Human Sleep and Sleep EEG

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Abstract. *This review article summarizes the basic knowledge from the field of sleep research. The emphasis is on the exploration of the rules of polysomnographic recording and scoring sleep stages as well as on results and opinions about the nature of sleep EEG. History of sleep research, sleep physiology, functions of sleep and mostly used experiments are briefly mentioned. Relevant spectral methods and methods inspired by dynamical systems theory are listed.*
Keywords: *sleep, EEG, rules of Rechtschaffen and Kales, cyclic alternating pattern, nonlinear, spectral measures.*

1. Introduction

The importance of sleep research is both in medicine and in theoretical area. There are many sleep disorders, e.g., the most frequent are insomnia, narcolepsy, sleep apnoea; many other disorders manifest themselves through sleep disturbances (e.g. depression, schizophrenia, Alzheimer disease [1], etc.). After the pain, sleep disturbances are the second most frequent indicator of illness.

During sleep, human brain goes through several psychophysiological states that are relatively stable. Many nervous centres are inactive, so brain becomes a less complex system and is a suitable object for mathematical modelling.

The beginning of modern sleep research dates back to the 1930s and is closely connected with the invention of the electroencephalography. In 1937, Loomis was the first to observe that sleep is not a homogeneous state during the whole night and described different stages of sleep based on EEG [2]. In 1953, Aserinsky and Kleitman observed a special state of sleep - rapid eye movement (REM) sleep, during which rapid, binocularly symmetrical eye movements occur, EEG pattern is similar to the one observed during wakefulness, and both respiratory and heart rates are increased in contrast to other sleep stages. Their experiments resulted in a relationship between REM sleep and dreaming: majority of people awakened from REM sleep reported dreams, whereas people awakened during nonREM sleep did not recall dreams [3]. From overnight recording of EEG and electrooculogram (EOG), Kleitman with Dement [4] specified the cyclic pattern of REM-nonREM sleep. One cycle of REM-nonREM lasts about 90-100 minutes and during the night, 4-5 cycles occur. Aserinsky and Kleitman also divided nonREM sleep into four stages: 1 through 4, ranging from the lightest sleep in stage 1 to the deepest sleep in stage 4.

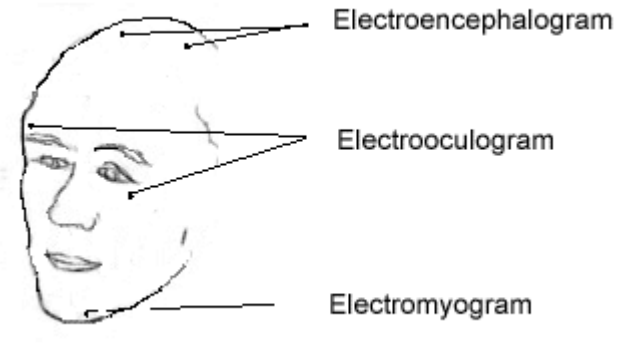

Figure 1: Placement of electrodes of polysomnographic measurement

2. Sleep Stages and the Rules of Rechtschaffen and Kales

The main states of vigilance are wakefulness, REM sleep and nonREM sleep. NonREM sleep is further divided into four Stages from the lightest Stage 1 to the deepest Stage 4. Stages 3 and 4 are referred to as slow wave sleep (SWS). The frequency of sleep Stages alters during the night - in the early hours of sleep SWS dominates, whereas REM sleep occurs more often in the second part of sleep. The portion of REM sleep during night alters with age - in new-born babies REM sleep lasts for 50%, in adults for 20%.

An essential method in human clinical and basic sleep research is polysomnography. It is composed of measuring electroencephalogram (EEG), electrooculogram (EOG) and electromyogram (EMG), see Figure1. Electroencephalography is the basic method with an excellent temporal resolution and lower spatial resolution of electrical activity of cerebral cortex. The quality of EEG recording depends on some technical parameters, see [5] for details.

Sleep Stages are scored according to "A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subject", which was elaborated in 1968 by a committee co-chaired by A.Rechtschaffen and A.Kales [6]. The purpose of these uniform and standard criteria was to increase the comparability and replicability of results from different laboratories. The Manual involves parameters, techniques and wave patterns of polysomnographic recordings. One channel of EEG, two channels of EOG and one channel of EMG are recorded. The EEG derivations are C4/A1 or C3/A2 according to the 10-20 electrode placement system (see Fig.2). The potentials for eyes movements recording are measured from 1 cm above and slightly lateral to the outher canthus of one eye and 1 cm below and lateral to the outer canthus of the second eye. The reference electrodes for both eyes are placed on the same ear lobe or mastoid. The EMG is recorded beneath the chin (mental, submental). The placement of electrodes of polysomnographic recording is illustrated on Fig. 1. The Stages are scored epoch-by-epoch in 20-30 s intervals.

Waking (Stage W)
There is a low voltage (10 − 30μV) and mixed frequency EEG during wakefulness (see Fig. 3). Possible features are alpha activity in EEG and relatively high tonic EMG.

Movement Time

If in more than half an epoch of the EEG or EMG signals are unclear due to amplifier blocking or muscle activity, the epoch is counted neither with sleep nor with waking, but is labelled as movement time. It is not the same as discrete body movements, which could be very short. Body movements can be a part of a sleep Stage or the movement time.

