds at specific pitches.

MEAN FIELD FORMALISM FOR ELECTRICAL STIMULATION OF NERVE FIBRE BUNDLES

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We describe a novel theoretical framework for modelling electrical stimulation of unmyelinated nerve fibre bundles. The electrical properties of a bundle of nerve fibres are derived from first principles using a mean field approach. This connects the microscopic properties of the fibres, such as membrane impedance and fibre diameter, with the macroscopic properties of tissue conductivity and permittivity.

Two mathematically equivalent formulations of the equations are given, corresponding to a volume conductor model and a bidomain model (bidomain models use a continuum description of the extracellular and intracellular space coupled by a cellular membrane). Both formalisms allow the calculation of spatiotemporal maps of quantities of interest including the extracellular potential and the sub-threshold membrane potential. The membrane potential comprises two components: a longitudinal mode, corresponding to current flow along the axon, and a transverse mode, corresponding to current flow across the axon.

A novel aspect of the volume conductor model is that the conductivity tensor is non-local, in the sense that Ohm's law relates the extracellular current density to the electric field at distant parts of the tissue via spatial convolution with the conductivity tensor. This accounts for flow of extracellular current via the intracellular space. This effect is not accounted for in standard volume conductor models.

An illustrative example of a point source electrode in an extensive fibre bundle is given to demonstrate the application of the framework and highlight difference to existing approaches.

The benefit of the mean field approach is that it provides a self-consistent way of modelling electrical stimulation of fibre bundles, linking current flow through tissue on a macroscale with axon polarisation on a microscale: both effects are determined by the microscopic properties and structure of the fibres.

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A CONSTRUCTIVE APPROACH FOR MODELING AXON PACKING IN WHITE MATTER

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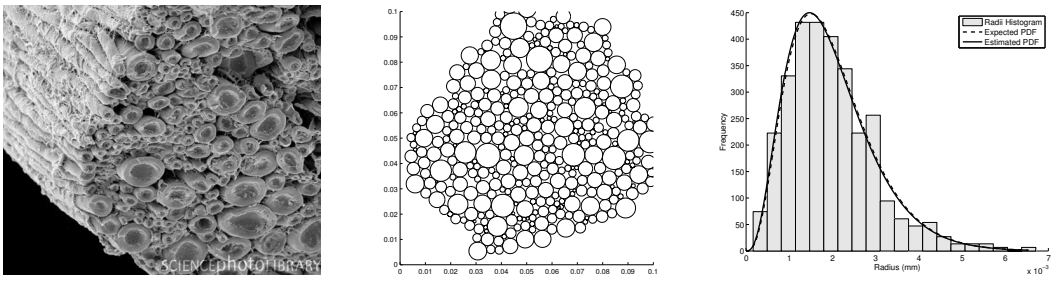
Introduction: The morphology of axons in the brain makes an important contribution to the measured diffusion weighted Magnetic Resonance signal in white matter. Although it is known that axons are encapsulated within nerve bundles in white matter, the geometrical distribution of axons inside nerve bundles has not been fully exploited in modelling the related diffusion weighted signal. In this work, we present an algorithm based on a constructive packing method to simulate models of the axon distributions. The resultant axon packings can be used later to investigate the Brownian motion of water molecules within the fiber tracts and related accumulated phase distributions. The method is controlled by parametric features of the tissue and statistical analysis is performed to compare the results with empirical data.

Method: Given a desired set of model parameters, our method starts by considering an axon with random radius derived from desired statistical distribution, proceeding in spiral layers to place further axons iteratively. The coordinates of new axon at each iteration are calculated using the coordinate information from adjacent axons in the same layer and the previous layer. The iterations continue until satisfying the desired number of axons calculated from the given radii distribution.

Results: A histological electron microscopy of a nerve bundle is depicted in Figure 1 (a), showing the actual packing of the axons. An example result of the algorithm is demonstrated in Figure 1 (b), showing the simulated axon packing in a nerve fibre. In order to measure the accuracy of the method, desired gamma distributions of axon radii are considered and compared with the resultant distributions as shown in Figure 1 (c).



