

# A Review of Sleep EEG Patterns. Part I: A Compilation of Amended Rules for Their Visual Recognition according to *Rechtschaffen* and *Kales*

Eine Übersicht über Schlaf-EEG-Muster. Teil I: Eine Zusammenstellung mit ergänzenden Regeln zu deren visueller Analyse

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## Summary

*Question of the study* The reliable evaluation of polysomnographic recordings (PSG) is an essential precondition for good clinical practice in sleep medicine. Although the scoring rules of *Rechtschaffen* and *Kales* [86] are internationally well established, they leave some room for different interpretations, and this may contribute to the limited reliability of visual sleep scoring. The German Sleep Society (DGSM) has set up a task force to devise ways to improve scoring reliability in the framework of their quality management programme. The intention was not to revise the rules of *Rechtschaffen* and *Kales* (R&K), but to facilitate their reliable application in sleep scoring and to support the development of standardized algorithms for computerized sleep analysis.

*Methods* The task force was formed in September 2004 as a subcommittee of the educational panel of the DGSM. The members of the task force are experienced in sleep scoring and have a background either in physiology, neurology, psychiatry, psychology, or biology. The aim of the task force was to provide interpretation aids and, if needed, specifications or amendments to the R&K rules for the scoring of sleep electroencephalogram (EEG) waveforms and patterns. Decisions were based on the nominal group technique of a nominal panel as the formal consensus-building process. The consensus process was based on scoring and face-to-face discussions of at least 40 examples for each pattern in four 2-day meetings.

*Results* Relevant EEG patterns for sleep stage scoring are alpha, theta, and delta waves, sleep spindles, K-complexes, vertex sharp waves, and sawtooth waves. If definitions for a given EEG pattern differed in the literature, the nominal group technique resulted in specifications and amended scoring rules for these EEG patterns. A second part including a series of examples with explanatory comments for each of these EEG patterns is under preparation.

*Conclusions* Amendatory scoring rules of those EEG patterns that are relevant for sleep scoring may contribute to increasing the reliability of visual sleep scoring and to support the development of standardized algorithms for computerized sleep analysis.

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Received: 15.05.06/Accepted: 22.08.06

**Keywords** Sleep EEG – scoring – alpha, theta, delta, vertex waves – K-complexes – sleep spindles – sawtooth waves

## Zusammenfassung

**Einleitung** Die reliable Evaluation polysomnographischer Ableitungen ist eine wesentliche Voraussetzung für die Gute Klinische Praxis (GCP) in der Schlafmedizin. Obwohl die Auswertungsregeln von *Rechtschaffen* und *Kales* (R&K) [86] international gut etabliert sind, lassen diese einigen Interpretationsspielraum. Dies wird als ein möglicher Grund für die relativ eingeschränkte Reliabilität visueller Auswertungen angesehen. Die Deutsche Gesellschaft für Schlafforschung und Schlafmedizin (DGSM) hat daher eine Task Force eingerichtet, die im Rahmen des Qualitätsmanagementprogramms der DGSM Vorschläge zur Verbesserung der Auswertungsreliabilität ausarbeiten soll. Die Intention dieser Task Force ist es nicht, die Regeln von R&K zu revidieren, sondern vielmehr deren reliable Anwendung in der Auswertung von Polysomnographien zu ermöglichen und damit auch die Entwicklung von standardisierten Algorithmen in der computerisierten Schlafanalyse zu unterstützen.

**Prozedere** Die Task Force bildete sich im September 2004 als eine Untergruppe der AG Ausbildung der DGSM. Die Task Force Mitglieder sind erfahren in der Schlaflauswertung und sind entweder Physiologen, Neurologen, Psychiater, Psychologen oder Biologen. Das Ziel der Task Force war die Bereitstellung von Interpretationshilfen und – wenn notwendig – von Spezifikationen oder Ergänzungen zu den R&K Regeln zur Erfassung von EEG-Wellenformen und -Mustern. Die Entscheidungen basierten auf einem nominalen Gruppenprozess als formales Konsensusverfahren. Für jedes Element beruhte der Konsens auf der Auswertung mit Gruppendiskussion von mindestens 40 Beispielen pro Wellenform oder EEG-Muster unter Berücksichtigung der Literatur im Rahmen von 4 zweitägigen Treffen.

**Ergebnisse** Relevante EEG-Muster für die Schlafstadienanalyse sind alpha-, theta- und delta-Wellen, Vertexwellen, Schlafspindeln, K-Komplexe und Sägezahnwellen. Sobald die Definitionen für eines dieser Muster in der Literatur divergierten, führte der nominale Gruppenprozess zu Spezifikationen und Ergänzungen der Angaben von R&K. Ein zweiter Publikationsteil in dem die hier erstellten Spezifikationen an einer größeren Anzahl von Beispielen dargestellt werden, ist derzeit in Vorbereitung.

**Diskussion** Die hier vorgestellten spezifizierten und ergänzten Erfassungsregeln für EEG-Muster mit Relevanz für die Schlafstadienanalyse sollen die Reliabilität der visuellen Auswertung verbessern. Gleichzeitig können die Ergebnisse als Grundlage für einen standardisierten Algorithmus der computerisierten Analyse genutzt werden.

**Schlüsselwörter** Schlaf-EEG – Scoring – alpha-, theta-, delta-Wellen – Vertexwellen – K-Komplexe – Schlafspindeln – Sägezahnwellen

## Introduction

This is the first of two papers on the visual analysis of electroencephalogram (EEG) patterns for sleep scoring. It provides amended scoring rules for the recognition of sleep EEG patterns based on the manual of *Rechtschaffen* and *Kales* [86, 87], while the second paper will contain a collection of commented examples for visual scoring for each of these EEG patterns.

In 1968 a Committee of the Association for the Psychophysiological Study of Sleep established a manual of standardized terminology and scoring rules for sleep staging edited by *Rechtschaffen* and *Kales* [86, 87], called below R&K manual. The manual has been used worldwide in basic sleep research with human subjects, and in clinical sleep evaluation. Thus the R&K manual has become the gold standard for sleep scoring. According to the R&K rules, sleep stages are defined by three physiological variables, the electroencephalogram (EEG), the electrooculogram (EOG), and the submental or mental electromyogram (EMG). Sleep stages are scored visually, based on the recognition of EEG waveforms and typical patterns within a fixed time interval of usually 30 s, called an epoch. The occurrence, frequency,

amplitude, shape, and temporal sequence of these patterns provide information on the sleep stage assigned to a given epoch. Thus, the reliable recognition and evaluation of EEG waveforms and patterns is a crucial step in the process of sleep scoring.

Despite the fact that the R&K manual is the gold standard for sleep scoring, it leaves some room for interpretation including the evaluation of graphoelements. This may be one reason for the rather low interrater reliability of scoring [29, 78, 83].

R&K stated that the manual ‘should be viewed as a working instrument rather than a statute’, and consequently they concluded that ‘experience with the manual may suggest possible revision’. Indeed, in the years following the publication of the R&K manual, a few critical discussions were published [50, 64], and supplementary scoring rules for the sleep EEG [53] as well as scoring rules for phasic events and the microstructure of sleep [6, 107] became available. In addition, different atlases with an extensive collection of examples for scoring polysomnograms became available for adults [19, 43, 97] and infants [4, 91a, 96]. Nevertheless, a comprehensive collection of definitions, scoring rules, and

selected examples for the rating of both EEG waveforms and specific graphoelements in sleep is still missing.

The German Sleep Society (DGSM) has set up a task force to devise ways to improve scoring reliability in the framework of their quality management program. The intention was not to revise the rules of R&K but to facilitate their reliable application in sleep scoring, and to support the development of standardized algorithms for computerized sleep analysis.

## Procedures

The task force was formed in September 2004 by the German Sleep Society as a subcommittee of the educational panel of the society. The members of the task force are experienced in sleep scoring and have a background either in physiology, neurology, psychiatry, psychology, or biology.

The group reviewed the definitions of EEG waveforms as well as of patterns being crucial for sleep EEG given by R&K and other groups. Each item was assigned and coordinated by one member of the task force; the selection of given definitions focussed on their relevance for scoring sleep. On the basis of the nominal group technique of a nominal panel as the formal consensus-building process in a series of methodology-oriented meetings, the published definitions were discussed and at least 40 examples for each waveform and pattern were scored. The examples were chosen from sleep recordings of adult male and female subjects of different age. The recording included the R&K montage with both central leads and additional EEG electrode positions according to the 10/20 system.

The consensus process resulted in the development of amended scoring rules, specifying frequency, amplitude, and shape for each EEG waveform and pattern. The amendments are based on the confirmed knowledge of the members of the task force, consolidated literature, and the applicability within the selected examples.

## Results

Sleep stage scoring is based on the assessment of specific EEG frequencies as well as on the recognition of specific sleep-related EEG patterns. Some definitions of waveforms and patterns were not detailed in the manual of R&K, as this committee clearly stated that they followed the terminology of the International Federation for Electroencephalography and Clinical Neurophysiology [76, 17]. Nevertheless, the definitions of some EEG frequencies bands, as they are in use with relevance to sleep scoring, differ from the definitions of EEG terminology, which is based on the normal and pathological waking EEG [49]. Therefore, the borders of frequency bands for sleep analysis may differ from those used in the terminology for the waking. This becomes especially important for slow delta waves, the differentiation between the upper alpha range and low spindle frequencies, as well as for the definition of the theta frequency range, which is not congruent with the *low-voltage mixed-frequency* EEG as defined by R&K. Furthermore, the frequencies of specific sleep EEG patterns such as sawtooth waves, K-complexes, or sharp vertex waves fall well into the delta/theta frequency ranges and must be differentiated by additional form criteria. In case of discrepancies between definitions for sleep scoring and EEG terminology, we followed the conventions that have been established for sleep scoring.

## 1. Alpha waves

The alpha rhythm in the range of 8 to 12 Hz of the human EEG was first described by *Hans Berger* [11] and was therefore also called 'Berger rhythm'. During relaxed wakefulness with eyes closed, the dominant EEG rhythm in the majority of adults is in the alpha frequency range of 8 to 12 Hz with a maximum occurrence over the occipital lobe. Alpha is significantly attenuated by eye opening or mental operations, specifically mental imagination [20]. In most subjects the amplitude of the alpha rhythm varies between 10 and 50  $\mu$ V with a maximum over the occipital regions [118].

In vitro preparations and in vivo recordings point to cortical generator sources in layer V of the occipital cortex, and EEG, MEG, and PET studies suggest that alpha is mainly generated over posterior brain regions (reviewed in [20]). The number and exact localization of these generators remain unclear, as well as the existence of anterior alpha generators with different spectral properties.

The between-subject distribution of the alpha frequency is approximately normally distributed, with a mean of 10 Hz and a standard deviation of approximately 1 Hz in young adults, while the peak frequency within subjects is very stable, with a standard deviation of approximately 0.5 Hz [118]. This individual alpha frequency increases from early childhood up to puberty (e.g. [74]) in a nonlinear manner in several growth spurts [35]. In adults and elderly subjects, the alpha rhythm shows a progressive slowing during wakefulness, decrease in amplitude, and a diminution of alpha blocking with eye opening [33].

### *Relevance for sleep scoring*

The transition from alert to relaxed wakefulness with eyes closed is accompanied by an increase of alpha activity, while the transition from relaxed wakefulness (with eyes closed) to stage 1 sleep is accompanied by a decrease of alpha activity and the appearance of a mixed-frequency, low-voltage EEG. During sleep, the amount of alpha activity decreases and alpha reappears either during transient EEG arousals in non-REM sleep [6], or in REM sleep. In excessively sleepy individuals, rhythmic alpha activity may be present with eyes open and attenuate with eye closure [100]. Furthermore, the occipital prominence abolishes with increasing sleepiness [106].

During REM sleep, an increase of EEG activity in the alpha range can be observed and can be separated in two components [20]: The first one comprises alpha background activity, predominately recorded in tonic REM sleep and attenuated in phasic REM sleep. This alpha attenuation has been interpreted as an electrophysiological correlate of visual dream contents. The second component is made up of well-defined bursts of alpha activity that occur with equal probability in tonic and phasic REM sleep. These bursts are easily distinguished from transient EEG arousal since their duration is usually shorter than 3 sec and they are not associated with concomitant increases in the amplitude of submental EMG activity.

Finally, the terms 'alpha delta sleep' and 'alpha sleep' describe the intrusion of alpha activity, which is superimposed on the EEG of non-REM (NREM) sleep. Its frequency is usually 1 to 2 Hz slower than alpha during wakefulness [22]. This sleep pattern was first noted in patients with psychiatric disorders by *Hauri* and *Hawkins* [48] and has been regarded as an indicator of unrefreshing sleep and reduced vigilance [81]. However, other groups [90, 91] observed this pattern also in subjects without any complaint

of unrestorative sleep. In spite of this pattern being consistent in those individuals over several years, a comprehensive explanation of the phenomenon is still lacking.