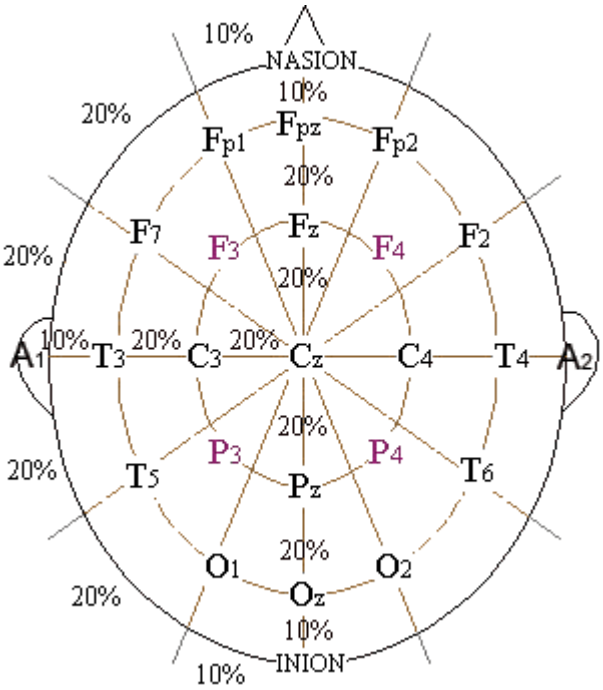

Figure 2: 10-20 electrode placement system for EEG measurement

Stage 1

Stage 1 is characterized by low voltage, mixed frequency EEG with the highest amplitude in 2-7 Hz range (see Fig. 3). The vertex sharp waves may occur; their amplitude can reach the value of about 200 μV. In Stage 1 after wakefulness slow eye movements can be present. The EMG level is lower than in the wakefulness. Stage 1 is also scored when the epoch is characterized with alpha activity combined with mixed frequency EEG and the amount of alpha activity is less than 50% of an epoch.

Stage 2

Stage 2 is characterized by wave patterns sleep spindles and K complexes and the absence of slow waves (see Fig. 3). K complex is a sharp negative wave followed by a slower positive one. Sleep spindles occur in 12-14 Hz frequency range. The duration of these patterns should be 0.5 s at minimum. If the time between two succeeding occurrences of sleep spindles or K complexes is lower than 3 min, this interval is scored as Stage 2, unless there are movement arousals or increased tonic activity. If the time interval is 3 min or more, it is scored as Stage 1.

Stage 3

20%-50% of the epoch of EEG record should contain waves with 2 Hz or slower and with the amplitudes above 75 μV if the epoch is scored as Stage 3, see Fig. 3. Sleep spindles and K complexes may occur during Stage 3.

Stage 4
Stage 4 has the same attributes as Stage 3, but waves with 2 Hz and slower with the amplitudes greater than 75 μV 50 appear more than 50% of the epoch.

Stage REM
Stage REM shows low voltage and mixed frequency (similarly to Stage 1) of EEG, sawtooth wave pattern is often present (see Fig. 3). EMG reaches the lowest level and episodic rapid eye movements occur (REMs).

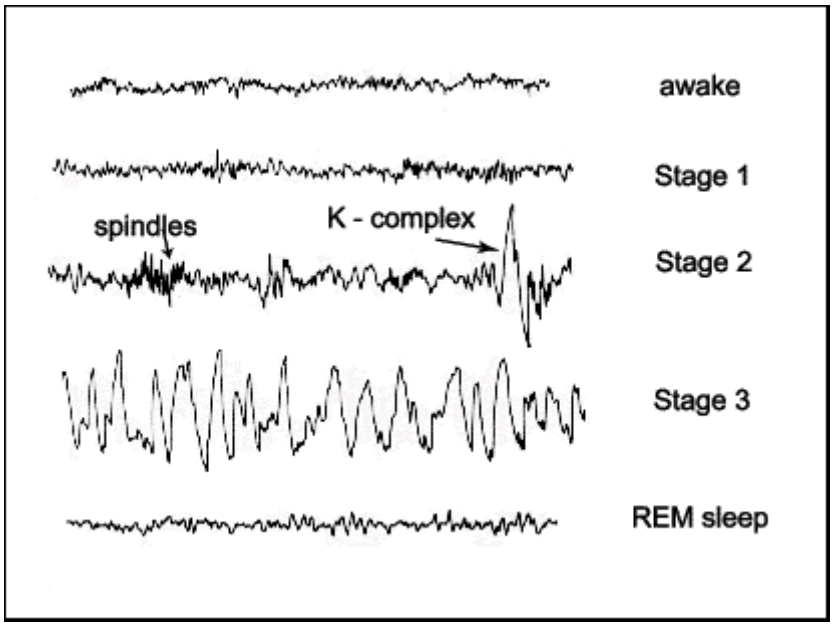

Figure 3: Wave pattern of different sleep Stages

There exist cases when no movement arousals are present, EEG exhibits a relatively low voltage and mixed frequency, and sleep spindles (K complexes) characteristic for Stage 2 alternate with typical features of Stage REM (REMs, the lowest EMG level). Then scoring follows these rules:

1. Stage REM: EMG is at the lowest Stage REM level or the rapid eyes movements are present.

2. Stage 2: interval between two sleep spindles or K complexes is less than 3 minutes. The rules of Rechtschaffen and Kales have been used for more than 35 years, but they have some dearths and disadvantages [7]:

- They ignore events shorter than 30 seconds. If the interval contains features from more than one Stage, it is scored as the Stage whose features have the longest duration.
- They are designated for healthy adult people and hence, it is not possible to score atypical patterns in cases of ill people or children.
- Some wave patterns (sleep spindles or K complexes) are not well defined, especially with respect to automated sleep scoring.