(a) (b) (c)

Figure 1: (a) Histological scanning electron micrograph (SEM) of a freeze-fractured section through a bundle of myelinated nerve fibres (Photo credit: Steve Gschmeissner/Science Photo Library), (b) Simulation result using constructive approach, (c) PDF of the expected (dashed line) and simulated (solid line) radii.

Conclusion: We have proposed a novel constructive method for simulating the placement of the axons in nerve bundles and have compared the results with empirical data. The accuracy of the method and the effect of different model parameters on the results have been investigated, showing a high degree of consistency of the method with the desired characteristics of nerve bundles.

OPTIMAL RECEPTOR LOCATIONS FOR AXONAL CHEMOTAXIS

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Introduction

Chemotaxis, namely detecting and following chemical gradients, plays a crucial role in many biological systems. However, gradient detection is constrained by several sources of noise, including stochastic fluctuations in receptor binding. Recent work has addressed the computations required to infer gradient direction optimally based on noisy measurements of binding events. However, how the receptors should be configured over the surface of the detector to make this optimized inference as accurate as possible is unknown.

Aims

The aims of the study are to quantify the expected accuracy of chemical gradient estimation supported by particular configurations of receptor locations on axonal growth cones, and to predict empirical configurations by assuming that estimation is optimized.

Methods

We formulate this problem in Bayesian terms in order to examine how the optimal receptor configurations depend on the sensing device's prior distribution over both the gradient and the background concentration. We evaluate the Fisher information matrix as a function of configuration, and, by optimizing various quantities stemming from this matrix, show how to arrange the receptors to minimize the expected (asymptotic) uncertainty of the gradient estimate.

Results

The configurations that optimize the Fisher information depend in interesting and surprising ways on the form of the external gradient and the range of concentrations. We find that receptors cluster around discrete locations on the surface of the growth cone, and that the number of clusters depends sensitively on the concentration range.

COMPUTING INFORMATION STORAGE FOR INPUT-DRIVEN SYSTEMS

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Introduction

Information theory and the framework of information dynamics [2] have been used to provide tools to characterise complex systems. In this case, the characteristic elements of computation are information storage, information modification and information transfer.

Information dynamics can also help to get a "wholistic" understanding of input-driven systems such as neural networks [1]. In this case, however, this means that we do not distinguish between the system and the input to the system throughout the experiment. This is an important for example for biological systems which perform non-trivial computations and also retain a short-term memory of past inputs.Many other real world systems like cortical networks are also heavily input-driven, and application of these tools may not necessarily lead to intuitively interpretable results.

Aims

The aim of our work is to extend the measurements used in the information dynamics framework for input-driven systems. With input-corrected information storage we hope to better quantify system behaviour, which will be important for heavily input-driven systems like artificial neural networks to abstract from specific benchmarks, or for brain networks where individual components cannot be tested in isolation or with arbitrary input data.

Methods

Using the existing framework of information dynamics, we derive a measurement of input-corrected information storage that is suitable for input-driven dynamical systems. In our method, effects of the input on the system are conditioned out, resulting in a method to describe what is known about a system independent of its particular input.

Results

Input-corrected information storage quantifies the contribution of the system to information storage alone, e.g., for a neural network a measure of difference to a pure feedforward unit driven by a particular input. We can show that, for simple systems, the computed quantities correspond with intuition of how much information storage these systems actually exhibit.

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ROLE OF MULTI-CELLULAR COLUMNS IN A MODEL OF CORTICAL SEQUENCE LEARNING

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Introduction

Hierarchical Temporal Memory (HTM) is a recently proposed conceptual model of cortical learning, inference and prediction [1]. It attempts at modelling the principles of computation used by the neurobiology (as opposed to implementing the actual neurobiological findings). We focus on an existing implementation that is based on a simplified model of cortical pyramidal cells where dendritic computation is crucial. The cells are organised in a region of cortical columns. Inspired by experimental evidence of cortical organisation, cells belonging to the same column share the feedforward connections of proximal dendrites, whereas every cell in the column receives an independent set of lateral connections via distal dendrites. The region learns by making use of structural plasticity to strengthen and weaken the permanence of synapses, as well as to remove and create new synaptic connections [2].