#### *Definitions and open questions*

R&K followed the terminology of the International Federation for Electroencephalography and Clinical Neurophysiology [17], thereby indicating an alpha frequency of 8 to 12 Hz. Some authors also allowed frequencies below 8 Hz (e.g. [69]) or up to 12 Hz [118], but the majority of the reviewed studies [22, 77] restricted the range from 8 to 13 Hz or from 8 up to less than 13 Hz [53]. Although amplitude is described as to vary between 10 and 50  $\mu\text{V}$  in most wake subjects, no amplitude criterion for alpha in sleep has been given by the authors cited above (figure A1).

R&K defined sleep stage 1 by the dominance of a 'low voltage, mixed frequency', the transition from wakefulness to stage sleep 1. This is best seen in the EEG when the waking pattern (with eyes closed) has well-defined alpha activity. Alpha attenuation is related to the shift from stage W to stage sleep 1 [22]. This transition is characterized by a decrease in the amount and frequency of alpha activity as well as in the amplitude of alpha activity. However, as already stated by R&K, there are subjects who show little or virtually no central alpha activity in the waking record. Therefore, *Hori et al.* [53] proposed an additional measurement of occipital EEGs in order to detect alpha rhythm. For the scoring of sleep, the recognition of theta activity supports the delineation of wakefulness and stage 1 sleep, which is especially difficult in persons with a non-alpha EEG during waking. Other problems are the high interindividual range of alpha frequency, the decrease in frequency with increasing age, and the increase of anterior prominence associated with sleepiness (see above).

#### *Summary of the task force*

The significance of alpha activity with respect to the scoring of sleep stages is crucial for the delimitation of wakefulness and sleep as well as for the recognition and scoring of arousals and awakenings. The transition from wake to sleep is characterized by a decrease in the amount and frequency of alpha activity as well as by a decrease in the amplitude of alpha activity.

The following criteria for the recognition of alpha waves are suggested:

1. In accordance with criteria used in the majority of the reviewed studies, the alpha rhythm should be restricted from 8 to less than 13 Hz ([8, 13], which means including 8, excluding 13 Hz) without establishing an additional amplitude criterion.
2. An additional occipital EEG lead is recommended because alpha is most prominent in this cortical region. This might be helpful in subjects showing only poor central alpha activity in the waking EEG.
3. The frequency criterion might be modified in older subjects with a clearly identifiable slowing of the resting EEG in wakefulness.

## **2. Theta waves**

The term 'theta' was first applied by *Walter and Dovey* [111] to the frequency range below alpha (4 to 7.5 Hz). Theta waves are EEG waves with a frequency of 3 to 7 Hz without a defined amplitude range [22] and a maximum over the central and temporal cortex. Since the frequency range has

been defined by clinical convenience, some authors use slightly different definitions for the lower boundaries, ranging from 2 to 4 Hz [23, 46, 74, 99].

Single cell recordings suggest that the appearance of cortical theta activity depends on rhythmic burst firing of cholinergic cells in the basal forebrain [21]. Studies by *Hughes et al.* [56] indicate that oscillations in the alpha and in the theta frequency band share a common cellular basis. Their *in vitro* studies with slices of the cat lateral geniculate nucleus (LGN) showed that strong activation of the metabotropic glutamate receptor (mGluR) can generate synchronized oscillations in the alpha (8 to 13 Hz) frequency band, whereas more moderate activation slows these oscillations to the theta frequency range (2 to 7 Hz). This led them to propose that mGluR1a-induced oscillations in the alpha and theta frequency range represent a potential route whereby the thalamus promotes EEG alpha and theta activity in the intact brain. Other explanations for the generation of theta waves are summarized in *Steriade and McCarley* [104]. Genetic variation in the dominant frequency of theta oscillations has been studied in different strains of rodents by *Franken et al.* [38]. This group of authors found also that a short-chain acyl-coenzyme A dehydrogenase (*Acads*) mutation had a highly specific effect on the theta peak frequency during sleep but not waking [105]. These results suggest that the metabolic fatty-acid beta-oxidation pathway may be implicated in the regulation of theta activity in the sleep EEG.

#### *Relevance for sleep scoring*

A shift from alpha to theta waves in the background EEG is one of the main indicators for the transition from wakefulness to sleep stage 1. Theta power in the waking EEG has been proposed as a marker of the homeostatic process S and its time course parallels that of subjective sleepiness (e.g. [1, 37]).

During development, the waking EEG in children between 1 and 4 years of age shows diffuse synchronous theta activity. Between 5 and 6 years of age, alpha and theta are equally prominent, while after 6 years of age alpha becomes the predominant rhythm of the waking EEG [96]. On the other end of the age continuum, elderly persons show again typical changes in the EEG with a slowing of the dominant frequency. The slowing of the wake EEG and the attenuation of typical sleep patterns contribute to the frequently encountered difficulty to delineate sleep and wakefulness in elderly persons. This is especially true for the transitional phase of sleep stage 1.

During sleep, theta activity appears again as the prominent EEG activity of REM sleep. In animal studies with depth electrode recordings, hippocampal theta, a regular EEG rhythm in the 4- to 8-Hz frequency range, is one of the typical correlates of REM sleep, besides ponto-geniculo-occipital (PGO) waves, rapid eye movements, and muscle atonia.

The theta activity is also the background of spindles and K-complexes during sleep stage 2. It is replaced to an increasing extent by delta waves in sleep stages 3 and 4.

For the scoring of sleep, the recognition of theta activity supports the delineation of wakefulness and sleep stage 1, which is especially important in persons with a non-alpha EEG during waking.

#### *Definition and open questions*

According to the scoring manual of R&K, both sleep stage 1 and REM sleep are characterized by the dominance of a *relatively low-voltage mixed-frequency EEG*. The R&K rules define stage 1 sleep by a *low-voltage mixed-frequency EEG*.

This pattern was further characterized by the prominence of a 2- to 7-Hz EEG activity (figure A2). In addition, R&K states that faster frequencies in stage 1 were mostly of lower voltage than the 2 to 7 Hz activity, while the highest voltage 2- to 7-Hz activity (about 50 to 75  $\mu\text{V}$ ) tended to occur in irregularly spaced bursts mostly during the latter portions of stage 1.

Nevertheless, the terms theta waves or theta activity were not used in the R&K manual. *Caraskadon* and *Rechtschaffen* [22] specify for stage 1: 'In addition, the EEG activity with the highest relative amplitude during stage 1 sleep is generally in the theta (3 to 7 cps) range', and add that 'Bursts of relatively high-voltage, very synchronous theta activity are common during the onset of stage 1 sleep in children and young adolescents' [22: p. 1363].

#### Summary by the task force

For the scoring of sleep, the correct identification of theta activity is essential for the delineation of wakefulness and sleep. The *low-voltage mixed-frequency* EEG, which is typical for stage 1 sleep according to R&K, includes 2- to 7-Hz activity without any definition of amplitude. This term is not congruent to the definition of theta waves.

The following criteria for the recognition of theta waves are suggested:

- It is proposed to define the frequency range of theta activity for sleep analysis from 3 Hz up to less 8 Hz ([3, 8]). No amplitude criterion is specified for the evaluation of theta waves, although the amplitude is normally well below 75  $\mu\text{V}$ , with the exception of vertex sharp waves (or transients).

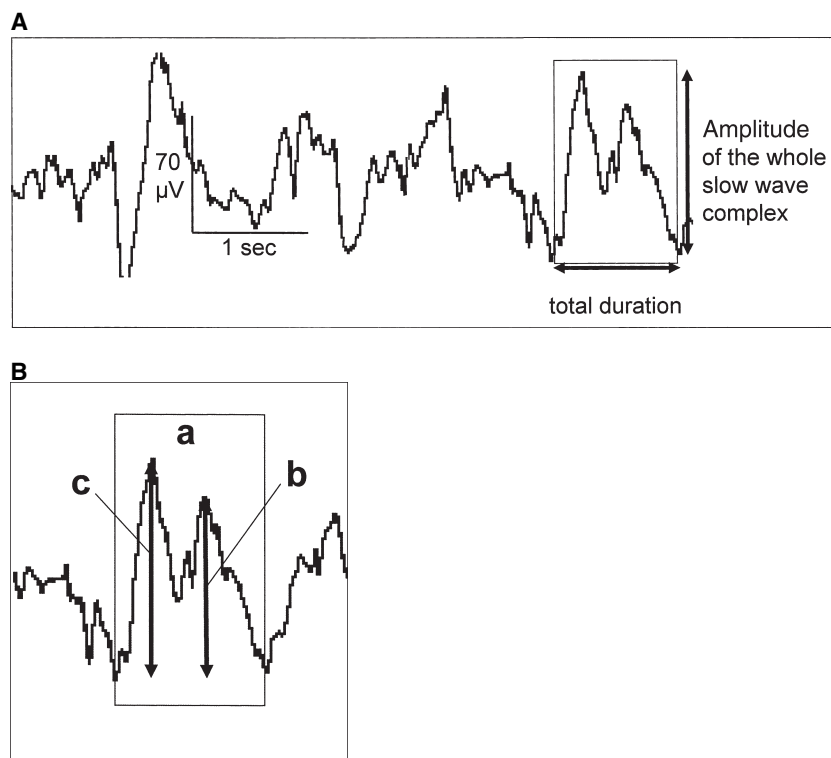
### 3. Delta waves

Walter [110] introduced the term delta to describe 'high voltage' (a few hundred  $\mu\text{V}$ ) slow waves of a frequency of

0.5 to 3 Hz in the waking EEG. In the EEG terminology, delta waves are defined by their frequency below 3.5 or 4 Hz without an amplitude criterion [118]. With respect to sleep scoring, however, delta waves are defined by frequency and amplitude according to R&K. The scoring of delta waves is further complicated by the fact that anterior delta activity is more sinus-shaped, with a frequency of about 1.5 to 2 Hz, while posterior activity is more often below 1 Hz, thereby showing polymorphic patterns [10].

The amplitude of delta waves corresponds to the amount of synchronization of cortical pyramidal cells. During the progression from waking to slow wave sleep (SWS), the thalamocortical neurons become progressively hyperpolarized. Sleep spindles and thalamic delta waves were shown to appear at different membrane potentials of the thalamocortical neurons [102]. A progressive hyperpolarization of thalamocortical neurons by a constant transition probability from fast to spindle oscillations and from spindle to the clock-like delta oscillations on the level of single neurons explain the temporal evolution of delta, sigma, and beta power during non-REM sleep [72]. In studies with mice, *Maret* et al. [70] could show that a single gene encoding the retinoic acid receptor beta (*Rarb*) essentially determines the contribution of delta power (defined as 1 to 4 Hz) relative to theta power (defined as 5 to 9 Hz) in the sleep EEG. *Rarb* receptors are presumably involved in brain development and plasticity, and in dopaminergic neurotransmission.

There exist various hypotheses on the function of delta activity in the sleep EEG. The amount of delta power during sleep depends on the duration of prior wakefulness and thus is assumed to be an indicator of the homeostatic process S [15, 28]. For some time now, SWS has also been implied in learning (reviewed in [40]), and even more recent hypotheses see the function of SWS in downscaling synaptic potentiation that have accumulated during wakefulness [108].



**Figure 1.** Illustration of the half amplitude criterion for scoring slow delta waves.

(A) Demonstration of amplitude and duration of the slow wave complex in total. (B) Illustration of the applicability of the half amplitude criterion. (a) Whole slow wave complex, (b) partial amplitude of one part of the complex, (c) amplitude of the whole complex. For scoring the whole complex as one single slow wave, the partial amplitude should be lower than 50 % of the total amplitude ( $c \leq b/2$ ).

### Relevance for sleep scoring

The amount of delta waves showing amplitudes of at least 75  $\mu\text{V}$  and a frequency of 2 Hz or less, the so-called slow waves, is the main criterion for scoring sleep stages 3 (20–50 % slow waves) and 4 (at least 50 % slow waves) according to R&K. As R&K suggested, slow waves should be measured ‘wave by wave’ (figure A3).

The time course of delta activity in the course of the night is well described, and SWS is most prominent in the first non-REM–REM cycle and decreases exponentially across the night [36, 61]. The transition between sleep stage 2 and SWS is often associated with a temporal accentuation of the anterior monomorph delta activity with a higher frequency range (1.5–2.9 Hz) more similar to K-complexes [10].

The amount of SWS or slow wave activity during sleep is maximal in young children and decreases markedly with age [13, 29]. Numerous studies (reviewed in [5]) and a meta-analysis [79] have shown that the rate of SWS decrease in lifespan is more pronounced in males, and therefore adult females showed a higher delta activity than males, particularly at old age.

### Definition and open questions

According to the rules of R&K, only slow waves with a frequency of 2 Hz or lower and amplitudes of at least 75  $\mu\text{V}$  measured from peak to peak are regarded for the scoring of sleep stages 3 and 4. In this definition, slow waves represent a part of the broader frequency band defined for delta waves, including frequencies up to 3 Hz [110] or 3.5 to 4 Hz (e.g. [118]). Some authors used a 50- $\mu\text{V}$  criterion to detect SWS (e.g. [115]) or restricted slow wave frequency below 2 Hz [53].