3. Cyclic alternating pattern

The alternation of the above defined sleep Stages represents the macrodynamics of brain. In the concrete sleep Stage the level of arousal is assumed to be stable [8]. Different approach gives the idea of cyclic alternating pattern (CAP). CAP is a "periodic EEG activity of nonREM sleep, characterized by sequences of transient electrocortical events that are distinct from the background EEG activity and recur at up to 1 min intervals" [9]. CAP is functionally connected with fluctuation of arousal. CAP sequences occur in all Stages 1, 2, 3 and 4 and in preference to 4 sleep onset, after awakeness during sleep and before the transition from nonREM to REM sleep [8]. In normal REM sleep CAP does not occur. The rate time (CAP)/time(NREM) in young adults is about 23% and increases with age [8].

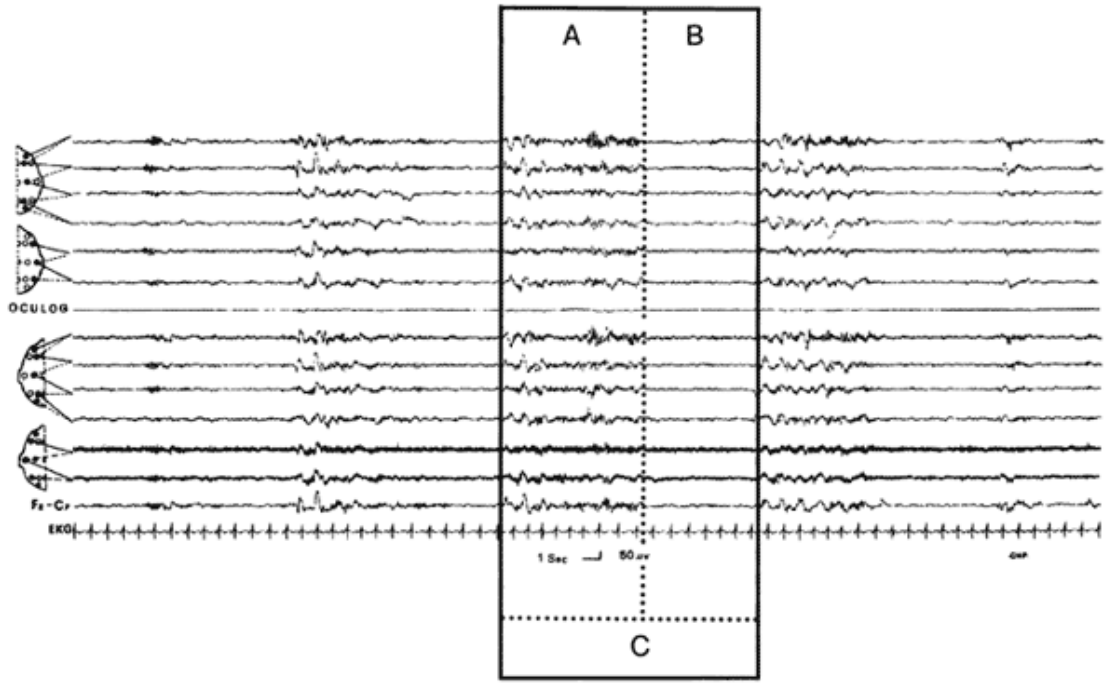

Figure 4: Cyclic alternating pattern (C) in sleep Stage 2.
A - phase A, B - phase B.
EEG derivations after international electrode placement: top 6 channels from top to bottom:
FP2-F4, F4-C4, C4-P4, P4-O2, F8-T4, T4-T6.
OCULOG - oculogram.
bottom 7 channels: FP1-F3, F3-C3, C3-P3, P3-O1, F7-T3, T3-T5, F2-C2.
EKG - electrocardiogram[1]

CAP is composed of two phases - phase A and phase B (see Fig. 4). At least two CAP cycles have to occur consecutively to be regarded as CAP sequence. Phase A represents events clearly outstanding from the background rhythm - abrupt changes in frequency and/or amplitude. Phase B is an intervening interval between phases A.

[1] Reprinted from Sleep Medicine 2, Terzano M. G. et al.: Atlas, rules, and recording techniques for the scoring of cyclic alternating pattern (CAP) in human sleep., 537-553, 2001 with permission from Elsevier

According to American Sleep Disorders Association CAP, sequences and microarousals may indicate instability or sleep disturbances with detrimental effects on sleep. Halász, Terzano et al. [10] presented an opposing idea: microarousals and CAP sequences are natural parts of the sleep texture. The physiological function of CAP could be protecting of reversibility of sleep and also in connection between the sleeping brain and his surrounding space to adapt to potential changes and danger.

4. Physiology of sleep

Sleep-wake cycle is regulated by multiple sleep and wake promoting systems, which are spread all over in the brain. Sleep begins with activation of the preoptic area of the anterior hypothalamus. Sleep promoting neurons project to wake-promoting centers and inhibit them with γ-aminobutyric acid (GABA) as neurotransmitter. The inhibition of wake-promoting neurons works on other sleep-promoting neurons and activates them, which results in intensifying the sleep process [11].

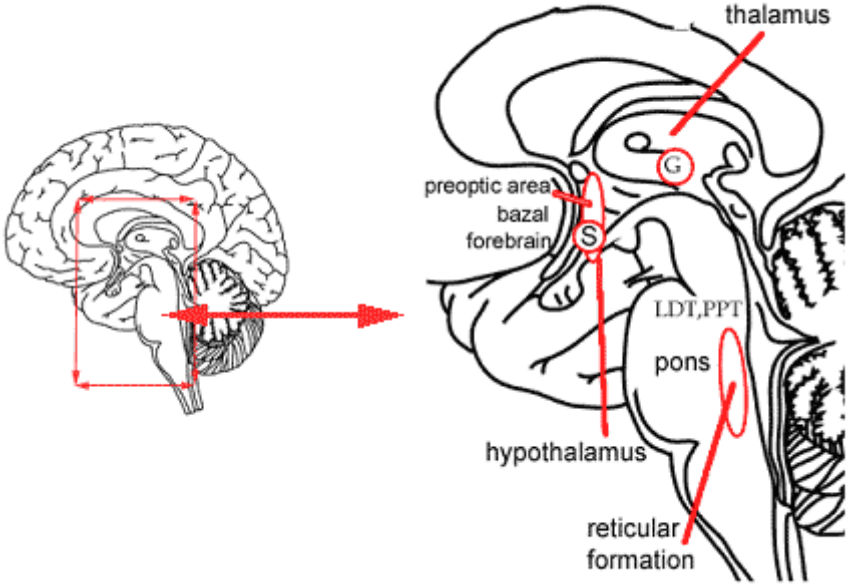

Figure 5: Sleep and waking centres. S - suprachiasmatic nucleus in hypothalamus, G – lateral geniculate nucleus in thalamus, LDT - laterodorsal tegmental nucleus, PPT – pedunculopontine tegmental nucleus, LDT, PPT in brain stem

REM sleep is regulated mostly by the brain stem; the two most important nuclei are laterodorsal (LDT) and pedunculopontine (PPT) tegmental nuclei. LDT and PPT project to thalamus, basal forebrain and the cortex, which output the desynchronized EEG pattern. The descending pathways to α motor neuron cause the skeletal muscle atonia [12]. Typical neuronal activity before the rapid eyes movements - PGO waves - rises from the pons and spreads through LGN (lateral geniculate nucleus) in thalamus to the occipital lobe [13, 12].

The waking and arousal promoting centers are located in the posterior hypothalamus, basal forebrain, mesopontine tegmentum and contain cholinergic, noradrenergic, serotonergic and histaminergic neurotransmitters [11]. The arousal starts in reticular activating system (RAS), which receives collateral inputs from visceral, motor and sensory systems. RAS projects to the forebrain and cortex via thalamic and extrathalamic neural pathways.

5. Function of sleep

For long time people were interested why sleep is so essential for the life. There are many theories, which try to explain functions and the purpose of sleep. Some of them satisfactorily interpret several facts, but broadly accepted theory that would explain all phenomena and experiments, does not exist till now. Here only the main theories are mentioned:

1. Conservation of energy: the main arguments for the purpose of sleep as reservation of energy are that during the sleep deprivation the energy consumption is increased and vice versa during sleep the basal metabolism is decreased about 5-25% [14].

2. Restoration of tissues and growth: during the first hours of sleep growth hormone excretion, cell mitosis and protein synthesis are increased. In the time of growth or after more laboured day the amount of NREM sleep is increased during the night. However J. Horne [15] criticized this theory. According to him cell mitosis occurs a few hours after food intake and has a circadian rhythm, the decreasing metabolic rate is in discrepancy with the protein synthesis that needs higher energy cost and the increased temperature of head after physical activity is the cause of increased rate of SWS.

3. Thermoregulation: in experiments with rats, long-term sleep deprived rats showed the temperature increased in about 10 degree [16], so sleep probably decreases the temperature.

4. Regulation of emotions [17]: in humans the sleep deprivation causes the disturbances of emotional behavior (such as concentration, interest for distinct goal, etc.), particularly SWS deprivation induces depressive or hypochondriacal states. So NREM sleep is likely to be involved in adjusting and regulating these emotions. This theory is supported by clinical observations that depressed patients show lower duration of NREM sleep as well as that metabolic rates and neuronal discharge are higher in brain regions that take control of emotions (limbic structures) during NREM sleep in contrast with waking state.

5. Neural maturation: one part of theories about sleep functions is concerned with REM sleep. The percentage of REM sleep of total sleep time decreases with age - in about 6. month of prenatal phase the children spend about 80% of sleep in REM sleep, but young adult people only 25% [14]. So it is assumed that during REM sleep the maturation of brain and myelinization of nerve fibers proceed.

6. Memory and learning: both types of sleep NREM and REM play a key role in memory consolidation and learning. There is an information transfer between cortex and hippocampus during the sleep that realizes the fixation of memory traces or during REM sleep the insignificant bindings are abolished [18]. With this reprocessing of information also the learning process is related. Several papers refer the improvement of performance perceptual or motor task after sleep [19, 20]. The improvement is due to sleep and not due to time interval or circadian factors.

6. Experiments

In research and also in medical care, it is necessary to perform investigations and experiments. In most experiments people are asked to refrain from alcohol, caffeine and other drugs that influence sleep during the study and have a regular sleep regime some time before.

Polysomnographic recordings of EEG, EOG, EMG are taken during the whole night to score the sleep Stages and the course of the sleep. In clinical practice ECG, blood pressure, blood oxygenation and breath rate are also measured.

Typical features of many sleep disorders are extreme fatigue and sleepiness during the day. In order to reveal the tendency to sleep during the day, the Multiple Sleep Latency Test (MSLT) is used. After a normal night person is lying in a dark quiet room and is asked to fall asleep. Time from the beginning of the test to the first epoch of sleep - the latency of the sleep – is measured. This procedure is repeated 4-5 times in two hour intervals during the whole day. The sleep onset is obtained on the basis of the polysomnographic measurements. The average sleep latency is evaluated. The value of average sleep latency less than 5 min is regarded as pathological [21]. In conditions similar to MSLT, the Maintenance of Wakefulness Test (MWT) is performed, but people are asked to remain awake during soporific circumstances. Again, the average sleep latency is evaluated and the value less than 11 min is taken as pathological [22].

Other types of experiments are also used in research of sleep regulation. Effects of sleep deprivation or daytime naps are investigated and forced desynchrony experiments are arranged. The night after sleep deprivation (or daytime naps) is compared with the baseline night, and the variation in EEG between them is evaluated. The most significant difference is in the amount of slow wave sleep. In the forced desynchrony protocol people are exposed to artificial length of the day, different from 24 hour cycles. They are deprived of external periodic light/dark cycle. During one third of the artificial cycle, the lights are turned off and people have the opportunity to sleep [23, 24].

Let us mention another type of experiments - from theoretical area - models of artificial neural networks (ANN), which are inspired by nervous system. Originally, the ANN were developed to solve problems in technical area, but today they can also be applied to physiological data. In sleep research, ANN are used in automated scoring of sleep Stages [25, 26] and in classifying artefacts in EEG [27].

7. Models of sleep regulation

Today it is generally accepted that there are three processes that regulate sleep: a homeostatic process, a circadian process and an ultradian process [28].

The homeostatic process takes control of the amount of sleep and wakefulness, so that the homeostasis is reached. It increases the fatigue and sleep propensity during wakefulness and decreases it during sleep. The indicator of homeostatic process is SWS, which occurs more in the first part of sleep and its presence during the night gradually decreases. The SWS activity is significantly enhanced during the recovery night after sleep deprivation [29]. In contrast, daytime naps cause the attenuation of SWS [28]. Until now, physiological centre of the homeostatic process has not been identified.

Circadian process reflects the influence of external events which oscillate with circadian rhythm. Circadian process represents the alternation of sleep propensity with cca 24 hours rhythm. Also, some other processes show circadian behavior - for example the core body temperature, plasma melatonin or cortisol concentration [24]. In the forced desynchrony protocol the circadian and homeostatic processes can be unlike the homeostatic process, the brain structure of the circadian pacemaker is known - it is the suprachiasmatic nuclei of the hypothalamus [24] (see Fig. 5).

Ultradian process administers the variation of nonREM and REM phases during the sleep.

One of the basic models is the two-process model of sleep regulation [23, 28]. It assumes the interaction of the homeostatic and circadian processes. The homeostatic variable S (sleep propensity) rises exponentially during wakefulness until it reaches the upper threshold H – the beginning of the sleep. During sleep, S decreases, un till it reaches the lower threshold L characteristic for the arousal. Both thresholds H and L change according to the phase of the day. The exponential function is fitted through 3 data points: the relative slow wave activity at the end of a normal night, after normal waking and after 40 hours of sleep deprivation.

Other models of sleep regulation propose variant interaction between homeostatic and circadian process or add other components, for review see [28].

8. Linear and nonlinear measures

Spectral theory is conventional and the most used linear tool in the analysis of biosignals. Spectral analysis is used to investigate the signal's power in the various frequency bands and also the mutual relationships between more signals. It is based on Fourier transform which displays signal in the frequency domain [30]:

$$X_k = \sum_{j=0}^{N-1} x_j e^{2\pi ijk/N} \qquad k = 0,\ldots,N-1 \tag{1}$$

where x_1, ..., x_i, ...x_N is the measured signal in the time domain, X_k is the amplitude corresponding to the k^{th} frequency and N is the number of values.

The power of particular frequency band is computed as the sum of modulus-square amplitudes belonging to this band. Here is an example of the power in alpha band (8Hz-12Hz):

$$P_\alpha = \sum_{k=k_{8Hz}}^{k=k_{12Hz}} \left|C_k\right|^2 \tag{2}$$

where k_{8Hz} is the lower limit of alpha band and k_{12Hz} is the upper limit of alpha band.

Another often computed index is coherence that reflects the degree of synchrony between signals from different derivations (brain areas). It is calculated as the ratio between crossspectrum of two signals and a product of their autospectra:

$$coh_{AB}(f) = \frac{\|P_{AB}(f)\|^2}{P_A(f)P_B(f)} \tag{3}$$

where PAB is the cross-spectral density of signals A,B and PA, PB are the autospectral density of these signals.