Aims

We investigate the effect of varying the size of cortical columns (in terms of number of cells) on the performance of a cortical region in a sequence learning task. In addition, we study the structural connectivity patterns of cells within a region after learning, as well as how they relate to neurobiological evidence of the structure in the neocortex.

Methods

We have computationally reproduced a small section of cortical region involving 400 cortical columns. Two variations of the number of cells per column are compared. Our first simulation investigates the influence of two cells per column in a learning task. Simulation II explores the effects of reducing the size of cortical columns to one cell. Both cortical configurations were trained over the same sequence of grayscale images.

Results

Results from our simulations suggest that the presence of more than one cell per column significantly aids the disambiguation of repetitive input patterns within complex training sequences. We also find that the learning process converts what is initially random connectivity into a highly modular network topology. Our results provide evidence that supports the potential role and importance of structural plasticity in cortical regions.

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REPRESENTATION AND INFERENCE IN THE VESTIBULAR SYSTEM

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Introduction

Evidence suggests that perception and action are based on neural mechanisms that explicitly represent uncertainty. The vestibular system provides an opportunity to develop and test circuit-level models of perception as causal inference. In this system the utility is clear, the goal of inference is unambiguous, and sources of relevant data are known.

Aims

We model the vestibular brainstem as a system that performs sequential inference using spikes to build a representation of head state. We make no assumptions about coding, but simply ask what can be inferred from observing spikes in vestibular sensory afferent neurons.

Methods

The posterior density of head state given a spike is the normalized product of the head state likelihood given a spike and the prior head state density. The prior is *not* subjective, it is the posterior given previous spikes. It follows that if we know the likelihoods we can calculate what the brain can infer about head state at spike arrival times (Without needing to know if or how it does this). In a probabilistic framework these likelihood functions are a complete representation of information transmission in the vestibular nerve, and are a prerequisite for any computational model of causal inference in the vestibular system. We estimated head state likelihood functions at spikes times of semicircular canal afferent neurons from data recorded from bullfrogs on a servo-controlled turntable. By treating secondary neurons as points in a map of head state space we constructed a neural particle filter model of how the posterior head state density could be represented by spikes in the brainstem.

Results

Individual sensory spikes transmit information about head state. They can be treated as measurements of state characterised by likelihood functions, visualised as maps of firing intensity over head state space. The model shows that spiking neurons could use these measurements to construct a representation of the posterior density of head state that is instantaneously updated at spike times. Experimental tests of the model are in progress. In this framework ‘firing rate’ and ‘firing time’ do not encode anything; they are simply the rate and times at which sensory neurons measure states. A correct model of information transmission by spiking sensory neurons may be important for effective design of prosthetic systems designed to replicate natural signals in the vestibular nerve. The model may be useful in other sensory systems.

POTENTIAL FOR OPTIMISING DEEP BRAIN STIMULATION THROUGH TREMOR QUANTIFICATION

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Introduction

Deep Brain Stimulation (DBS) of sub-cortical areas such as the Subthalamic Nucleus (STN) can be used to minimise tremor in patients who do not respond to pharmaceutical therapies. DBS devices utilise a constant-current or constant-voltage biphasic pulse train. The amplitude, pulse duration and frequency of the stimulus can be adjusted manually. At present, the stimulus is set based on clinical observation of tremor and side-effects. An ideal stimulus is difficult to determine due to the large number of parameter permutations. Furthermore, little is known about how these parameters interact with each other.

Aim

We aimed to develop an objective measure of tremor severity and determine how varying stimulus parameters influenced the tremor.

Methods

We recruited six patients with Essential Tremor into the study. They had DBS implants in the Posterior Subthalamic Area (PSA). Tremor was recorded using four electromagnetic motion sensors placed on the patients' elbows and wrists. Each DBS parameter was adjusted systematically and for each trial patients were asked to hold their arms outstretched in front while counting to ten followed by a nose-finger-nose exercise. A range of objective measures including tremor amplitude, frequency, velocity and C90 volume were derived from collected motion data.

