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Analysis of signal fluctuations of Cortical Spreading Depression: Preliminary findings

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ABSTRACT

In this work, we apply Detrended Fluctuation Analysis (DFA) to study the dynamics of electrical cortical activity in rats during the phenomenon of Cortical Spreading Depression (CSD), as well as the periods before and after this phenomenon. The characteristic of CSD is reduced electrical activity that occurs and spreads in the cerebral cortex after the application of electrical, chemical or mechanical stimulus. Our results show that the electrocorticogram signal shows long range temporal correlations and scaling behavior, except during the pre-CSD burst phase (significant increase of amplitude provoked by stimulus).

1. Introduction

Physiological time series such as interbeat heart intervals, stride intervals, electroencephalogram etc., show highly irregular behavior, characterized by non-stationarity and nonlinearity, with fluctuations that occur at all time scales. Over the past decade, various methods derived from statistical physics were applied on physiological signals under healthy and pathological conditions. These studies show that under healthy conditions fluctuations in physiological signals exhibit long range power law correlations and that certain pathological states may cause alterations in this scale invariant (fractal) correlation property [1–13].

Cortical Spreading Depression (CSD) is a self-propagating wave of cellular depolarization characterized by a reversible suppression of neural bioelectrical activity for a period of minutes. It can be induced by local mechanical, chemical or electrical stimulus, and propagates from stimulated site to adjacent regions of the brain with a velocity 2–3 mm/min. Roughly 15–20 min after the stimulus, the recovery of initial state is observed [14–16]. This phenomenon was observed and described for the first time in 1944 by Brazilian researcher Aristides Leão [17] while studying the electrocorticogram (ECoG) of experimental epilepsy in anesthetized rabbits at the Harvard Laboratory of R.S. Morison. The underlying mechanism of CSD involves changes in distribution of ions (K^+, Ca^{2+}, Cl^-) between intracellular and extracellular compartments [18–22],

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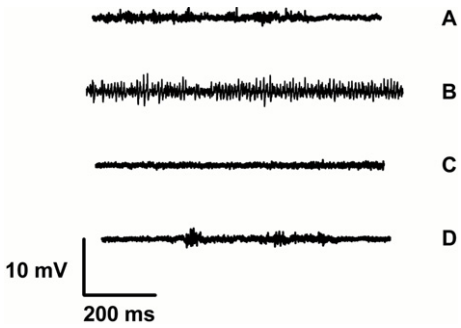

Fig. 1. Segments of temporal series of ECoG signal (A) before the stimulus, (B) the burst phase, (C) during the CSD and (D) after the CSD.

excitory compounds as glutamate [23,24], and other excitory transmitters [25,26]. It is accompanied with increase of glucose utilization and O_2 consumption. The recovery of CSD depends on metabolism [14], and this phenomenon has been observed in almost all gray matter regions of central nervous system: cortex, cerebellum, olfactory bulb, thalamus, retina and spinal cord [17,27–31]. It has been studied in vitro and in vivo in various animal species [14], and humans [32,33]. There is evidence that CSD is related to some clinical disorders such as migraine, epilepsy, cerebral ischemia, intracerebral hemorrhage during head injury, and transient global amnesia [34].

Besides a significant amount of experimental and theoretical work (several mathematical models and computer simulations were developed to model wave propagation, pathophysiology and cellular mechanisms associated with Spreading Depression (SD) [35–39]), the underlying mechanisms of this phenomenon and its role in clinical disorders are still not completely understood [34,40,41]. In this work we apply Detrended Fluctuation Analysis (DFA) to study scaling properties of the fluctuations in ECoG signal which was recorded during the phenomenon of Cortical Spreading Depression induced in cortex of rats. We calculate the DFA exponents, which indicate the presence of long range correlations, and compare scaling properties of different phases of the process.

2. Materials and methods

2.1. Experimental details

Five adult male albino Wistar rats (provided by the Department of Nutrition of the Federal University of Pernambuco) of age between 90–120 life-days were submitted to the recording of the Cortical Spreading Depression of the cortical electrical activity. Under anesthesia with a mixture of urethane+chloralose (1.000: 40 mg/kg, ip), the head was secured in a stereotaxic device (David Kopf, USA), and a 10 mm diameter trephine hole was drilled on the skull, over the sensory-motor cortices of both hemispheres. We used a glass micropipette (10 μm tip diameter; Borosilicate, World Precision Instruments) filled with NaCl 2M as electrode record.

The CSD was evoked by applying a cotton ball (1–2 mm diameter) soaked with KCl (2% solution) at a cortical point located 5–10 mm from the recording site.

During the ECoG record the rectal temperature was measured continuously and kept around $36.5 \pm 1\,^{\circ}$C by means of a heating pad, placed under the animal. The extracellular records were carried through glass microelectrodes. The recording microelectrode was an Ag–AgCl with input resistance of 20 MΩ placed in the parietal region in the sensory-motor cortex about 1.5 to 2.5 mm anterior and 1 to 2 mm lateral to bregma.

A second electrode was placed on the nasal bones and had served as common reference (reference electrode) to the recording electrodes. The signals of ECoG had been amplified and filtered at 3 KHz for the spontaneous activity. The ECoG was digitalized by a continuous period of 2 h through an analog–digital converter (DIGIDATA 1322, Axon Instruments Corp.) in a personal computer. The CSD was provoked, after 25–30 min of recording, by 1 min chemical stimulation with 2% KCl as described above.

2.2. ECoG data analysis

Fig. 1 represents segments of temporal series of the ECoG signal: (A) control, before the stimulus, (B) burst phase, which is characterized by a significant increase in amplitude (compared by Leão with tonic–clonic epileptic seizure [17]), (C) during the CSD, and (D) after the CSD. The duration of burst phase in all cases was from 3 to 5 s while the other intervals lasted between 2 and 10 min.

To quantify time correlations in these intervals we use Detrended Fluctuation Analysis (DFA), introduced by Peng et al. [42]. This method is a modified root-mean-square analysis of random walk, and was successfully applied to detect long range correlations in DNA sequences [1], non-stationary time series of physiological signals [8,9,11–13,43–49], weather