The principal assumption for using spectral theory in stochastic processes is the stationarity of the process. This property is never exactly fulfilled in the case of EEG [31], but can be approximated on very short time intervals (several seconds). For more details about assumptions and algorithms from spectral theory see [30, 31]. Some results of applying spectral analysis to sleep EEG are incorporated in the Rechtschaffen and Kales rules for scoring sleep Stages [6].

Dumermuth et al. [32, 33] have computed power and coherence spectra of all-night sleep EEG. In accordance with their results the integrated power and integrated coherence (0.1-7.0 Hz and 7.1-12.0 Hz) increases during SWS with regard to wakefulness. During REM phase the power decreases, but the coherence between hemispheres increases or maintaines at the same level, it is most evident in the biparietal area [32]. The average power in frequency band 0-6 Hz is maximal in Stage 4; in the band 6-10 Hz it is in Stage 4 or in the Stage 3; in 12-14 Hz band in Stage 2 and in the band 14-30 Hz it is in the Stage 1 [33], see Figure 6. The shape of power spectra is similar in every Stage - the higher power is in the lower frequencies and vice versa. The range of power, e.g. the difference between highest and lowest power, varies with Stages, the lowest range is in Stage 1 (12-14 dB) and increases with the depth of sleep to Stage 4 (29-32 dB). In Stage REM the range of power is between Stages 1 - 2 and in waking it is similar as in Stage 1. Coherence in the low frequencies (0-8 Hz) is maximal in REM sleep, in the middle frequencies (8-14 Hz) in Stage 3 or 4 and in the highest frequencies (14-30 Hz) again in REM sleep, it is most pronounced between symmetrical interhemisferic derivations.

Achermann et al. have done more precise coherence analysis of sleep EEG [34]. They have evaluated Stage-dependent and topographic-dependent (intrahemisferic, interhemisferic - homologous and non-homologous comparisons) coherence spectra. Coherence spectra between homologous derivations have declining frequency-dependent shape, in contrast with all others derivations with flat, low-level spectra. In NREM sleep the coherence spectra show outstanding peak in sigma band (13-14 Hz) in all derivations and smaller peaks in alpha and low delta bands. In coherence spectra in REM sleep these peaks are attenuated.

Merica and Blois [35] have compared the power in different frequency bands in NREM versus REM sleep episodes within sleep cycle as well as during the course of the night. Within NREM episode the power in β(14.75Hz-30Hz) band changes reciprocally to the slower bands (δ (0.5 Hz-3.75 Hz), θ (3.75 Hz-6.75 Hz)). In the last quarter before REM phase powers in all bands but β decreases and β power increases. The decrease of α (6.75 Hz-12.5 Hz), σ (12.5 Hz-14.75 Hz), θ and δ powers persists also on the first 30 % of time of REM phase and then the powers are stable. β power remains at the same level as on the end of NREM phase. The power of slower frequency bands (α, θ and δ) in NREM decreases during the night. Power of delta band decreases in accordance with the homeostatic process. The evolution of beta and sigma powers during the night differ from the slower bands: after the second episode of NREM sleep their power increase. The course of powers of all bands in REM sleep is similar - it increases in all cases.

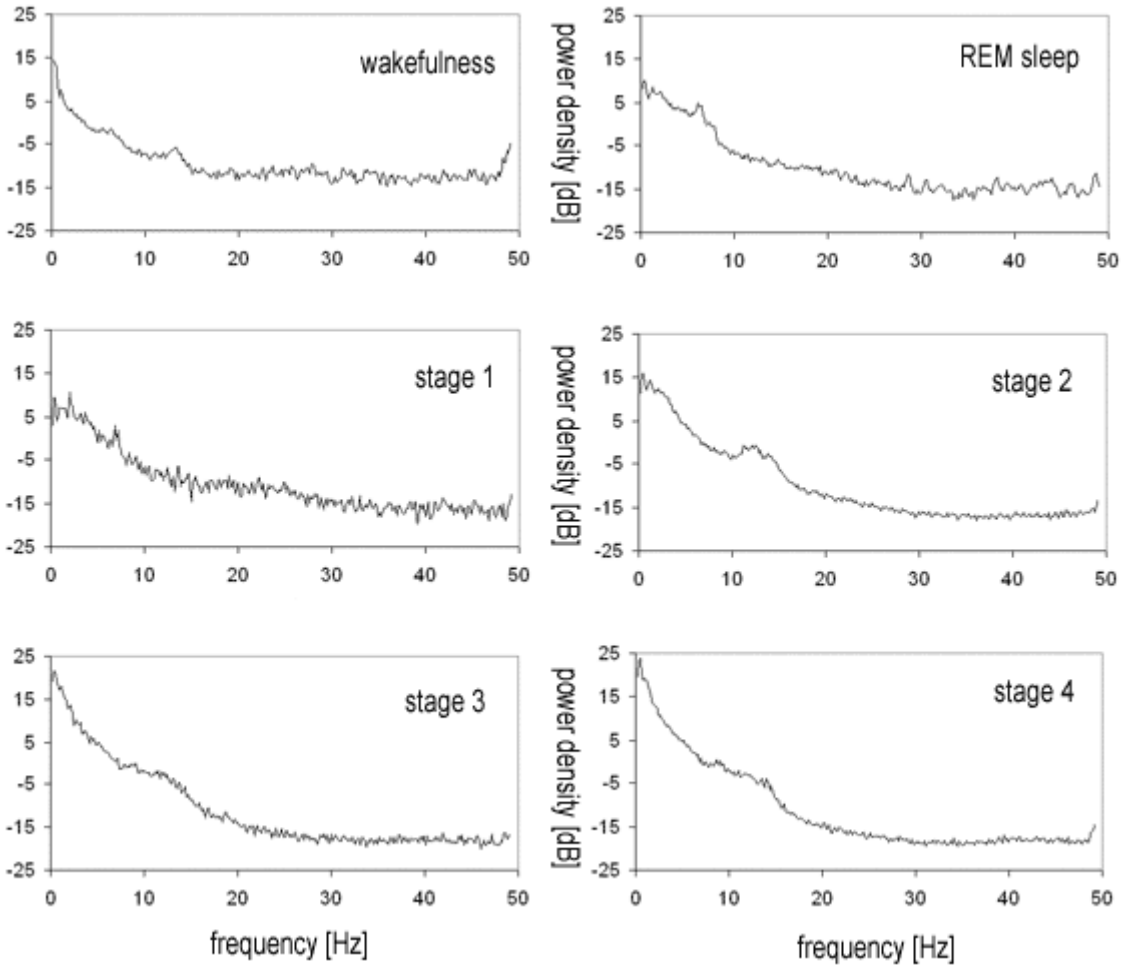

Figure 6: Power spectra density in different sleep Stages and in wakefulness, the EEG derivation is Fpz-Cz, all signals are from the first episode of NREM-REM sleep cycle. 0dB = 1μV^2/0.25Hz. In the low-frequency part of the spectra (0-10 Hz) the maximal power is in the SWS, in the 12-14 Hz band the power is maximal in the Stage 2 and in the fast-frequency band the power is maximal in wakefulness.

Ferri et al. [36] have focused on the analysis of high-frequency bands: β(15-25 Hz), γ1 (25-35 Hz) and γ2 (35-45 Hz). The powers in these frequency bands do not show significant changes, but the peaks of ratios of β/γ2 and γ1/γ2 are highly correlated with occurrence REM sleep, during the NREM phases these ratios decrease. The delta power displays reciprocal activity with these two ratios.

However, brain as the highest control system with many feedbacks appears to be a suitable object for nonlinear theory. Many methods of nonlinear theory are based on reconstruction of the phase space. According to Taken's theorem [37] it is possible to reconstruct a phase space topologically equivalent with the original one from a single observable variable. The reconstruction is done by time delay embedding (or related methods). From single variable X we obtain vectors in m-dimensional phase space: $x_i = (X_i, X_{i+\tau_i}, X_{i+2\tau}, ..., X_{i+(m-1)\tau})$, τ is the time delay, m is the embedding dimension. Since we have only limited amount of data, the proper choice of m and τ is crucial for the good reconstruction. The irregular nonperiodic time series that are nevertheless deterministic and are just slightly predictable are called chaotic time series. Two main properties of chaotic systems are self-similarity and sensitive dependence on initial conditions. These features can be treated by computing correlation dimension D_2 and Lyapunov exponent λ.

First attempts to apply variables from nonlinear theory to EEG appeared after publication of the Grassberger-Procaccia algorithm (GPA) for computing the correlation dimension D_2. D_2 is a measure of a complexity of the system. For deterministic systems D_2 reaches finite values (suffcient embedding dimension must be specified), especially for chaotic systems it is noninteger value and for stochastic systems it is determined as high as the embedding dimension m. The finite estimate of D_2 determines the number of effective degrees of freedom of the deterministic dynamical system. GPA is based on computation of the correlation sum [38]:

$$C_2(\varepsilon) = \frac{2}{(N)(N-1)} \sum_{i=0}^{N} \sum_{j>i}^{N} \Theta(\varepsilon - \|x_i - x_j\|) \tag{4}$$

where x_i, x_j are vectors in the phase space, N is the number of vectors and $\Theta(\varepsilon - ||x_i - x_j||))|$ is the Heaviside function, which is equal one if the pair of vectors x_i, x_j are less than a geometrical distance ε and zero otherwise. D_2 is defined as:

$$D_2 = \lim_{\varepsilon \to 0} \lim_{N \to \infty} \frac{\ln C_2(\varepsilon)}{\ln(\varepsilon)} \tag{5}$$

C_2 is computed for several values of embedding dimension m. For deterministic signals $C_2(\varepsilon)$ shows a power-law behavior, so if we take the local slope of ln C2 against ln ε, then the value of the plateau is taken as the estimate of D_2. In the case of EEG there are some factors which influence the exactness of the result: the number of the data, the signal to noise ratio and the stationarity of the data. It is necessary to find a compromise of the data size, which is of sufficient length, but the stationarity can be assumed. Another characteristic, often used in analyses of physiological data, is the largest Lyapunov exponent λ - the measure of the exponential divergence of trajectories in the phase space [38]:

$$\delta_{\Delta n} \cong \delta_0 e^{\lambda \Delta n} \tag{6}$$

where δ_0 is the beginning distance between two close trajectories in the phase space and $\delta_{\Delta n}$ is this distance after Δn time steps. Positive value of λ implies the presence of chaos behind the time series. Likewise as D_2, the precise value of λ is not easy to compute for EEG, there are the same sources of problems, including stationarity level and noise corruption.

In 1985, Babloyantz et al. [39] predicted the existence of the low-dimensional chaotic attractor in the sleep Stages 2 and 4 (for Stage 2, $D_2 = 5.03$, for Stage 4 $D_2 = 4.05$, $\lambda \in (0.3, 0.8)$). Other results also conclude that D_2 in sleep are smaller than in awake EEG. D_2 seems to be highest in REM sleep and smallest in the Stage 4 [40, 41]. Similarly, the deeper the sleep, the lower the values of λ [42]. However, the finite estimates of D_2 of EEG were received with skepticism by many researchers from the area of nonlinear dynamics [41]. They pointed to some crucial details of the algorithm and assumptions, which may not hold (e.g. stationarity, the sufficient data size, proper embedding). Theiler et al. [43] have demonstrated that also for time series with insufficient length from autocorrelated Gaussian noise GPA gives spurious estimations of D_2 and proposed to omit those pairs of vectors which are closer than autocorrelation time. The re-examination of the previous results using this correction [44] showed that the low-dimensional estimate of D_2 in EEG was the artefact of the temporal autocorrelation.

Another approach to investigate the nonlinear nature of signals is to compare them with surrogate data. Surrogate data are created by preserving one discriminatory property, while other properties

are changed. It is a statistical test that aims at finding out whether the data from different classes of processes could give similar values of the chosen property [41]. If the nonlinearity of the signal is assumed, the null hypothesis is stated that it is linear stochastic process and a set of stochastic surrogate data is made. If the value of computed property of the original signal is significantly different from the values of surrogates, the null hypothesis can be rejected. In opposite case it is possible to look for a new discriminatory property. More information about surrogate data method can be find in [45, 41, 38]. In testing the surrogate data, D_2 does not appear to be the best discriminatory measure to distinguish between deterministic and stochastic nature of EEG [41]. Pereda et al. [46] have computed the fractal exponent β and the D_2 of sleep Stages using surrogate data in order to reveal whether finite estimates of D_2 are due to the nonlinear character of EEG, or whether EEG is better described as linearly-correlated noise. The power spectra of stochastic processes show the power-low decrease with frequency ($1/f^{\beta}$). Following their results, only SWS displays nonlinear structure (D_2 of original EEG differs significantly from surrogate data), EEG spectra of the Stages 1, 2 and REM sleep show a frequency power-low dependence $1/f^{\beta}$ with β between 1 and 3. For β holds the more complex signal the lower value of β. Between β and D_2 there is a negative linear relationship in Stages I, II, REM and in wakefulness. Both indexes estimate the complexity of signal, however β is preferable to D_2 due to its less time demanding computation.

Olbrich et al. [47] used autoregressive modeling of EEG during sleep and surrogate testing. The null hypothesis of linearly correlated noise has to be rejected if less than 2 % of segments have length of 1s for every Stage of sleep. As a consequence of nonstationarity, the percentage of rejection rose with the length of segments.

For detection of mutual relationship between more signals nonlinear methods can be used as well. Pereda et al. [48] have applied multivariate nonlinear time series analysis to investigate the interdependencies between channels C3/A2 and C4/A1 during all sleep Stages. The results were very sensitive to used parameters, the significance of results were checked by surrogate data. According to their results the interdependencies between these channels increased with depth of sleep and were mostly of linear type.

The question whether EEG is deterministic or chaotic is still open, although the chance that the process behind EEG is low-deterministic is small. However, nonlinear measures could be beneficial in effort to find appropriate variables for characterizing and describing various psychophysiological states of the brain. Today we do not expect that the values of D_2 will tell us the number of differential equations needed to describe the dynamical system. D_2 is interpreted as a measure of the system ”complexity”.

Fell, Röschke et al. [49] used several spectral and nonlinear measures in order to find the 12 best variables for discriminating the sleep Stages. The best discrimination is achieved with the combination of spectral entropy, λ, entropy of amplitudes, D_2 and spectral edge. If the number of variables is limited to 2 or 3, the lowest error is obtained by combining λ, entropy of amplitudes and D_2. Complexity measures related to the concept of entropy rates estimation were reported by Rosipal [50] as appropriate indicators in classification of brain states. This suggests that nonlinear measures may offer additional information about the brain state.

5. Conclusion

Sleep is traditionally classified into sleep Stages that are scored after a system of rules of Rechtschaffen and Kales (RKS). It is based on wave patterns and characteristics of polysomnographic recordings. RKS has been used from 1968 and became the basic method for visual sleep analysis.

In current research, big effort is spent on developing new systems suitable for automated scoring of sleep Stages. Many variables are tested for describing subtle changes in psychophysiological state of the brain. In the community of EEG researchers there are supporters of classical spectral methods and also their opponents from nonlinear group. It is unlikely that EEG can be regarded as stemming from a purely deterministic system, although brain certainly must contain deterministic features. In spite of some unresolved problems, the new nonlinear measure seems to be successful, if the main demand is to distinguish between normal and pathological states. In this effort the simultaneous usage of linear and nonlinear approaches appears to be more powerful than preferring only one of them.

Acknowledgement

This work was supported by Slovak Grant Agency for Science (grant No 2/4026/04).

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