Results

All objective measures were correlated with each other except for frequency which showed an inverse relationship in some cases. These measures showed that only a small range of parameters were useful in giving benefit to the patient. It is difficult to determine which measure provided the most valuable tremor severity information. We expect these measures will be combined to give a single measure or index. Such a measure could be used to optimise DBS parameter settings and patient outcomes in the near future.

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THE EFFECTS OF SYNAPTIC REGULATION ON A NEURAL FIELD MODEL OF EPILEPSY

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Introduction

The spread of seizure-like behaviour through the cortex is facilitated not only by hyper-excitable, hyper-synchronous neuronal population firing, but by overcoming the regulatory mechanisms of brain activity, such as feedback, feed-forward and surround inhibition. These control mechanisms attempt to stabilise such pathological behaviour. We suggest an additional network regulatory mechanism in the form of a 'shunting' effect based on endogenous synaptic regulation of input currents, an important neurophysiological function whose mechanism is difficult to incorporate in macroscopic models of brain dynamics.

Methods

A time delayed thalamo-cortical neural model is modified to include conductance-based synapses which are more realistic compared to previous models which use current-based synapses. This has a significant effect on the overall network dynamics. A nonlinear summation of the synaptic currents is introduced that incorporates local feedback from the membrane potential and an 'active' time constant that varies inversely with the amount of input or drive to the network. The result is a more physiologically detailed description of the synaptic current produced by post-synaptic potentials. The dynamics of the new model are examined via a bifurcation analysis of a set of delayed differential equations, and an exploration of relevant bifurcation parameters. These pertinent parameters are: the external input, network balance, reversal potentials and thalamo-cortical coupling. These parameters are highly relevant to epileptogenesis. The results are compared to those of the original model with current-based synapses and the differences interpreted physiologically.

Results

The system dynamics and oscillatory properties of the new model differ significantly from the previous model. This is largely due to the 'shunting' effect of synaptic regulation, which acts as a network control mechanism. In particular, within the same parameter ranges for the network input and balance, the oscillatory behaviour is markedly different given the same conditions. In the new model oscillatory behaviour is modulated via non-synaptic mechanisms, for example by changes in the ionic micro-environment, namely the reversal potentials.

Conclusion

Both synaptic and non-synaptic regulations of network behaviour are important neuro-physiological functions which fundamentally effect synaptic transmission and homeostatic balance. They should not be neglected in realistic neural models of epilepsy, particularly when examining seizure initiation, spread and termination.

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FUNCTIONAL DESIGN OF THE ELECTROSENSORY SYSTEM IN SHARKS

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Introduction

Studies of electrosensory prey detection in sharks and their relatives have led to interesting models of target-selective adaptive filtering mechanisms in cerebellar-like circuits in the vertebrate hindbrain. However, despite the fact that in normal operation shark electroreception involves using a spatially distributed set of sensors to locate moving targets in space, little attention has been paid to spatial information processing in this system. Accurate models of measurements provided by sensors are a prerequisite for designing and analyzing intelligent systems that utilize sensor data. The aim of this work is to construct a realistic model of spatial and temporal information acquisition by a shark's electrosensory system.

Methods

We previously developed a detailed three-dimensional model of *Squalus acanthias*, including the peripheral electrosensory system. Here we present a finite element model of the sense organs, the ampullae of Lorenzini, with realistic geometrical and electrical properties. We have simulated the electrical response characteristics of the organs in the shark, in a virtual environment containing prey-like electric sources, and uniform electric fields resembling motion-induced and other fields encountered in the ocean.

Results

The canal provides a low-resistance pathway to the apical surface of the sensory epithelium, functionally parallel to a high-resistance pathway through skin and connective tissue to the basal surface. This causes most of the voltage between the canal pore and the interior of the head to appear across the receptor epithelium. The effect is larger for longer canals, but the gain provided by this mechanism even in the longest canals, several centimetres long, is insufficient to translate the reported behavioural sensitivity, in the order of nanovolts per centimetre, into the millivolt-level signals necessary for transduction in the receptor cells. Restricting current flow to the tip of the receptor cell kinocilium creates a high voltage gradient at that point. The organs are directionally sensitive for both uniform and dipole sources, responding best to uniform fields parallel to the canal, and to dipole sources along a line extending parallel to the canal. From this model and neurophysiological data we can estimate the firing intensity of primary sensory afferent neurons as a function of prey parameters in the predator's reference frame. Then using the 3D reconstruction we can build a complete set of likelihood functions for prey parameters given the sense data.

