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Sleep Slow Wave-Bistability and the Connection Between the Sleeping Brain and the Environment—Neurobiological Considerations

Péter Halász[1] | Anna Szűcs[2] | Carlotta Mutti[3,4] | Liborio Parrino[3,4]

[1]Szentágothai Doctoral School, Semmelweis University, Budapest, Hungary | [2]Institute of Behavioural Sciences, Semmelweis University, Budapest, Hungary | [3]Unit of Neurology, Department of Medicine and Surgery, University of Parma, Parma, Italy | [4]Sleep Disorders Centre, Department of Medicine and Surgery, University of Parma, Parma, Italy

Correspondence: Anna Szűcs (szucsan@gmail.com)

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ABSTRACT

The aim of this paper is to review the phenomenon of bistability, a basic working mode of NREM sleep. The perpetual oscillations between two opposite states in sleep slow waves, K-complexes as well as in the Cyclic Alternating Pattern (CAP) on a different time scale; appear spontaneously and can also be elicited by sensory stimulation, representing a response of the sleeping brain. Although there is extensive literature on bistability, its significance remains obscure. In this paper, spontaneous and elicited bistable patterns are reviewed and the global presence of bistable oscillations in NREM sleep is highlighted. We discuss the relationship of bistability with sleep homeostasis and the overlap with the CAP, with emphasis on A1 phase. We collect data along the hypothesis that bistability would provide a perpetual and flexible interface between the sleeping brain and the environment across the night, protecting the continuity of sleep and, in parallel, preserving the brain's adaptive connection with the environment.

1 | