

## Correlation dimension of EEG slow-wave activity during sleep in narcoleptic patients under bed rest conditions

Raffaele Ferri<sup>a,\*</sup>, Salvatore Pettinato<sup>b</sup>, Lino Nobili<sup>c</sup>, Michel Billiard<sup>d</sup>,  
Franco Ferrillo<sup>c</sup>

<sup>a</sup>*Sleep Research Center, Department of Neurology, Oasi Institute, Via Conte Ruggero 73, 94019 Troina, Italy*

<sup>b</sup>*Department of Neurophysics, Oasi Institute, Via Conte Ruggero 73, 94018 Troina, Italy*

<sup>c</sup>*Center for Sleep Medicine, Department of Motor Sciences, University of Genoa, Genoa, Italy*

<sup>d</sup>*Sleep and Wake Disorders Unit, Gui-de-Chauliac Hospital, Montpellier, France*

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### Abstract

The calculation of the correlation dimension ( $D_2$ ) was applied to the study of the profiles of EEG slow-wave activity in nine narcoleptic subjects and nine sex- and age-matched control subjects who, following a baseline night recording, were maintained on 16 h of diurnal sleep deprivation and, thereafter, submitted to a 32-h bed rest protocol. The reversibility test allowed us to reject the null hypothesis that the time series considered in our study were generated by a static transformation of a linear Gaussian random process. Similarly, all profiles showed a positive largest Lyapunov exponent. Finally, the computation of  $D_2$  showed an average value of 5.27 (0.68 S.D.) in normal controls and 4.05 (1.49 S.D.) in narcoleptic patients ( $p = 0.067$ ). Four of the narcoleptic patients showed values of  $D_2$  lower than 4, this was never observed in the normal controls ( $p = 0.0294$ ). This study indicates that the mechanism of sleep–wake regulation in narcolepsy shows a somewhat lower degree of complexity as compared to normal controls. In particular, these data seem to confirm the already suggested different and simpler coupling between the homeostatic process of sleep regulation and the circadian and ultradian drives to sleep that occurs in bed rest condition in this disease. © 1999 Elsevier Science B.V. All rights reserved.

**Keywords:** Slow-wave activity; Power-spectra; Sleep regulation; Deterministic chaos; Stochastic process; Correlation dimension; Narcolepsy

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\*Corresponding author. Tel.: +39-935-9361111; fax: +39-935-653327.

E-mail address: rferri@oasi.en.it (R. Ferri)

## 1. Introduction

The two-process model of sleep regulation was proposed by Borbély (1982) which postulates the existence of a sleep-dependent process, termed Process S, and of a sleep-independent circadian process, termed Process C. In this model, the level of Process S, as reflected by the EEG slow-wave activity, corresponds to the sleep-related aspect of sleep propensity. Sleep onset is triggered when S approaches an upper threshold and awakening occurs when S reaches a lower threshold (Daan et al., 1984; Borbély et al., 1989; Ackermann et al., 1993). Finally, in this model a simple additive interaction between the two processes is postulated.

In recent studies, the calculation of the correlation dimension ( $D_2$ ) of the profiles of EEG slow-wave activity during sleep, in normal subjects, allowed us to conclude that sleep regulation might be considered as a deterministic non-linear process with an average dimension above three. Moreover  $D_2$  does not show significant changes across consecutive nights, in the same subject, and does not seem to change significantly with age in children and young adults (Ferri et al., 1996, 1998). The implications of these results are that a number of variables higher than three with non-linear interactions are needed in order to model sleep regulation and the time course of the EEG slow-wave activity during sleep.

This kind of approach has not yet been applied to pathological sleep; for this reason, we decided to analyze the profiles of EEG slow-wave activity in a group of narcoleptic subjects who participated in a previous study in which their sleep was recorded for 32 h under bed rest conditions, following 16 h of diurnal sleep deprivation (Nobili et al., 1995). The main conclusion of such a first study was that EEG slow-wave activity profiles of narcoleptic patients show in this experimental condition a reduction of the strength of circadian and circasemidian mechanisms while the ultradian drives to sleep seem to be more active thus resulting in a less complex wake-sleep distribution (Nobili et al., 1996); thus, the hypothesis tested in the present study is that such profiles show a

lower degree of complexity, compared to those of normal controls, as reflected by their  $D_2$ .

## 2. Subjects and method

### 2.1. Subjects

In this study we reanalyzed the profiles of EEG slow-wave activity during sleep of 9 narcoleptic patients who participated in a previous study (Nobili et al., 1995). The patients (seven males and two females) were aged 20–55 years and had been chosen on the basis of the presence of at least two sleep onset REM-sleep episodes at the Multiple Sleep Latency test (MSLT), cataplectic episodes and excessive diurnal sleepiness, reflected by a mean sleep latency at the MSLT of 5 min or less. Also their genetic status was studied and showed a DR2 positivity in the HLA region. None of the patients was taking drugs at the moment of the recording and in the three previous weeks. Nine healthy age- and sex-matched paid volunteers were also included in this study after having obtained their informed consent.

### 2.2. Experimental protocol

All subjects, patients and normal controls, slept in the laboratory for an adaptation night (23.00–07.00 h). During the following day, subjects were prevented from sleeping for 16 h and their alertness status was controlled by the laboratory staff. Starting at 23.00 h, their sleep was then recorded for 32 h in a bed rest protocol. The room was without windows and sound proof; a dim light (10 lux) was maintained constant throughout the study. Subjects were not allowed any activity but they could ask for soft drinks and meals; they were unaware of the time of the day. These conditions were kept constant for 32 consecutive hours.

Subjects were instructed to sleep as long as possible and communication between them and one member of the laboratory staff, by means of an interphone and a video monitor, was only allowed for meal delivery and bathroom pauses.

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