Conclusions

In contrast to recent suggestions that the canal has strong capacitive properties, we have shown that hypersensitivity of sharks' electric organs can be explained by an electrical lensing effect in which biomaterials having different impedances are arranged to focus electric fields onto molecular receptors. The performance of shark electroreceptors appears to be near-optimal within the constraints imposed by physics and information theory. Our maps of the measurements made by the electroreceptor array provide a foundation for further studies on mechanisms of perception, behavioural choice and sensorimotor control using *Squalus* as a model.

SCALE-FREE DYNAMICS IN CORTEX AFTER PERINATAL HYPOXIA

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Following perinatal hypoxia, neonatal cortex follows a stereotypical recovery sequence that includes a period of "burst suppression", during which the EEG exhibits sudden, irregular fluctuations of highly variable size and shape. Clinical outcome depends critically on this phase, ranging from complete recovery to permanent cognitive or motor disability and even death. Here we analyze the statistical properties of burst suppression in neonatal EEG recordings. We show that fluctuations in burst size exhibit long-tailed power law distributions up to a remarkable six orders of magnitude. Despite this immense variability, their average shape at all temporal scales can be rescaled to a near universal template. Deviations from universality include a flattening of fluctuation shapes at long time scales and the expression of leftward or rightward asymmetry. These features are consistent with the phenomenon of crackling noise that arises in disparate physical systems such as crumpling paper, ferromagnetic materials subject to a slowly increasing external field, and earthquakes, all of which exhibit scale-free bursty events. Here, as in studies of crackling noise, the average shapes shed light on the underlying mechanisms, which are still poorly understood during burst suppression. Using simple phenomenological models, we show how changes to the average shapes arise from different forms of state-dependent damping. Statistical analysis of the variability and average shapes of bursts holds promise for new diagnostic opportunities in this critical clinical window and will inform future biologically-detailed models.

BRAIN NETWORKS: ROLES OF STABILITY AND GEOMETRY IN DETERMINING CONNECTIVITY

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When viewed as networks of nodes, brain networks are characterized by short path lengths, high clustering, significant modularity, and other characteristic graph-theoretic measures. Historically, much analysis of brain network structure has involved postulating networks of various types —random, regular, small-world, modular, hierarchical — and determining which have similar network measures to those found experimentally.

In parallel with pure graph-theoretic considerations, physical constraints, such as minimizing brain volume, wiring, and metabolism, have also been advanced. More recently, requirements of brain stability have been invoked to further constrain possible structures. Similarly, it has been suggested that the embedding of brain networks in physical space must be taken into account to properly account for their properties.

Here, we show that considerations of stability and the geometric embedding of networks in the convoluted cortex are sufficient to account for most observed properties of experimentally determined brain networks, without introducing a priori modularity, hierarchy, or other connectivity structure.

Comparison with detailed structural MRI data shows close agreement with a model that has the same connectivity properties at every point in the cortex — i.e., homogeneous, isotropic connectivity. This agreement encompasses both mean network measures and their distributions. Links between spatial variations of measures and convolution structure are explained and confirmed by comparison with data.

These results imply that physical effects strongly constrain brain network structure and corresponding connection matrices, and explain why the latter have forms that are commonly interpreted as hierarchically modular. We find that homogeneous connectivity on the 2D convoluted structure of the real cortex is sufficient to account for most features of the observed connectivity. Given the specificity of function in particular brain areas, inhomogeneous structure is undoubtedly present, but our findings imply that it is superposed on a primary, approximately uniform architecture.

CONSEQUENCES OF MODULARITY AND CYCLIC MOTIFS FOR THE COLLECTIVE DYNAMICS OF A CORTICAL NETWORK MODEL

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Introduction

The structure of the networks formed by neurons is an active area of research [1, 2]. Understanding of the consequences of complex neuronal network structure on its dynamics may lead to advances in understanding biological information processing, biomimetic artificial intelligence and further the understanding of disorders which are hypothesised as arising from abnormal connectivity structures. Complex topological structure has been observed to be present in brain networks on a variety of scales and is proposed to have functional consequences. Network topology has been shown to affect the collective dynamics that arise in networks [1, 2], yet quantitative approaches to exploring such effects are under developed for neuronal networks.

Aims

Simulations are used to examine the affect of network modularity on hyperexcitability, a dynamical network state associated with epileptic seizures. In addition to this, the affect of introducing an over abundance of three node loops or 'cyclic motifs' within these network modules are considered. This work aims to shed light on whether such topological structure could be a plausible cause of hyperexcitability. Specifically, we aim to provide some initial insights into the consequences of hypothesised connectivity consisting of a combination of modular structure and cyclic motif abundance on network population rates.

Methods

The impact of introducing an over-abundance of cyclic motifs within a modular structure is investigated using simulations of a spiking network model of both excitatory and inhibitory integrate and fire neurons with balanced excitation and inhibition. Comparisons are made with a null-hypothesis random network in terms of time-dependent population and individual firing rates, in response to noisy external drive.

Results

Simulations illustrate how these topological features may contribute to the hyperexcitable state. This study also represents early work in the analysis of directed networks with multiple neuron classes, balance of excitation and inhibition and complex topology, all of which are present in neuronal networks [1].

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A MODEL OF RETINAL GANGLION CELL AXON ACTIVATION THRESHOLDS FOR SUPRACHOROIDAL RETINAL PROSTHESES

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Introduction

Retinal ganglion cell (RGC) axons run between RGCs and the optic disc, presenting a problem for the localisation of phosphenes with retinal prostheses. Ideally, an electrode should not activate axons of distant RGCs while it is activating proximal RGCs. The aim of this study is to estimate thresholds for the activation of axons by suprachoroidal electrodes for a range of stimulus parameters.

Methods

We construct a series of models, from simple to more complex and realistic. (1) A single axon embedded in a homogeneous medium; this is a classic model with well-known results. (2) A model that incorporates several retinal layers with unequal conductivities. (3) An anisotropic bi-domain model representing a bundle of parallel axons separated by thin extracellular space.

Finite element modelling and analytic methods are used to solve the models with parameters typical for suprachoroidal retinal prostheses.

Results

Our model predicts a marked threshold asymmetry with respect to the polarity of the biphasic stimulus (anodic-first or cathodic-first), while experimental studies report a reverse asymmetry, which is also less pronounced. Further, we find that the thresholds predicted by the model for direct axonal activation exceed the thresholds observed experimentally by at least a factor of two, and typically ten. These are indications that another mechanism may be responsible for the activation.

Conclusion

Our findings suggest that, for suprachoroidal prostheses, the requirement of localised neural activation places only a loose constraint on the stimulation strategy.

Acknowledgements

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OPTIMIZED DENDRITIC MORPHOLOGIES FOR NOISY INPUTS

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Dendrites are the cellular protrusions of neurons receiving the majority of synaptic inputs. We investigated the structure - function relationship of the dendrites of model neurons optimized for input order detection of stochastic inputs. For this purpose, we used an inverse method based on a genetic algorithm. In this method, via iterative test and selection steps, the genetic algorithm finds an dendritic structure as good as possible for a user-selected neural computation. In a previous study, we generated model neurons optimized for reacting strongly to two groups of synaptic inputs occurring in one, but not the reverse temporal order. In the current study, we added both temporal noise (synapse activation times) and spatial noise (synapse placement) to this computational task.

We observed that the model neurons which were exposed to a more noisy input generally had smaller dendritic trees. We explain this finding by the fact that for input-order detection, sampling from more varied responses is advantageous.

ANALYTIC APPROACH TO DETERMINING THRESHOLD VALUES FOR EPIRETINAL VISUAL PROSTHESES

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Introduction

Retinal implants are neuroprosthetic devices that can restore vision to people who have lost their photoreceptors. Estimating threshold currents for activating retinal ganglion cells (RGCs) in epiretinal prostheses is an important factor in designing a successful implant. We have developed an analytic method that predicts the required threshold current for epiretinal stimulation of RGCs.

Method

A three-layer model is used consisting of the vitreous, nerve fibre layer, and an anisotropic layer representing other cell types in the retina. Assuming that a point source is located in the vitreous, we solve Poisson's equation in three dimensions for the vitreous and Laplace's equations for the other layers. The nerve fibre layer is modelled by novel equations that describe how the extracellular current density depends non-locally on the extracellular electric field due to the passage of current from distant points through the extracellular space. Appropriate boundary conditions are applied to find solutions to the partial differential equations. Finite element analysis is employed to illustrate the validity of the solutions.

Results

The analytic expression derived for the nerve fibre layer response to electrical stimulation was used to estimate the current threshold when the stimulating electrode was moved parallel and perpendicular to the nerve fibre layer. A nonlinear least-squares optimisation technique was used to estimate the unknown parameters of the model in order to match the model result to experimental threshold data for activation of rabbit RGCs available in the literature.

Conclusions

The three-layer model presented here captures most of the qualitative behaviour of nerve fibre layer response to extracellular electrical stimulation and, therefore, can be used for designing visual prostheses.

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A DYNAMICAL PATTERN THEORY OF NEURAL FIRING VARIABILITY

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Introduction

Cortical neurons in the living brain usually fire highly irregular spike trains during both spontaneous and evoked activity. There are currently two leading theories to explain the mechanisms behind this variability: one requires a precise balance between excitatory and inhibitory synaptic inputs so that a neuron's membrane potential can be modelled as a random walk; while the other requires temporally synchronized synaptic inputs, which produce highly variable spike trains in real neurons, but have not yet been explained theoretically. These theories capture different and limited aspects of neural firing variability as observed experimentally and have not yet been comprehensively reconciled.

Aims

This study aims to show that spatially extended, coherent dynamical patterns at the network level provide a novel way of bringing together the theories of balanced excitation/inhibition and synchronized inputs in order to explain single neuron variability in the context of the larger neural circuit. The theory presented here provides a mechanism for coherent structures to exist in a neural network even if individual neurons appear to behave chaotically, enabling complex and precise neural processing to take place.

Methods

This study introduces a spatially extended, two-dimensional spiking neural circuit which reproduces a range of observed biological features and can display localized, dynamical patterns of firing neurons in the network and highly variable spike trains in individual neurons. This is extensively analysed both numerically and mathematically.

Results

When excitation and inhibition are approximately balanced (as in the balanced inhibition theory), localized dynamical patterns self-organize in the network. These naturally produce inputs to neurons in the network that are transiently synchronous in time and space. The synchronized input theory has previously recognised that this form of input can produce spiking variability consistent with in vivo cortical neurons, but has never been able to explain how this synchronization can arise. In this way, the network model encapsulates both the balanced and synchronous input theories to explain cortical neuron firing variability in the framework of localized dynamical patterns.

THE MESOSCOPIC MODELING OF BURST SUPPRESSION DURING ANAESTHESIA

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The burst suppression pattern is well recognized as a major class of generalised electroencephlagram (EEG) waveform abnormality and is evoked by a variety of anesthetic agents, as well as by a range of pathophysiological processes. While the electroencephalographic phenomenon and clinical implications of burst suppression have been studied extensively, the physiological mechanisms underlying its emergence remain unresolved and obscure.

In this regard mean field models describing the rhythmogenesis of the EEG may be able to provide important insights. To date such models have been shown to offer plausible explanations for a range of EEG phenomena that include the resting (or spontaneous) alpha rhythm, spectral changes induced by anaesthetic agents and the emergence of epileptiform activity. However these models have not been systematically investigated regarding their ability to produce patterns of burst suppression activity under physiologically plausible parametric changes.

Here we show that one well known mean field EEG model, the Liley model [1], while unable to produce burst suppression unmodified, is able to produce burst supression-like behaviour during modeled anaesthesia by the addition of a number of mean-field driven slow systems ostensibly at macroscopic (thalamic driving), mesoscopic (slowed cortico-cortical conduction) and microscopic (activity dependent synaptic resource utilisation).

The Liley model with the presence of multiple timescales is shown to exhibit burst suppression under physiologically plausible parameterisations for anesthesia with a wide variety of bursting behavior observed. The implications of such dynamics for a more complete physiological understanding of the EEG and the mechanisms that serve to modify ongoing brain activity necessary for purposeful behaviour and consciousness will be discussed.

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MODELING SELECTIVE ATTENTION IN AN INSECT VISUAL NEURON

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Whether considering a lion focused on a single zebra within a panicked herd, or a dragonfly capturing flies amidst swarms of prey and conspecifics, each animal selects a single object amongst distracting stimuli. Little is known about the neuronal mechanisms that allow animals to accomplish this 'attentional' task. Diverse evidence from functional imaging and physiology to psychophysics, highlights the importance of 'competitive selection' in attention for vertebrates, artificial intelligence and even in fruitflies. Although direct neural correlates for such attention are scarce, we have recently demonstrated responses from an identified dragonfly visual neuron, the 'centrifugal small target motion detector' (CSTMD1), that perfectly match a model for competitive selection within the limits of neuronal variability (r^2=0.83). Responses of CSTMD1 to individual moving targets differ in both magnitude and time course depending on location of the target within the cell's receptive field. However, responses to *two* simultaneous targets almost always match those elicited by one of the two targets acting alone. Successive repetition of stimulus pairs over variable sizes, separation and contrasts all elicit responses equivalent to single targets, regardless of whether the 'winner' is the stronger stimulus if presented by itself. Here we examine *winner-takes-all networks* as putative components of the small target detection system, considering biologically plausible implementations and how they might contribute to the physiological responses of CSTMD1. By examining such competitive selection models we gain insight into how the pre-synaptic elements to CSTMD1 could be arranged to permit the 'absolute' encoding of a single target in a multiple target environment.

LOW DIMENSIONAL MODEL OF BURSTING NEURONS

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Introduction

A computationally efficient, biophysically-based model of neuronal behavior is presented; it incorporates ion channel dynamics while preserving simplicity by representing only one slow and two fast ion currents. It is capable of a wide variety of dynamics observed *in vivo*. The model is a hybrid of two classical models of neuronal bursting, the Rose-Hindmarsh and Wilson models.

Aims

We aim to explore a simple bursting neuronal model capable of reproducing a range of dynamical behaviors commonly observed in physiological recordings of neuronal cells. Simple models are tractable and computationally efficient. Such models can provide a bridge between the cellular dynamics of single-neuron models and mean-field models that model whole population of neurons.

Methods

The model was derived from the four dimensional Wilson model of neocortical neurons. We replaced the two slow equations with a single slow current similar in form from the Hindmarsh-Rose neuron model. Our model equations were simulated by a classical Runge-Kutta routine and dynamical properties were explored in parameter space.

$$C\frac{dV}{dt} = -g(V)(V-V_1) - g_R R(V-V_2) - H + I_{ext}, \quad (1)$$

$$\frac{dR}{dt} = -\frac{1}{\tau_R}[R - R_\infty(V)], \quad (2)$$

$$g(V) = \nu_0 + \nu_1 V + \nu_2 V^2, \quad (3)$$

$$R_\infty(V) = 0.79 + r_1 V + r_2(V-V_3)^2 \quad (4)$$

$$\frac{dH}{dt} = -\frac{1}{\tau_H}[H - g_H(V-V_h)]. \quad (5)$$

Results

The equations are shown to provide a wide array of physiological dynamics in terms of spiking patterns, bursting, subthreshold oscillations, and chaotic firing. The model does not require the artifical resets required in integrate-and-fire neurons.

Analysis shows that, despite its simplicity, the model is capable of simulating an extensive range of spiking patterns. Several common neuronal behaviors observed *in vivo* are demonstrated, depending on model parameters. These behaviors are classified into dynamical classes using phase diagrams whose boundaries in parameter space prove to be accurately delineated by linear stability analysis.

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