Narcolepsy
PRELIMINARY INVESTIGATION OF THE APPPLICABILITY OF GEOMETRIC BROWNIAN MOTION (GBM) TO MODEL THE EVOLUTION WITH TIME OF THE LEVEL OF DROWSINESS OF NARCOPLECTIC SUBJECTS

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Introduction: According to a Mayo Clinic definition, narcolepsy is a chronic sleep disorder characterized by overwhelming daytime drowsiness and sudden attacks of sleep. Its exact cause is unknown. It is sometimes accompanied by a sudden loss of muscle tone (cataplexy) that leads to weakness and loss of muscle control. Diagnosing narcolepsy can be complex and can require several tests. Only 25% of people who have narcolepsy have been diagnosed and are receiving treatment. In this paper, we solely consider narcoleptic subjects without cataplexy, and we present a first, preliminary investigation of the evolution with time of the level of drowsiness (LoD) in such narcoleptic subjects.

Our hypothesis is that the analysis, especially automatic, of the time evolution of LoD could ultimately lead to new diagnostic tools. Additionally, an understanding of this evolution would enable the design and construction of drowsiness monitoring systems specifically tailored to narcoleptic subjects, allowing them to drive more safely and/or to obtain/recover a driving license.

Materials and methods: Since the evolution of the LoD over time, referred to here as “LoD signal”, is inherently random, one should definitely treat each such signal as being a realization of an underlying random process (RP). In a previous publication, we showed that the LoD of healthy subjects evolves in time according to a particular RP model called Geometric Brownian Motion (GBM). Our goal here is to determine whether or not the LoD of narcoleptic subjects also evolves according to GBM.

According to a well-established procedure, a given signal can be declared to be GBM if

1. (1) the logarithms of the ratios of successive values are normally distributed, and
2. (2) these ratios are uncorrelated (in time),

For the first check, we applied, to each (LoD) signal, established graphical methods, i.e. the quantile-quantile (QQ) plot and the histogram. For the second, we looked at the scatter plot of “log-ratios” versus time for each signal to see whether there was any (time) correlation between the logarithms of the ratios of successive values.

Results: The LoD signals used here were produced using a drowsiness monitoring system built in our group, consisting of a camera mounted on a pair of eyeglasses and imaging one eye. Using data obtained at 60 Hz, it produced one LoD value every 20 second. A total of 4 distinct subjects performed the following tests at two different states of sleep deprivation over one day: (1) 4 subjects performed psychomotor vigilance tasks (PVTs), leading to 4x2 LoD signals, each having 29 samples, corresponding to a duration of 10 minutes; (2) 3 subjects performed driving tests in a high-fidelity driving simulator, leading to 3x2 LoD signals, each having 49 samples, corresponding to a duration of 16 minutes.

We subjected all 14 LoD signals to the above pair of statistical tests, and obtained one LoD value every 20 second. A total of 4 distinct subjects produced one LoD value every 20 second. A total of 4 distinct subjects performed the following tests at two different states of sleep deprivation over one day: (1) 4 subjects performed psychomotor vigilance tasks (PVTs), leading to 4x2 LoD signals, each having 29 samples, corresponding to a duration of 10 minutes; (2) 3 subjects performed driving tests in a high-fidelity driving simulator, leading to 3x2 LoD signals, each having 49 samples, corresponding to a duration of 16 minutes.

We subjected all 14 LoD signals to the above pair of statistical tests, and concluded that all of them could be well modeled by a GBM RP model.

Conclusions: The preliminary results described here suggest that, similar to a healthy subject, the LoD of a narcoleptic patient evolves according to a GBM.

Restless Legs Syndrome (RLS)
PLANTAR REFLEX EXCITABILITY FLUCTUATIONS IN RESTLESS LEGS SYNDROME PATIENTS

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Introduction: An evening state of spinal hyperexcitability has been proposed to be a possible cause of evening increases in restless legs syndrome (RLS) symptoms however the aetiology is unknown. Spinal hyperexcitability, indicated by a positive Babinski sign (plantar reflex), may be caused by a loss in supraspinal inhibition. Thus the objective of the current study was to investigate, by assessing the plantar reflex, if there were diurnal changes in spinal excitability in RLS participants compared to healthy controls.

Materials and methods: Thirteen RLS participants and 13 healthy control participants’ plantar reflex responses were assessed electromyographically and kinematically in the evening (PM) and the morning (AM) using a specialised Babinski reflex hammer.

Results: RLS participants showed a circadian variation in plantar reflex responses whilst control participants did not. RLS participants evening ankle angle changes were larger (median PM: 7.91°; AM: 6.79°, p = 0.03) and faster (median PM: 2.40°/s; AM: 2.14°/s, p = 0.00) compared to morning responses. In addition RLS participants displayed significantly smaller changes in ankle angle (median PM RLS: 7.91°; control: 25.66°, p = 0.04; AM RLS: 6.79°, control: 20.96°, p = 0.02) and significantly slower ankle movements (median PM RLS: 2.40°/s; control: 6.68°/s, p = 0.04; AM RLS: 2.14°/s; control: 6.42°/s, p = 0.01) in the evening and the morning as well as significantly lower lateral gastrocnemius maximum amplitude in the morning (median AM RLS: 0.03 mV, control: 0.08 mV, p = 0.04) compared to control participants.

Conclusions: The current study supports the theory of RLS circadian fluctuations in spinal excitability. An unexpected finding was decreased plantar reflex responses in RLS participants compared to healthy control participants. However this finding does support the theory of mechanical hypoesthesia in RLS patients.

Insomnia
PREVALENCE OF INSOMNIA AND ITS CLINICAL PHENOTYPES IN EPILEPSY AND THEIR RELATION TO SEVERITY OF DEPRESSION

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Introduction: Sleep is frequently disturbed in epilepsy. On the other hand, depression is vastly present in many patients with epilepsy (PWE). Various reasons exist, including intrinsic factors and pharmacotherapy. Few studies include clinical assessment of insomnia as a separate clinical comorbidity in epilepsy. The aim of this study was to assess the prevalence of insomnia and its clinical phenotypes in PWE and their relationship to depression severity.

Materials and method: PWE were enrolled according to proven diagnosis, made at tertiary epilepsy and sleep centers. Insomnia diagnosis was placed based upon complaints obtained during somnological interview. Healthy control (HC) group was enrolled based on random principle from general population. Clinical insomnia phenotype (CIP) was divided into 3 categories: sleep-onset insomnia (SOI), sleep-maintenance insomnia (SMI), mixed-phenotype insomnia (MI). The latter were obtained from Hamilton’s Depression-rating Scale (HAM-D), which also served for depression assessment, with severity ranging from mild to moderate to severe. T-test and Chi-square test were used for statistical analysis.

Results: Overall, 169 PWE and 100 HC participated in our study. In PWE group 80 subjects (47.3%) complained of insomnia, compared to 27 (27%) in HC group (p < 0.001). The distribution of insomnia phenotypes among PWE with insomnia was as follows: SOI – 27.5%, SMI – 13.2%, MI – 59.3%. According to HAM-D scale 37.3% had no depression, 31.3% – mild, 27.1% – moderate and 4.2% – severe. Using Chi-square analysis we found out the prevalence of various CIPs according to severity of depression subgroups, showing a tendency to have more cases of pure SOI in mild depression and more MI in moderate-to-severe depression (p < 0.001).

Conclusions: Our results show significantly higher prevalence of insomnia in epilepsy. Also we report description of CIP, which are differently distributed according to depression severity with pure SOI tending to occur more in mild while MI in moderate-severe depression.

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