R&K stated that in some cases it might be difficult to decide ‘whether two contiguous potentials of the same polarity represent a single wave or two separate faster waves’ resulting in a ‘certain amount of unreliability... because it is sometimes difficult to define a wave by visual inspection’. Therefore Hori et al. [53] proposed to score a wave complex of two contiguous waves as a single slow wave as long as the amplitude of the intermediate positive component does not exceed 50 % of the preceding negative component of the wave complex. This criterion seems to be important if the delta amount of an epoch is questionable in order to differ between sleep stage 2 and SWS, and stages 3 and 4, respectively.

Terzano and colleagues [107: p. 75] defined delta bursts as a ‘sequence of at least two or more waves of delta band (0.5–4 Hz), >100  $\mu\text{V}$  in amplitude or with a voltage of at least 1/3 greater than that of background activity’.

The Japanese committee [53] proposed to measure the amplitude from peak to peak even if fast waves appear superimposed on a slow wave; while bursts of slow wave activity preceding or accompanying arousals should not be considered as slow waves for stage scoring.

### Summary of the task force

The scoring of delta waves is crucial in differentiating between sleep stages 2, 3, and 4 or at least between stage 2 and SWS. A false scoring of SWS-relevant delta waves, e.g. a failure in the differentiation of one or more delta waves, will result in false amounts of SWS per epoch.

The following criteria for the recognition of slow delta waves are suggested:

- While in the EEG terminology the delta frequency is defined as the range from 0.5 to 4 Hz, for the purpose of sleep stage scoring the lower part of this frequency band ranging from 0.5 to 2 Hz ([0.5, 2]) is called slow delta (waves). Only these slow delta waves are used to score the amount of delta for sleep staging (stages 3 and 4, slow wave sleep).
- According to R&K, such waves should be rated as slow delta if their amplitude reaches at least 75  $\mu\text{V}$ . Although not being stated explicitly in the text of R&K, the selected examples indicate that one branch of a slow delta is sufficient for assessing their amplitude.
- To simplify the attempt to differentiate between slow deltas and K-complexes, possible K-complexes embedded into series of slow waves should be scored as slow delta waves according to Hori et al. [53] (see below).
- In accordance with Hori and colleagues [53], the additional ‘criterion of half amplitude’ is recommended: At first the amplitude of the whole wave complex should be measured, subsequently the amplitudes of each single component of the slow wave complex. If the partial amplitude is lower than 50 % of the total amplitude, one single wave should be rated. If the amplitude of one single part of the complex exceeds 50 % of the amplitude of the whole complex, each part should be scored as an individual delta wave (see figure 1).

## 4. Vertex sharp waves

Vertex sharp waves (VSW) are one of the graphoelements of NREM sleep occurring early in the night at the end of the sleep onset process, right before the appearance of spindles and K-complexes [51, 88]. This pattern was first mentioned by Liberson [66]. He described localized paroxysmal waves with a frequency of 3 to 6 Hz at the beginning of sleep over the vertex. These waves, however, do not persist but are followed by slower waves, which originate from frontal and temporal regions. VSWs may occur in isolation or as trains [9].

Vertex sharp waves indicate an altered state of cerebral responsiveness [119]. They can appear spontaneously or in response to sensory stimuli. Harsh et al. [47] hypothesized that VSWs may be related to averaged ERP components. Colrain et al. [25] found the N300 amplitude in an auditory oddball paradigm during sleep to be largest when trials were included that elicited a vertex sharp wave, still prominent but smaller in trials with elicited K-complexes, and very small in the other trials during sleep. Sekine et al. [94] hypothesize that the two electroencephalogram phenomena N300 and VSW are generated by an identical synchronizing mechanism in the brain. With low-resolution brain electromagnetic tomography (LORETA) the vertex wave was localized bilaterally into the mesial frontal lobe with a maximum at the medial frontal gyri (/BA 6) [119].

However, the functional significance of vertex sharp waves is still unclear. Peszka and Harsh [84] observed an increase in VSWs (and K-complexes) in recovery sleep after sleep deprivation, while Nicholas et al. [73] observed an increase in the amplitude of the N350 (at Cz), provided that trials including VSWs were averaged in a recovery night after a night with sleep fragmentation. Bastien et al. [9] pointed out that ‘they are much more consistent with the view that the synchronized activity of large numbers of neural units required to generate theta frequency events such as VSWs (and N350) and delta frequency events such as K-complexes

(and N550), are indicative of the decreased thalamo-cortical arousal levels associated with sleep, and are probably the result of the same generator mechanisms responsible for producing spontaneous EEG activity within those frequencies'.

Vertex sharp waves develop during early infancy. At the age of 5 to 6 months, a few broad VSWs can be observed [55, 96]. During the following development, the vertex sharp waves become sharper, shorter in duration, and more repetitive in time [55]. VSWs with a similar pattern as seen in older children and adults appear at the first time around the age of 16 months [55]. In elderly subjects, VSW may become small and inconspicuous and are often poorly demonstrable [74].

#### Definitions and open questions

R&K described vertex sharp waves as a graphoelement that may occur in the late sleep stage 1 ('...during the latter portions of the stage, vertex sharp waves may appear, often in conjunction with the high amplitude 2 to 7 cps activity. The amplitude of the vertex sharp wave is occasionally as high as 200  $\mu\text{V}$ '; figure A4). Besides this definition, several later definitions of VSW vary with regard to details concerning the shape, amplitude, and frequency of this pattern and the sleep stages in which VSW occur:

The descriptions of this pattern ranges from 'compounded potential; a small spike discharge of positive polarity precedes the large following negative wave, which is almost always the most prominent pattern of the discharge. Another small positive spiky discharge usually follows' [74] over 'bilateral synchronal, mono- or biphasic, initially surface-negative transients' [62] (figure 2A,B), 'an EEG potential of cuspidate morphology' that is 'triphasic, characterized by a principal component of negative polarity, preceded and followed by components of positive polarity of brief duration and low voltage' [107: p. 29] to 'a sharp waveform distinguished from background activities' [53: p. 306]. In addition, the amplitude should be at least 75  $\mu\text{V}$  [53] or up to 200  $\mu\text{V}$  [62], while R&K stated that the amplitude 'is occasionally as high as 200  $\mu\text{V}$ ' and Terzano et al. [107: p. 29] described 'a variable voltage (up to 250  $\mu\text{V}$ )'. The frequency of VSWs could be within the theta range [43],

ranging from 5 to 14 Hz [53], or corresponds to a duration from 170 to 250 ms [62] or 50 to 200 ms [107]. R&K do not explicitly give a criterion of frequency. VSWs mainly occur in late sleep stage 1 and early sleep stage 2, but also REM sleep [62]. However, there is agreement that in multichannel EEG recordings the amplitude of this pattern is maximal at the vertex. If clinically relevant sharp vertex waves have to be distinguished from rolandic spikes in sleep taking into consideration the shape and the distribution of the EEG pattern [74].

#### Summary of the task force

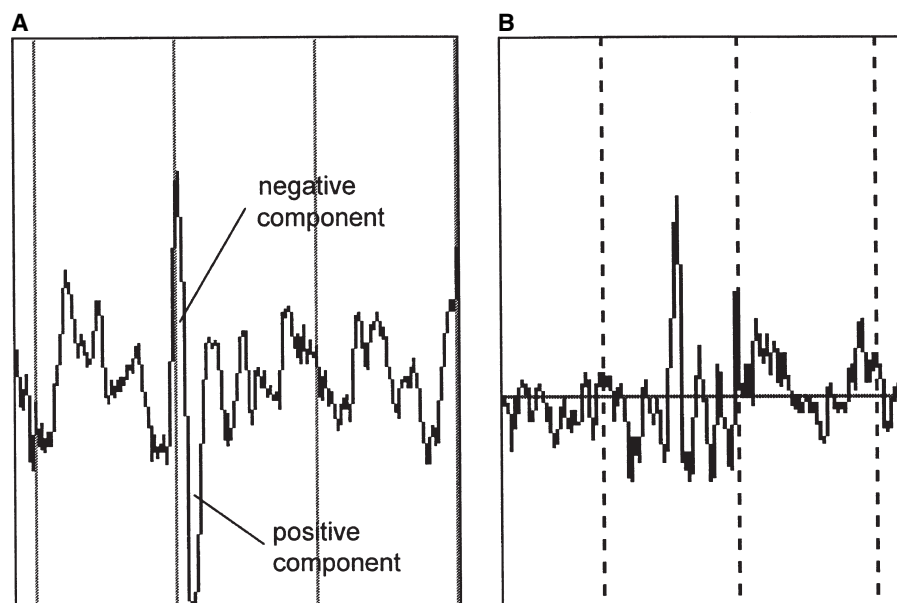
VSWs are not an obligatory pattern when scoring sleep stage 1, but their identification may be helpful, especially at the beginning of the night. They must be carefully distinguished from K-complexes and slow waves.

The following criteria for the recognition of vertex sharp waves are suggested:

- A vertex sharp wave is mono- or biphasic with a large initially negative wave which may be preceded by a small spiky discharge of positive polarity (figure 2).
- In the case of a biphasic VSW, the amplitude of the positive component should be at least 50 % but not more than 100 % of the negative component.
- VSWs may occur in isolation or as trains.
- The overall amplitude of the vertex sharp wave should be at least twice as high as the basic EEG activity within the 5 s preceding a single vertex sharp wave or a train of VSW (figure A4). VSWs should not be scored if they occur within high amplitude theta activity.
- Since R&K did not explicitly state a frequency criterion, it is suggested to use the 4- to 6-Hz frequency range according to Kubicki et al. [62].

## 5. K-complexes

The K-complex is a prominent waveform of the sleep EEG. It was described for the first time by Loomis et al. [68] as 'random wave'. Seventy years later, its functional role is still a matter of debate (for review, see [24, 45]). Although a



**Figure 2.** (A) Example of a biphasic vertex sharp wave (VSW). (B) Example of a monophasic VSW.



K-complex can appear spontaneously, many authors consider it to be an arousal reaction because it can be triggered by external and possibly internal stimuli [59] and it is often followed by a manifest arousal or even an awakening (e.g. [95]). Others assume that K-complexes are an indicator of information processing during sleep and inhibit arousal on the cortical level [24], and thus may have a sleep-protective role. In any case, it is well documented that K-complexes are associated with autonomous activation (e.g. [54]).

K-complexes are the result of synchronized burst firing within extended cortical networks during sleep, which trigger and synchronize other sleep activities in the thalamus (reviewed in [3]). Both the evoked and the spontaneous K-complexes show a frontal maximum [8] and are usually bilaterally symmetrical [26].

Low-amplitude, long-duration K-complexes are apparent at 5 months of age, while the faster negative component appears between 3 and 5 years of age and becomes more pronounced during adolescence [75]. During adult age, the K-complex decreases in frequency and amplitude [27, 65]. The number of K-complexes per time decreases in the course of the night and from cycle to cycle (e.g. [31]), as does the probability to elicit a K-complex by a defined stimulus.

#### Definitions and open questions

Loomis et al. [68, page 426] described the shape of a K-complex as follows: ‘[...]it appears in the record as a swing down (sometimes up and then down) and then up, corresponding to a negativity [...] and then a positivity [...]’. Later on, repeated efforts were made to delineate subtypes of the K-complex, obviously because this pattern is quite variable [34, 59, 80, 88, 109]. However, none of these classifications of K-complexes gained broad acceptance.

R&K defined the K-complex as a prominent waveform having ‘a well delineated negative sharp wave which is immediately followed by a positive component. The total duration of the complex should exceed 0.5 seconds’. They acknowledged that waves of 12 to 14 Hz ‘may or may not constitute a part of the complex’ (figures 3 and A5). They defined the K-complex and the sleep spindles as indicators of the transition from sleep stage 1 and REM to sleep stage 2 (or stages 3 and 4).

Others described a K-complex as a ‘bi-triphasic EEG complex, consisting of an initial rapid negative component and a successive slow wave sometimes fused with final rapid components (sleep spindles)’ [107: p. 57] or ‘the abrupt onset of a negative sharp wave, which is immediately followed by a high amplitude positive slow wave.... The K-complex

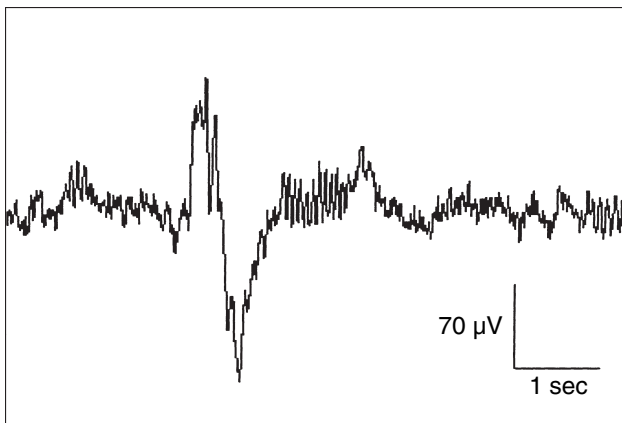


Figure 3. Typical K-complex with subsequent sleep spindle.

waveform may or may not be accompanied by sleep spindle activity’ [53: p. 307]. Although the initial negative component is described as a ‘sharp wave’, judging from the examples in R&K obviously the usual definition of a sharp wave in EEG terminology (duration of 70–200 ms) has not been applied. The recognition of a K-complex becomes even more complicated because ‘polyphasic negative positive waves’ [53: p. 307] or an ‘early fast component with multiphasic aspect’ [107: p. 57] might precede the onset of the negative sharp wave.

For sleep stage scoring, a reliable detection of the first K-complex after the transition from waking to sleep is essential. In the R&K manual, no absolute or relative amplitude criterion for K-complexes is mentioned. Probably in an effort to avoid the scoring of random low-amplitude activity as K-complexes and thus increase the reliability of sleep stage scoring, some groups have suggested the use of a minimum amplitude criterion for K-complexes. Terzano et al. [107] proposed a minimal amplitude of 75  $\mu\text{V}$  with a maximum expression at the vertex, while the Japanese committee [53] suggested the introduction of a minimum peak-to-peak amplitude criterion of 200  $\mu\text{V}$ . This latter group of authors [53] further requested that a K-complex must be clearly distinguishable from the background EEG activity and stated that ‘a waveform should not be considered as a K-complex if it occurs within 5 s preceding or following high voltage delta waves’ [57: p. 307], while R&K requested a differentiation between K-complexes and delta waves whenever possible. The amplitude criterion of Hori and colleagues [53] may result in an artificially low K-complex count in patients with low-voltage EEG.

There is a general agreement in the literature that the minimal duration of a K-complex is 0.5 s with no explicit maximal duration. Since R&K did not define how the duration should be measured, Hori et al. [53: p. 307] defined this duration as the time between rapid onset and ‘the peak of a trailing negative wave, which follows the major positive component, neglecting other overlapping waves’. However, this definition suggested a clear triphasic pattern, which contradicts the definitions of R&K and others [107] describing both bi- and triphasic patterns. When a clear-cut ‘trailing negative wave’ is not present, the definition of Hori et al. [53] creates additional ambiguity.

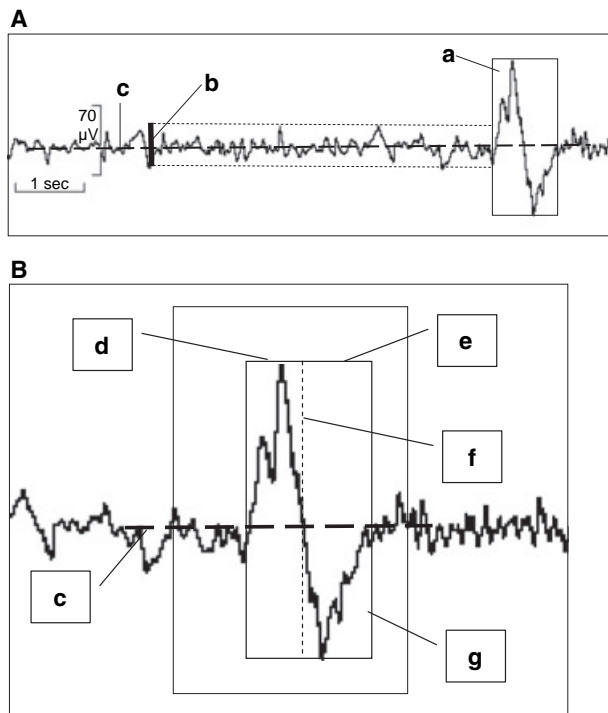
#### Summary of the task force

R&K defined no amplitude criterion for K-complexes, but they repeatedly elaborate on incipient K-complexes and on the necessity to distinguish K-complexes from vertex sharp waves and from other similar activities that do not match the precise wave form of the K-complex. This indicates that they had a relatively restrictive definition of K-complexes in mind. For sleep stage scoring, a reliable detection of the first K-complex after the transition from waking to sleep is essential. Its variable morphology requires a distinction from vertex sharp waves occurring mainly in stage 1 and the delta waves of sleep stages 3 and 4.

The following criteria for the recognition of K-complexes are suggested:

- The K-complex waveform begins with the abrupt onset of a negative sharp wave, which is immediately followed by a high-amplitude positive slow wave (figure 4A). In any case, the duration of the initial negative component must be shorter than that of the subsequent positive component. The onset of the negative component should be clearly identifiable, although polyphasic waves are sometimes





**Figure 4.** Illustration of K-complex criteria

(A) K-complex with surrounding EEG. (a) K-complex (rectangle circumscribes amplitude and duration of K-complex), (b) amplitude of background EEG 5 sec prior to the K-complex, (c) EEG-baseline (B) K-complex, enlarged. (c) EEG-baseline, (d) duration of negative component of K-complex, (e) duration of positive component of K-complex, (f) amplitude of negative component of K-complex, (g) amplitude of positive component of K-complex, (d + e) total duration of K-complex, (f + g) amplitude of K-complex.

For scoring of sleep stages, a K-complex must meet the following criteria (see result section 5):

- (i)  $d > e$  The description of the wave form (see 5.1) must be met.
- (ii)  $f + g \geq b * 2$  Amplitude of K-complex bigger than double of preceding EEG background (see 5.2)
- (iii)  $d + e \geq 0.5$  sec Duration of K-complex longer than 0.5 sec (see 5.3)
- (iv)  $2 * f \geq g$  and  $2 * g \geq f$  Amplitude of the positive component (g) should be at least 50% of the negative component (f) and vice versa (see 5.4)

observed just before the onset of the negative K-complex sharp wave.

- To be clearly distinguishable from background activity, the amplitude should be at least twice as high as the background within 5 s preceding the K-complex (figure 4A). After reviewing a series of samples, the task force found that those waves that were unanimously accepted as K-complexes were generally at least twice as high as the background EEG activity before the event, regardless of the absolute amplitude. Therefore, the task force suggests a relative rather than an absolute (in  $\mu V$ ) amplitude criterion for K-complexes. In patients where K-complexes appear in series [107], the suggestion of *Hori et al.* [53] that K-complexes should not be scored when they are followed by high-amplitude delta waves caused considerable disagreement in the detection of K-complexes. Due to this fact, the task force suggests that only the preceding but not the following EEG amplitude should be taken into consideration.
- The duration of a K-complex must be at least 0.5 s. The duration of a K-complex is measured from the offset of the initial negative wave from baseline to the point where the positive wave reaches baseline again (figure 4B).

- The amplitude of the positive component should be at least 50 % of the negative component and vice versa (figure 4B).

## 6. Sleep spindles

According to R&K, the presence of sleep spindles or K-complexes is the main criterion for scoring sleep stage 2. The first reference to sleep spindles (in short: spindles) was made by *Loomis et al.* [57, 67].

Spindles are generated in thalamic nuclei [103] with the exception of the anterior nuclei group. The nucleus reticularis of the thalamus serves as the pacemaker for spindle generation, thereby using inhibitory mechanisms. Spindles are thought to be expressions of thalamocortical circuits [101]. Spindles are distributed all over the human scalp, but the majority of them appear in central regions [71]. Research employing forced desynchronization protocols showed a moderate sleep-dependent but strong circadian modulation of sigma power (12–15 Hz) with opposite phase positions of the slow (12.25–13 Hz) and fast (14.25–15 Hz) sigma band power [32]. This effect, which was demonstrated in young subjects, is greatly attenuated in the elderly [113]. A bimodal distribution of EEG activity in the sigma range was also shown by other groups [58, 114, 117] with the slower sigma activity ( $\sim 11.5$  to 13/14 Hz) being predominant over the frontal areas, while the faster activity (around 14 Hz) is maximal over midline central and parietal areas.

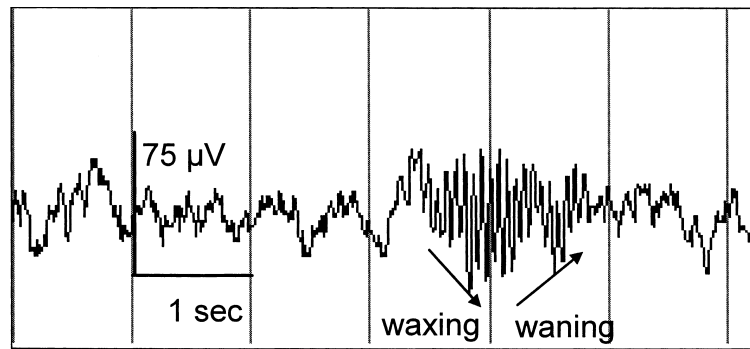
However, the function of spindles thus far remains undefined. Some research findings suggest that they inhibit arousal of the sleeping human and thus preserve sleep continuity [116]. Recent data suggest a role of spindles in memory consolidation [41]. They are thought to depend on neural maturation, because they do not appear before the age of 3 months in the human subject [98]. *Bonnet and Moore* [14] described a close relationship between the first nocturnal occurrence of spindles and the subjective perception of sleep and an increase in the auditory sleep threshold within 1 min after the first occurrence of a spindle.

Spindle periodicity seems to be genetically determined with a large interindividual (3–8/min) and a small intraindividual variation [52]. Amplitude, density, and frequency of spindles decrease with age [85], while no age differences were found for spindle duration [44]. Spindle frequency might be influenced by sleep disorders, neurological disorders, and substances, e.g. benzodiazepines [7, 16, 39]. In particular, the spindle frequency can be elevated above 14 Hz under the influence of benzodiazepines [63, 112].

### Definitions and open questions

R&K defined spindles as 'a transient EEG pattern of waves with a frequency of 12 to 14 Hz of at least 0.5 sec duration, thus one should be able to count 6 to 7 distinct waves within the half-second period' (figures 5 and A6). According to R&K, neither the 'spindle' shape (waxing and waning) nor an amplitude criterion is a requirement for the definition of spindles.

The Italian committee [107: p. 72] defined a spindle as 'sequences of sinusoidal and fusiform waves at 12–15 Hz' with a duration of 0.5 to 2.0 s, amplitude of 5 to 50  $\mu V$ , and a density from 3 to 10 per min. The Japanese Committee [53: p. 307] gave an enlarged frequency range and added also an amplitude criterion ('trains of 12 to 16 Hz waves of 10  $\mu V$  or greater amplitude, composed of at least six consecutive waves, or train duration of longer than 0.5 s'). While both committees follow R&K and did not specify 'spindle shape'



**Figure 5.** Sleep spindle showing the waxing and waning criterion.

as a necessary criterion, *De Gennaro and Ferrara* [30] requested a waxing and waning criterion with a progressive increase followed by a gradually decreasing amplitude presented on a low-voltage background EEG. These transient patterns should last from 0.5 to 3.0 s with Fourier frequency in the 12- to 16-Hz range. A waxing and waning criterion including frequencies from 7 up to 16 Hz was also used by the group around *Steriade* (e.g. [103]).

Despite the fact that spindles originate in the reticular nuclei of the thalamus, a bilaterally symmetrical appearance is neither described nor postulated. There still is a lack of an elaborated canon of criteria, which represents the impact of different factors on the pattern of spindles: age, substances (e.g. benzodiazepines), or neurological disorders.

#### *Summary of the task force*

The identification of sleep spindles is crucial to the scoring of sleep stage 2 and serves to distinguish stage 2 from stage 1 but also from stage REM.

The following criteria for the recognition of sleep spindles are suggested:

- Sleep spindles are defined as trains of waves that are clearly distinguishable from background activity (figure 5) by frequency and the waxing and waning shape. Following R&K, the spindle frequency range should be 12 to 14 Hz, the minimum duration 0.5 s, without defining a maximal duration.
- The minimum peak-to-peak amplitude of spindle should be 10  $\mu$ V (measured from the most negative to the most positive peak of the six highest subsequent waves of the spindle, even if spindles appear superimposed on a slower wave or K-complex; figure 5).
- The spindle frequency range can be altered in neurological diseases and following medication intake. These alterations may be regarded as sleep spindles if the criteria of duration and waxing and waning are met. With respect to the rather limited literature on this topic, exact frequency bounds could not be given here. For the distinction between sleep spindles with lower frequencies and alpha intrusions, the typical spindle shape and transient occurrence (on average 3–8/min) of sleep spindles becomes an important additional criterion.

## 7. Sawtooth waves

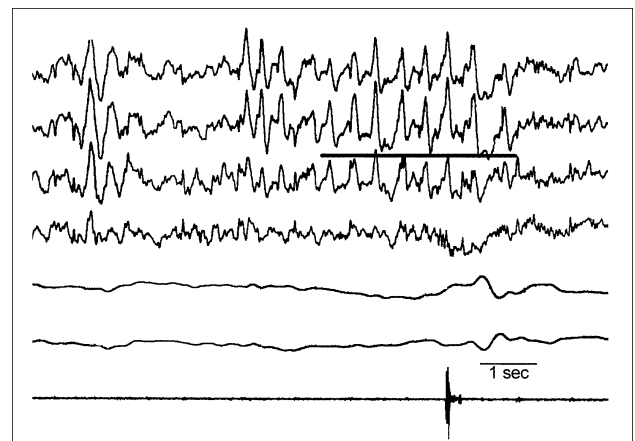
Sawtooth waves (STW) are the only typical EEG pattern of REM sleep. *Berger et al.* [12] and *Schwartz* [93], who first described STW, observed a temporal association with series of rapid eye movements. There are only few studies on the localization of STW. A maximum of STW over the vertex

and frontal brain areas was already mentioned in early studies [12, 60, 93]. This topographic distribution pattern of STW, with a local maximum at the midline, was later confirmed in a small-scale EEG mapping study [18]. *Sato et al.* [89] found a regular temporal relationship between the occurrence of STW and the reduction of muscle tone at the beginning of REM sleep. Within a time window of 1000 s, there emerged a typical sequence of (i) a generalized body movement followed by (ii) a drop in muscle tone and (iii) STW, and finally (iv) the occurrence of the first rapid eye movements. The first STW occurred already in sleep stage 2, shortly before REM sleep onset. In an own study, we found a specific temporal distribution of STW density with an increase in the last 4 to 5 min before REM sleep, a maximum in the first few minutes of REM sleep, followed by a drop to lower values [92]. In normal sleeping persons, STW density is lowest in the first REM sleep phase and increases in later REM sleep phases [42, 82]. STW density was found to be higher in patients with narcolepsy [42].

The function of STW is unknown, as well as its course over the lifespan.

#### *Definitions and open questions*

STW are defined by R&K as a ‘series of three or more low to medium amplitude EEG waves in the frequency range 2 to 6 Hz with an initial slow increase, followed by a steep decrease’ (figure 6), which gives the waves a typical notched or sawtooth appearance (figures 6 and A7). This definition thereby following the proposals of *Berger et al.* [12] leaves some room for interpretation because neither amplitude (‘low to medium’) nor the typical notched or sawtooth appearance



**Figure 6.** Sawtooth waves (STWs) in REM sleep. From top to bottom, four EEGs (F3, C3, C4, O2), left and right EOG, and mental EMG. Eight seconds recording time; STWs are underlined.

was explained in detail. While *Hori et al.* [53] did not define STW, the Italian committee [107: p. 90] requested 'trains of three or more angular waves at 2 to 5 Hz frequency, of 20 to 100  $\mu\text{V}$  amplitude'. They further stated that 'conventionally at least two waves of the train must have the following morphology: a first slow negative peak followed by a sharp decline culminating in a positive peak'. The most precise definition was given by *Geisler et al.* [42], who accepted STW if three or more EEG waves with a frequency of 2–5 Hz and an amplitude of 20 to 100  $\mu\text{V}$  had a parallel decreasing slope of more than 80 degrees, given a paper speed of 10 mm/s (figure A7). In a sample of 20 normal sleepers, *Pearl et al.* [82] found an STW density of 0.97 events per minute in REM sleep. The mean frequency of STW was 2.5 Hz with a scatter of 1.5 to 5 Hz. STW occurred in groups with a mean duration of 7 s and a range of 2–26 s. As a further criterion, *Aldrich* [2] mentioned the topographic distribution of STW with a maximum over frontal and central brain regions.

#### Summary of the task force

STWs are the only typical EEG pattern of REM sleep. They are helpful in indicating an attempt of the organism to enter REM sleep, although they are not essential to score REM sleep.

The following criteria for the recognition of sawtooth waves are suggested (figure 6):

- STW are defined as a series (burst) of at least three or more waves with a triangular shape.
- The beginning of an STW is characterized by a notched negative wave of 20–100  $\mu\text{V}$ , while it ends with a decreasing slope of more than 80 degrees (by a given paper speed of 10 mm/s or the same temporal resolution for displaying digital recordings; calibration amplitude: 70  $\mu\text{V}/1\text{ cm}$ ; time constant: 0.3 s). The steepness of the decreasing slope should be 284  $\mu\text{V}/\text{sec}$  or more. This slope should be similar in all STW within one burst.
- STW should be in the frequency range of 1.5 to 5 Hz [82]. Each sawtooth wave starts and ends at the most positive point.
- If STW is used in the context of visual sleep staging, and not for scientific purposes, a less stringent definition of STW-like activity may be helpful to recognize and delineate REM sleep, especially when other criteria of REM sleep are not well pronounced.

#### Discussion

The objective of the present paper was to collect and compare definitions and scoring rules for EEG patterns in sleep. The present amended scoring rules were restricted to EEG patterns that are relevant for sleep scoring, except EEG arousals and non-EEG patterns. Besides R&K, other definitions are considered insofar as the manual of R&K did not dispose a precise definition.

The intention of the task force was not to revise the rules of R&K, but to facilitate their reliable application in sleep scoring. All suggestions refer to R&K-relevant EEG derivations (C3-A2 and C4-A1) and standard EEG amplifier settings, with the additional proposal to add an occipital lead for a better recognition of alpha waves. Age and gender effects were not specified within the amended rules. Individual differences in the EEG sleep were considered by applying a relative rather than an absolute amplitude criterion, with the exception of slow waves.

#### Future perspectives

Further developments of scoring rules should focus on the following topics:

1. Differences between clinically defined frequency bands of the waking EEG and the EEG frequency bands of sleep should be harmonized.
2. Empirical evidence for age- and gender-dependent variations of EEG patterns should be compiled and integrated into updated recommendations.
3. The influence of different sleep disorders and other clinical conditions, as well as the potential influence of pharmacological substances on EEG patterns in sleep, should be regarded.
4. A concerted effort should be made to discuss the applicability of the amended rules in the context of computerized sleep analysis, by bringing together experts in visual sleep scoring and in automatic sleep analysis.

#### Acknowledgement

We thank Stephany Fulda for her very helpful, sustaining, and inspiring comments when preparing this manuscript.

#### References

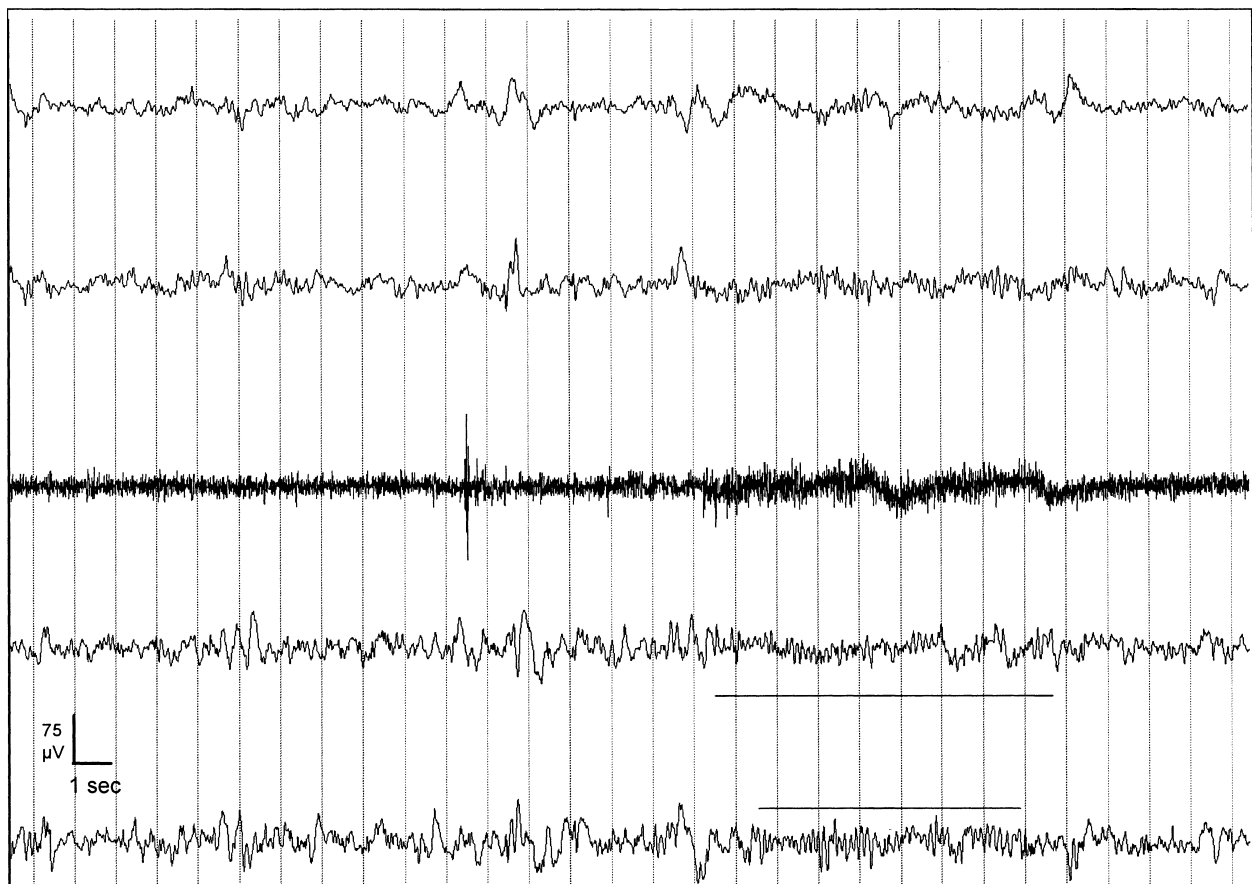
- [1] Aeschbach D, Matthews JR, Postolache TT, Jackson MA, Giesen HA, Wehr TA: Dynamics of the human EEG during prolonged wakefulness: evidence for frequency-specific circadian and homeostatic influences. *Neurosci Lett* 239: 121–124, 1997.
- [2] Aldrich M: Sawtooth waves. In: Carskadon MA (ed.): *Encyclopedia of sleep and dreaming*. Macmillan, New York, pp 527–528, 1993.
- [3] Amzica F, Steriade M: The functional significance of K-complexes. *Sleep Med Rev* 6: 139–149, 2002.
- [4] Anders T, Emde R, Parmelee A: A manual of standardized terminology, techniques, and criteria for scoring of states of sleep and wakefulness in newborn infants. UCLA Brain Information Service, NINDS Neurological Information Network, Los Angeles, 1971.
- [5] Armitage R, Baker FC, Parry BL: The menstrual cycle and circadian rhythms. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice in sleep medicine*. 4th edition, Saunders, Philadelphia, pp 1266–1277, 2005.
- [6] Atlas Task Force of the American Sleep Disorders Association: EEG-arousals: scoring rules and examples. A preliminary report from the sleep disorders. *Sleep* 15: 173–184, 1992.
- [7] Bassetti CL: Sleep and stroke. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice of sleep medicine*. 4th edition, Saunders, Philadelphia, pp 811–830, 2005.
- [8] Bastien C, Campbell K: The evoked K-complex: all-or-none phenomenon? *Sleep* 15: 236–245, 1992.
- [9] Bastien CH, Crowley KE, Colrain IM: Evoked potential components unique to non-REM sleep: relationship to evoked K-complexes and vertex sharp waves. *Int J Psychophysiol* 46: 257–274, 2002.
- [10] Beier KM, Kubicki S: Kortikale Verteilung zweier  $\delta$ -Frequenzen im langsamen Schlaf. *Z EEG-EMG* 18: 47–51, 1987.
- [11] Berger H: Über das Elektroencephalogramm des Menschen. *Arch Psychiat Nervenkr* 87: 527–570, 1929.
- [12] Berger RJ, Olley P, Oswald I: The EEG, eye movements and dreams of the blind. *Q J Exp Psychol* 14: 192–186, 1962.
- [13] Bliwise DL: Normal aging. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice in sleep medicine*. 4th edition, Saunders, Philadelphia, pp 24–38, 2005.
- [14] Bonnet MH, Moore SE: The threshold of sleep: perception of sleep as a function of time asleep and auditory threshold. *Sleep* 5: 267–276, 1982.

- [15] Borbély AA: A two process model of sleep regulation. *Hum Neurobiol* 1: 195–204, 1982.
- [16] Bové A, Culebras A, Moore JT, Westlake RE: Relationship between sleep spindles and hypersomnia. *Sleep* 17: 449–455, 1994.
- [17] Brazier MA: Preliminary proposal for an EEG terminology by the Terminology Committee of the International Federation for Electroencephalography and Clinical Neurophysiology. *Electroencephalogr Clin Neurophysiol* 13: 646–650, 1961.
- [18] Broughton R, Hasan J: Quantitative topographic electroencephalographic mapping during drowsiness and sleep onset. *J Clin Neurophysiol* 12: 372–386, 1995.
- [19] Butkov N: Atlas of clinical polysomnography. Vol I & II. Synapse Media, Medford, OR, 1996.
- [20] Cantero JL, Atienza M, Salas RM: Spectral features of EEG alpha activity in human REM sleep: two variants with different functional roles? *Sleep* 23: 746–750, 2000.
- [21] Cape EG, Manns ID, Alonso A, Beaudet A, Jones BE: Neurotensin-induced bursting of cholinergic forebrain neurons promotes gamma and theta cortical activity together with waking and paradoxical sleep. *J Neurosci* 20: 8452–8461, 2000.
- [22] Carskadon MA, Rechtschaffen A: Monitoring and staging human sleep. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice in sleep medicine*. 4th edition, Saunders, Philadelphia, pp 1359–1393, 2005.
- [23] Clarenbach P (ed.): *Schering-Lexikon Schlafmedizin*. 2nd edition, MMV Medizin Verlag, Munich, 1998.
- [24] Colrain IM: The K-complex: a 7-decade history. *Sleep* 28: 255–273, 2005.
- [25] Colrain IM, Webster KE, Irst GH, Campbell KB: The roles of vertex sharp waves and K-complexes in the generation of N300 in auditory and respiratory-related evoked potentials during early stage 2 NREM sleep. *Sleep* 23: 97–106, 2000.
- [26] Cote KA, de Lugt DR, Langley SD, Campbell KB: Scalp topography on the auditory evoked K-complex in stage 2 and slow wave sleep. *J Sleep Res* 8: 263–272, 1999.
- [27] Crowley K, Trinder J, Kim Y, Carrington M, Colrain IM: The effects of normal aging on sleep spindle and K-complex production. *Clin Neurophysiol* 113: 1615–1622, 2002.
- [28] Daan S, Beersma DGM, Borbély AA: The timing of human sleep: recovery process gated by a circadian pacemaker. *Am J Physiol* 246: R161–R178, 1984.
- [29] Danker-Hopfe H, Kunz D, Gruber G, Klösch G, Lorenzo JL, Himanen SL, Kemp B, Penzel T, Röschke J, Dorn H, Schlögl A, Trenker E, Dorfner G: Interrater reliability between scorers from eight European sleep laboratories in subjects with different sleep disorders. *J Sleep Res* 13: 63–69, 2004.
- [30] De Gennaro L, Ferrara M: Sleep spindles: an overview. *Sleep Med Rev* 7: 423–440, 2003.
- [31] De Gennaro L, Ferrara M, Bertini M: The spontaneous K-complex during stage 2 sleep: is it a ‘forerunner’ of delta waves? *Neurosci Lett* 291: 41–43, 2000.
- [32] Dijk DJ, Shanahan TL, Duffy JF, Ronda JM, Czeisler CA: Variation of electroencephalographic activity during non-rapid eye movement and rapid eye movement sleep with phase of circadian melatonin rhythm in humans. *J Physiol* 505: 851–858, 1997.
- [33] Duffy FH, Albert MS, McAnulty G, Garvey AJ: Age-related differences in brain electrical activity of healthy subjects. *Ann Neurol* 16: 430–438, 1984.
- [34] Ehrhart J, Ehrhart M, Muzet A, Schieber JP, Naitoh P: K-complexes and sleep spindles before transient activation during sleep. *Sleep* 4: 400–407, 1981.
- [35] Epstein HT: EEG developmental stages. *Develop Psychol* 13: 629–631, 1980.
- [36] Feinberg I, Floyd TC: Systematic trends across the night in human sleep cycles. *Psychophysiol* 16: 283–291, 1979.
- [37] Finelli LA, Baumann H, Borbély AA, Achermann P: Dual electroencephalogram markers of human sleep homeostasis: correlation between theta activity in waking and slow-wave activity in sleep. *Neuroscience* 101: 523–529, 2000.
- [38] Franken P, Malafosse A, Tafti M: Genetic variation in EEG activity during sleep in inbred mice. *Am J Physiol* 275: R1127–R1137, 1998.
- [39] Gaillard JM, Blois R: Spindle density in sleep of normal subjects. *Sleep* 4: 385–391, 1981.
- [40] Gais S, Born J: Declarative memory consolidation: mechanisms acting during human sleep. *Learn Mem* 11: 679–685, 2004.
- [41] Gais S, Molle M, Helms K, Born J: Learning-dependent increase in sleep spindle density. *J Neurosci* 22: 6830–6834, 2002.
- [42] Geisler P, Meier-Ewert K, Matsubayashi K: Rapid eye movements, muscle twitches and sawtooth waves in the sleep of narcoleptic patients and controls. *Electroencephal Clin Neurophysiol* 67: 499–507, 1987.
- [43] Geyer JD, Payne TA, Carney PR, Aldrich MS: Atlas of digital polysomnography. Lippincott Williams & Wilkins, Philadelphia, 2000.
- [44] Guazzelli M, Feinberg I, Aminoff M, Fein G, Floyd TC, Maggini C: Sleep spindles in normal elderly: comparison with young adult patterns and relation to nocturnal awakening, cognitive function and brain atrophy. *Electroencephal Clin Neurophysiol* 63: 526–539, 1986.
- [45] Halász P: K-complex, a reactive EEG graphoelement of NREM sleep: an old chap in a new garment. *Sleep Med Rev* 9: 391–412, 2005.
- [46] Harris CD: Neurophysiology of sleep and wakefulness. *Respir Care Clin* 11: 567–586, 2005.
- [47] Harsh J, Voss U, Hull J, Schrepfer S, Badia P: ERP and behavioural changes during the wake/sleep transition. *Psychophysiol* 31: 244–252, 1994.
- [48] Hauri P, Hawkins DR: Alpha-delta sleep. *Electroencephal Clin Neurophysiol* 34: 233–237, 1973.
- [49] Herrmann WM, Schäfer E: *Pharmako-EEG*. Ecomed, Landsberg, pp 14–15, 1987.
- [50] Himanen SL, Hasan J: Limitations of Rechtschaffen and Kales. *Sleep Med Rev* 4: 149–167, 2000.
- [51] Hori T, Hayashi M, Morikawa T: Topographic EEG changes and the hypnagogic experience. In: Ogilvie RD, Harsh (eds): *Sleep onset: normal and abnormal processes*. American Psychological Association, Washington, DC, 1994.
- [52] Hori A, Kazukawa S, Endo M, Kurachi M: Sleep spindles in twins. *Clin Electroencephal* 20: 121–127, 1989.
- [53] Hori T, Sugita Y, Koga E, Shirakawa S, Inoue K, Uchida S, Kuwahara H, Kousaka M, Kobayashi T, Tsuji Y, Terashima M, Fukuda K, Fukuda N: Proposed supplements and amendments to ‘A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects’, the Rechtschaffen & Kales (1968) standard. *Psychiatry Clin Neurosci* 55: 305–310, 2001.
- [54] Hornyak M, Cejnar M, Elam M, Matousek M, Wallin BG: Sympathetic muscle nerve activity during sleep in man. *Brain* 114: 1281–1295, 1991.
- [55] Hughes JR: The development of the vertex sharp transient. *Clin Electroencephal* 29: 183–187, 1998.
- [56] Hughes SW, Lörincz M, Cope DW, Blethyn KL, Kékesi KA, Parri R, Juhász G, Crunelli V: Synchronized oscillations at  $\alpha$  and  $\theta$  frequencies in the lateral geniculate nucleus. *Neuron* 42: 253–268, 2004.
- [57] Jankel WR, Niedermeyer E: Sleep spindles. *J Clin Neurophysiol* 2: 1–35, 1985.
- [58] Jobert M, Poiseau E, Jähmig P, Schulz H, Kubicki S: Topographical analysis of sleep spindle activity. *Neuropsychobiol* 26: 210–217, 1992.
- [59] Johnson LC, Karpan WE: Autonomic correlates of the spontaneous K-complex. *Psychophysiol* 4: 444–452, 1968.
- [60] Jouviet M, Michel F, Mounier D: Analyse électroencéphalographique comparée du sommeil physiologique chez le chat et chez l’homme. *Rev Neurol* 103: 189–204, 1960.

- [61] Knowles JB, MacLean AW, Salem L, Vetere C, Coulter M: Slow-wave sleep in daytime and nocturnal sleep: an estimate of the time course of 'Process S'. *J Biol Rhythms* 1: 303–308, 1986.
- [62] Kubicki S: Vigilanz und Schlaf. In: Zschocke S (ed.): *Klinische Elektroenzephalographie*, 2nd edition, Springer, Berlin, 2002.
- [63] Kubicki S, Haag-Wüsthoff C, Röhmel J, Herrmann WM, Scheuler W: The pharmacodynamic influence of three benzodiazepines on rapid eye movements, K-complexes and sleep spindles in healthy volunteers. *Hum Psychopharmacol* 3: 247–255, 1988.
- [64] Kubicki S, Herrmann WM, Höller L, Scheuler W: Kritische Bemerkungen zu den Regeln von Rechtschaffen und Kales über die visuelle Auswertung von Schlaf-EEG-Aufzeichnungen. *EEG EMG Z* 13: 51–60, 1982.
- [65] Kubicki S, Scheuler W, Jobert M, Pastelak-Price C: Der Einfluß des Alters auf die Schlafspindel- und K-Komplex-Dichte. *Z EEG-EMG* 20: 59–63, 1989.
- [66] Liberson WT: Problem of sleep and mental disease. *Digest Neurol Psychiat* 12: 93–108, 1944.
- [67] Loomis AL, Harvey EN, Hobart GA: Electrical potentials of the human brain. *J Exp Psychol* 19: 249–279, 1936.
- [68] Loomis AL, Harvey EN, Hobart GA: Distribution of disturbance patterns in the human electroencephalogram, with special reference to sleep. *J Neurophysiol* 1: 413–430, 1938.
- [69] MacLean AW, Lue F, Moldofsky H: The reliability of visual scoring of alpha EEG activity during sleep. *Sleep* 18: 565–569, 1995.
- [70] Maret S, Franken P, Dauvilliers Y, Ghyselinck NB, Chambon P, Tafti M: Retinoic acid signalling affects cortical synchrony during sleep. *Science* 310: 111–113, 2005.
- [71] McCormick L, Nielsen T, Nicolas A, Ptito M, Montplaisir J: Topographical distribution of spindles and K-complexes in normal subjects. *Sleep* 20(1): 939–941, 1997.
- [72] Merica H, Fortune RD: State transitions between wake and sleep, and within the ultradian cycle, with focus on the link to neuronal activity. *Sleep Med Rev* 8: 473–485, 2004.
- [73] Nicholas CL, Trinder J, Colrain IM: Increased production of evoked and spontaneous K-complexes following a night of fragmented sleep. *Sleep* 25: 42–47, 2002.
- [74] Niedermeyer E: Sleep and EEG. In: Niedermeyer E, Lopes da Silva F (eds.): *Electroencephalography: basic principles, clinical applications, and related fields*. Williams and Wilkins, Baltimore, pp 193–207, 2005.
- [75] Niedermeyer E: Maturation of the EEG: development of waking and sleeping patterns. In: Niedermeyer E, Lopes da Silva F (eds.): *Electroencephalography: basic principles, clinical applications, and related fields*. Williams and Wilkins, Baltimore, pp 167–191, 1993.
- [76] Noachtar S, Binnie C, Ebersole J, Mauguière F, Sakamoto A, Westmoreland B: A glossary of terms most commonly used by clinical electroencephalographers and proposal for the report form for the EEG findings. *The International Federation of Clinical Neurophysiology. Electroencephalogr Clin Neurophysiol Suppl* 52: 21–41, 1999.
- [77] Noachtar S, Binnie C, Ebersole J, Mauguière F, Sakamoto A, Westmoreland B: Glossar der meistgebrauchten Begriffe in der klinischen Elektroenzephalographie und Vorschläge für die EEG-Befunderstellung. *Klin Neurophysiol* 35: 5–21, 2004.
- [78] Norman RG, Pal I, Steward C, Walsleben JA, Rapoport DM: Interobserver agreement among sleep scorers from different centers in a large dataset. *Sleep* 23: 901–908, 2000.
- [79] Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV: Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep* 27: 1255–1273, 2004.
- [80] Paiva T, Rosa A: K-complex variability in normal subjects. In: Terzano MG, Halász P, Declerck AC (eds.): *Phasic events and dynamic organization of sleep*. Raven Press, New York, pp 167–184, 1991.
- [81] Pascualy R, Buchwald D: Chronic fatigue syndrome and fibromyalgia. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice of sleep medicine*. 3rd edition, Saunders, Philadelphia, pp 1040–1050, 2000.
- [82] Pearl PL, LaFleur BJ, Reigle SC, Rich AS, Freeman AA, McCutchen C, Sato S: Sawtooth wave density analysis during REM sleep in normal volunteers. *Sleep Med* 3: 255–258, 2002.
- [83] Penzel T, Behler PG, von Buttler M, Conradt R, Meier M, Möller A, Danker-Hopfe H: Reliabilität der visuellen Schlafauswertung nach *Rechtschaffen* und *Kales* von acht Aufzeichnungen durch neun Schlaflabore. *Somnologie* 7: 49–58, 2003.
- [84] Peszka J, Harsh J: Effect of sleep deprivation on NREM sleep ERPs and related activity at sleep onset. *Int J Psychophysiology* 46: 275–286, 2002.
- [85] Principe JC, Smith JR: Sleep spindle characteristics as a function of age. *Sleep* 5: 73–84, 1982.
- [86] Rechtschaffen A, Kales A (eds.): *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. BIS/BRI, UCLA, Los Angeles, 1968.
- [87] Rechtschaffen A, Kales A (eds.): *Ein Manual der standardisierten Terminologie, Techniken und Auswertung der Schlafstadien beim Menschen*. Dtsch. Übersetzung. In: *Kompodium Schlafmedizin*. ecomed, Landsberg/Lech, 2002.
- [88] Roth B: The clinical and theoretical importance of EEG rhythms corresponding to states of lowered vigilance. *EEG Clin Neurophysiol* 13: 395–399, 1961.
- [89] Sato S, McCutchen C, Graham B, Freeman A, von Albertini-Carletti I, Alling DW: Relationship between muscle tone changes, sawtooth waves and rapid eye movements during sleep. *EEG Clin Neurophysiol* 103: 627–632, 1997.
- [90] Scheuler W, Stinshoff D, Kubicki S: The alpha-sleep pattern. Differentiation from other sleep patterns and effect of hypnotics. *Neuropsychobiol* 10: 183–189, 1983.
- [91] Schneider-Helmert D, Kumar A: Sleep, its subjective perception, and daytime performance in insomniacs with pattern of alpha sleep. *Biol Psychiat* 37: 99–105, 1995.
- [91a] Scholle S, Schäfer T: Atlas of states of sleep and wakefulness in infants and children. *Somnologie* 3: 163–241, 1999.
- [92] Schulz H, Meier J, Walther BW: The temporal distribution of sawtooth waves at the onset of REM sleep. *Somnologie* 9 (Suppl 1): 48, 2005.
- [93] Schwartz B: EEG et mouvement oculaires dans le sommeil de nuit. *EEG Clin Neurophysiol* 14: 126–128, 1962.
- [94] Sekine A, Niiyama Y, Kutsuzawa O, Shimizu T: A negative component superimposed on event-related potentials during light drowsiness. *Psychiat Clin Neurosci* 55: 473–478, 2001.
- [95] Sforza E, Jouny C, Ibanez V: Cardiac activation during arousal in humans: further evidence for hierarchy in the arousal response. *Clin Neurophysiol* 111: 1611–1619, 2000.
- [96] Sheldon SH: *Evaluating sleep in infants and children*. Lippincott-Raven, Philadelphia, 1996.
- [97] Shepard JW: *Atlas of sleep medicine*. Futura Publishing, New York, 1991.
- [98] Shibagaki M, Kijono S, Watanabe K: Spindle evolution in normal and mentally retarded children: a review. *Sleep* 5: 47–57, 1982.
- [99] Smit DJ, Posthuma D, Boomsma DI, Geus EJ: Heritability of background EEG across the power spectrum. *Psychophysiol* 42: 691–697, 2005.
- [100] Stampi C, Stone P, Michimori A: The alpha attenuation test: a new quantitative method for assessing sleepiness and its relationship to the MSLT. *Sleep Res* 22: 115, 1993.
- [101] Steriade M: Brain electrical activity and sensory processing during waking and sleep states. In: Kryger MH, Roth T, Dement WC (eds.): *Principles and practice of sleep medicine*. 4th edition, Elsevier, Philadelphia, pp 101–119, 2005.
- [102] Steriade M, Contreras D, Curro-Dossi R, Nunez A: The slow (<1 Hz) oscillation in reticular thalamic and thalamocortical neurons: scenario of sleep rhythm generation in interacting thalamic and neocortical networks. *J Neurosci* 13: 3284–3299, 1993.

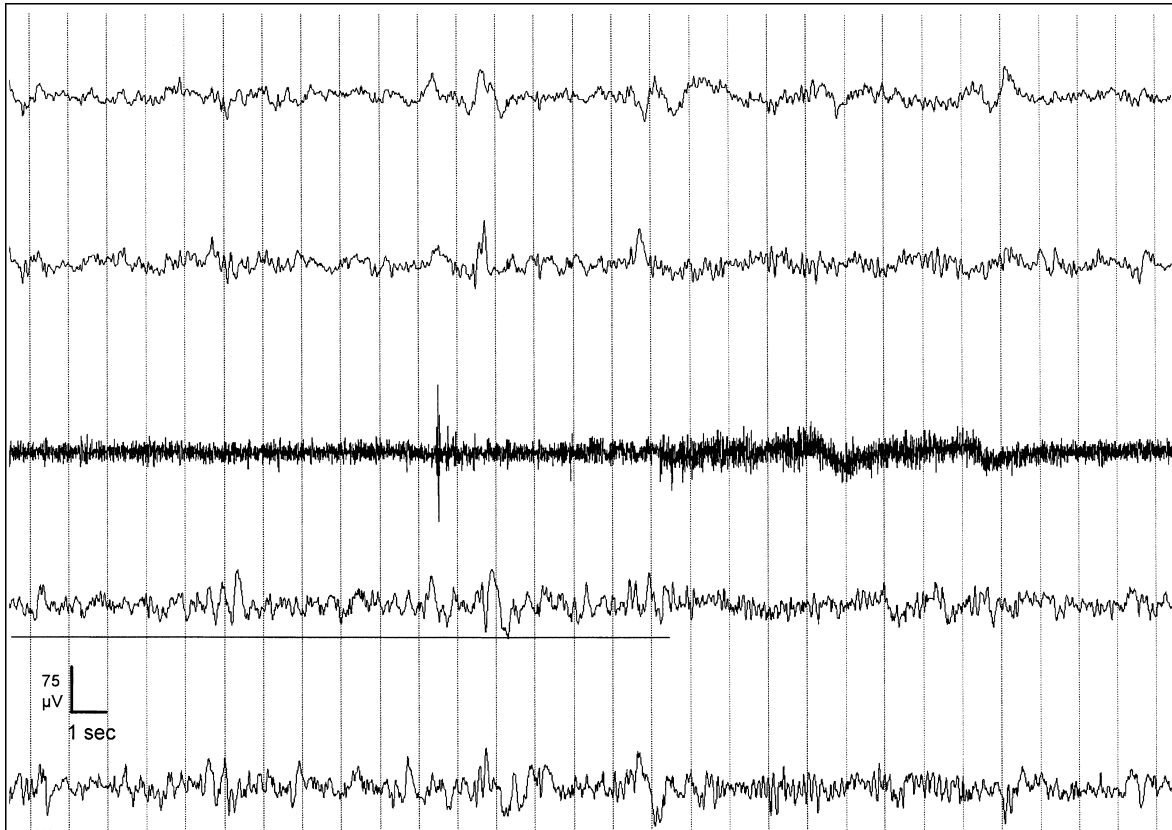
- [103] Steriade M, Domich L, Oakson G, Deschenes M: The deafferented reticular thalamic nucleus generates spindle rhythmicity. *Neurophysiol* 57: 260–273, 1987.
- [104] Steriade M, McCarley RW: Brainstem control of wakefulness and sleep. Plenum Press, New York, 1990.
- [105] Tafti M, Petit B, Chollet D, Neidhart E, de Bilbao F, Kiss JZ, Wood PA, Franken P: Deficiency in short-chain fatty acid beta-oxidation affects theta oscillations during sleep. *Nat Gen* 34: 320–325, 2003.
- [106] Tanaka H, Hayashi M, Hori T: Topographic characteristics and principal component structure of the hypnagogic EEG. *Sleep* 20: 523–534, 1997.
- [107] Terzano MG, Parrino L, Mennuni GF (eds.): *Eventi Fasicci e Microstruttura del Sonno/Phasic Events and Microstructure of Sleep (Consensus Conference)*. Martano Editore, Lecce, Italy, 1997.
- [108] Tononi G, Cirelli C: Sleep function and synaptic homeostasis. *Sleep Med Rev* 10: 49–62, 2006.
- [109] Ujszászi J, Halász P: Late component variants of single auditory evoked responses during NREM sleep stage 2 in man. *EEG Clin Neurophysiol* 64: 260–268, 1986.
- [110] Walter WG: The electroencephalogram in cases of cerebral tumor. *Proc R Soc Med* 30: 579–598, 1937.
- [111] Walter WG, Dovey VJ: Electroencephalography in cases of sub-cortical tumor. *J Neurol Neurosurg Psychiat* 7: 57–65, 1944.
- [112] Weeß H-G, Steinberg R: Hypothese für einen cholinergen Mechanismus beim BZD-Entzug. In: Baumann (ed.): *Biologische Psychiatrie der Gegenwart*. Springer, Berlin, pp 626–629, 1993.
- [113] Wei HG, Riel E, Czeisler CA, Dijk DJ: Attenuated amplitude of circadian and sleep-dependent modulation of electroencephalographic sleep spindle characteristics in elderly human subjects. *Neurosci Lett* 260: 29–32, 1999.
- [114] Werth E, Achermann P, Dijk DJ, Borbély AA: Spindle frequency activity in the sleep EEG: individual differences and topographic distribution. *EEG Clin Neurophysiol* 103: 535–542, 1997.
- [115] Williams RL, Karacan I, Hirsch CJ: *Electroencephalography (EEG) of human sleep: clinical applications*. Wiley, New York, 1974.
- [116] Yamadori A: Role of spindles in the onset of sleep. *J Med Sci* 17: 97–111, 1971.
- [117] Zeitlhofer J, Gruber G, Anderer P, Asenbaum S, Schimicek P, Saletu B: Topographic distribution of sleep spindles in young healthy subjects. *J Sleep Res* 6: 149–155, 1997.
- [118] Zschocke S: *Klinische Elektroenzephalographie*. Springer, Berlin, 2002.
- [119] Zumsteg D, Hungerbühler H, Wieser HG: *Atlas of adult electroencephalography*. Hippocampus, Bad Honnef, 2004.

## Appendix

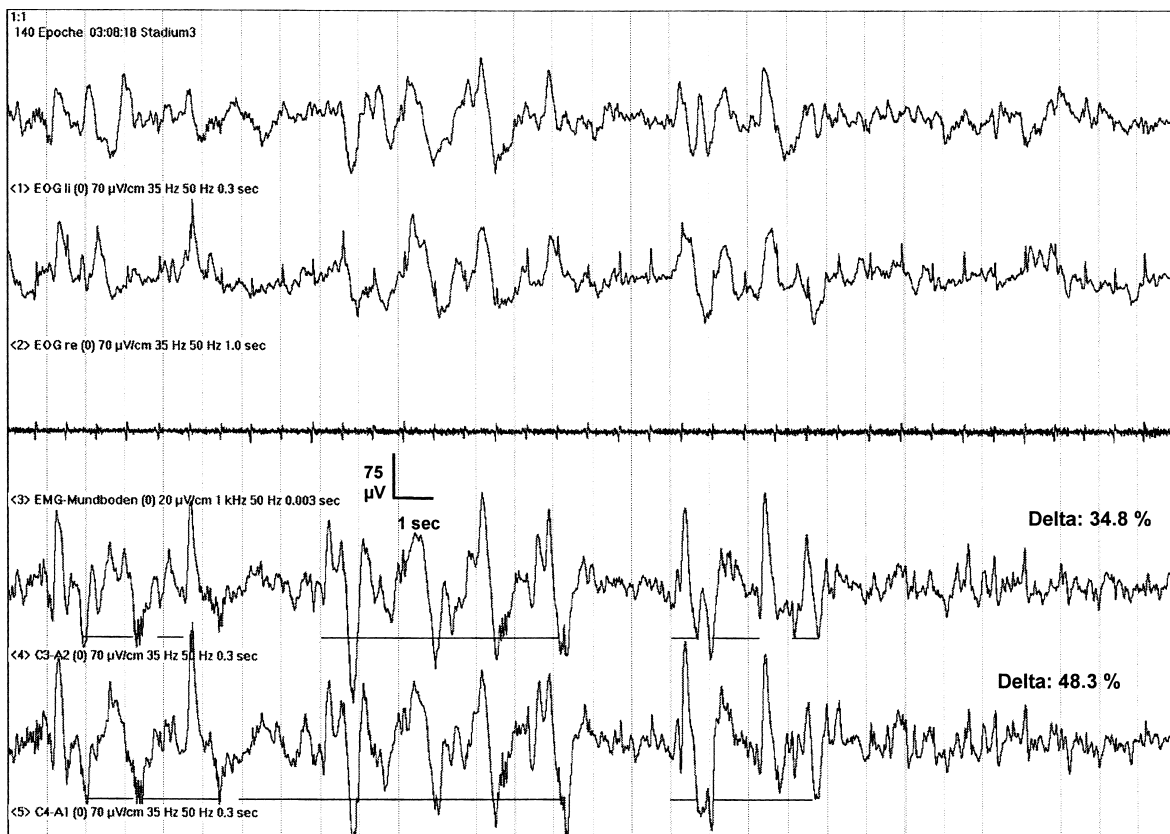


**Figure A1.** One epoch (30 s) with marked alpha activity. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (mental EMG), and two EEGs (C3-A2, C4-A1).

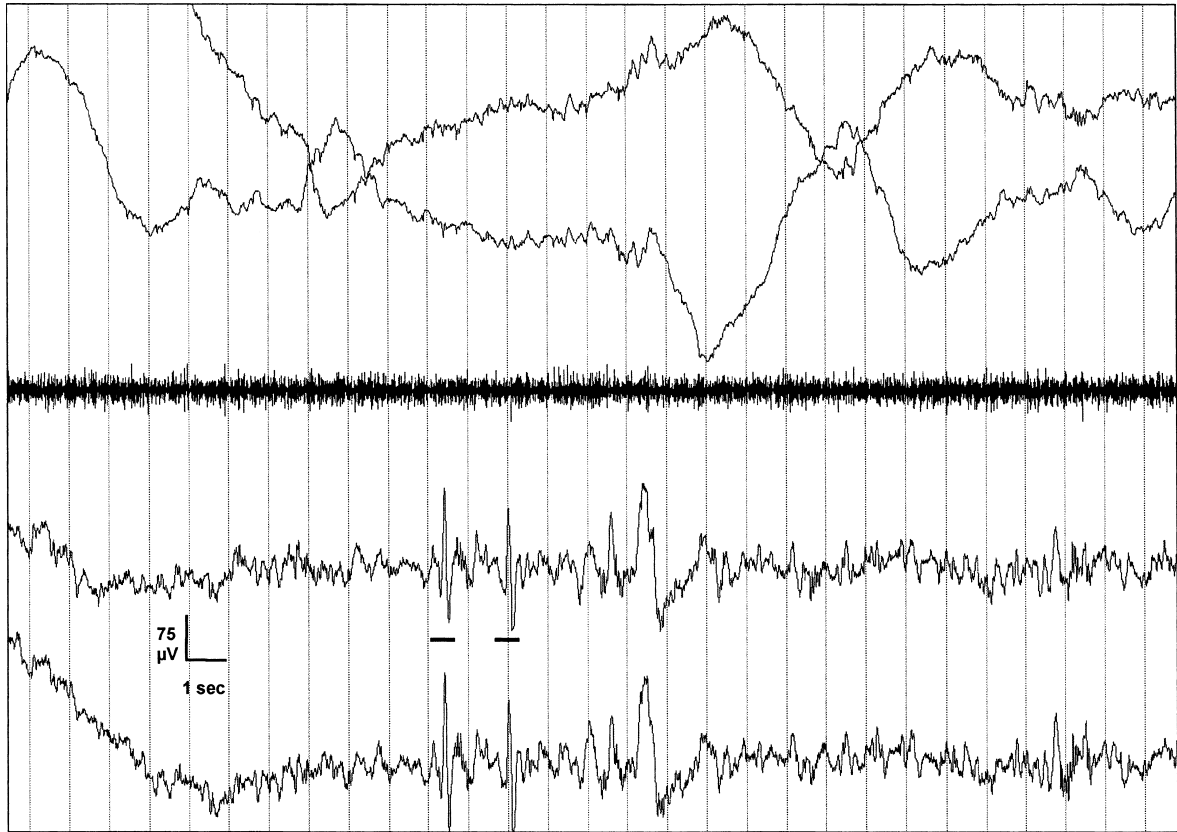




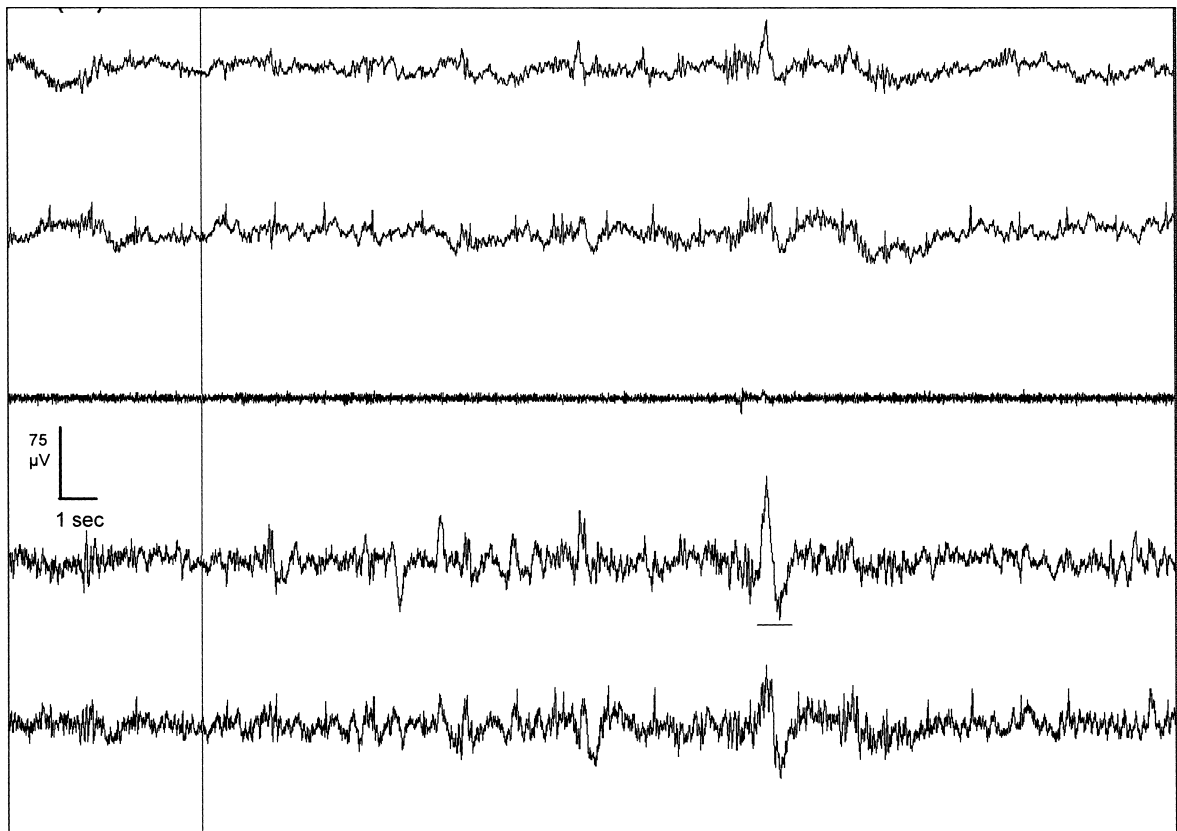
**Figure A2.** One epoch (30 s) with marked theta activity. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (mental EMG), and two EEGs (C3-A2, C4-A1).



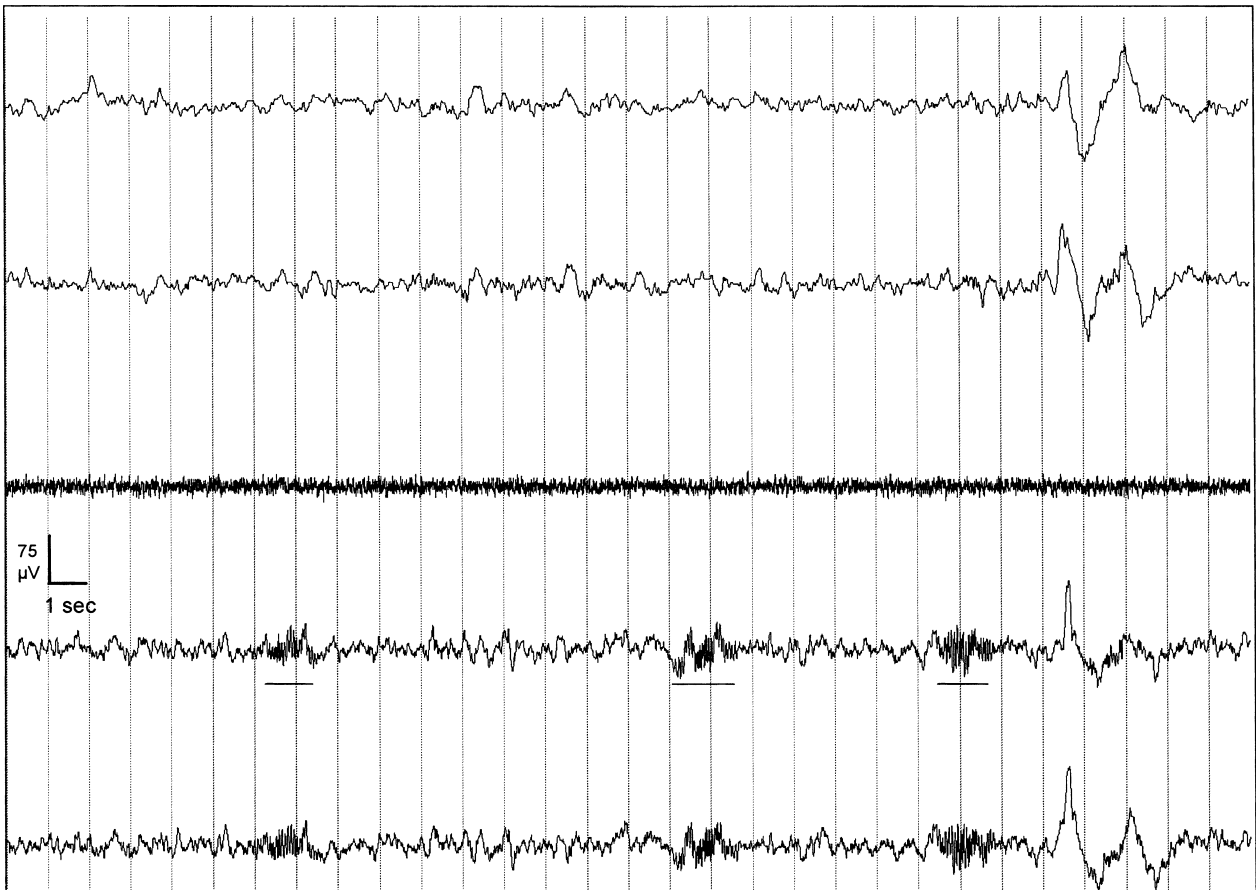
**Figure A3.** One epoch (30 s) with slow wave activity during SWS; slow waves are underlined. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (submental EMG), and two EEGs (C3-A2, C4-A1).



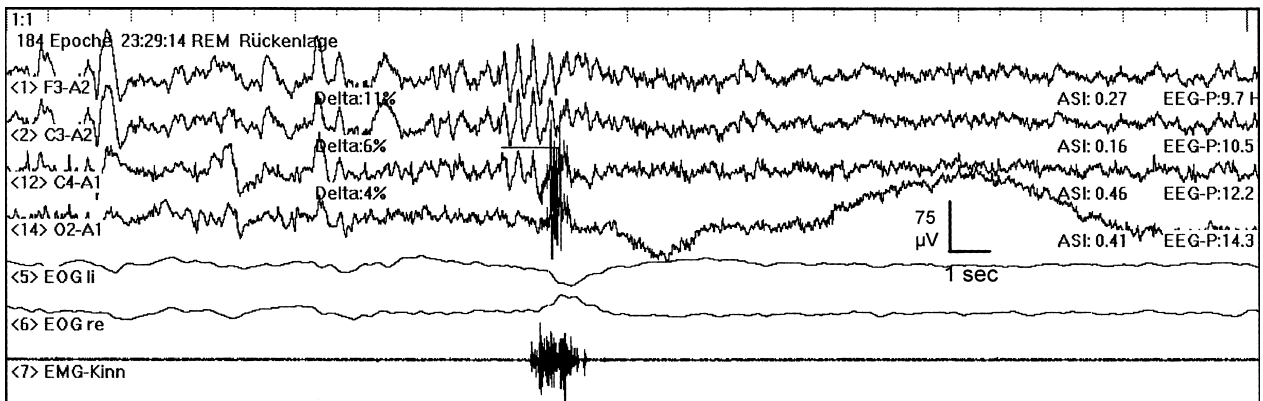
**Figure A4.** One epoch (30 s) with vertex sharp waves (VSW); typical VSWs are marked. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (mental EMG), and two EEGs (C3-A2, C4-A1).



**Figure A5.** One epoch (30 s) with a typical K-complex. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (mental EMG), and two EEGs (C3-A2, C4-A1).



**Figure A6.** One epoch (30 s) with sleep spindles; typical sleep spindles are underlined. From top to bottom: left and right electro-oculogram (EOG), electromyography of chin (mental EMG), and two EEGs (C3-A2, C4-A1).



**Figure A7.** One epoch (30 s) showing sawtooth waves (underlined). From top to bottom: four EEGs (F3-A2, C3-A2, C4-A1, O2-A1), left and right electro-oculogram (EOG), and electromyography of chin (mental EMG).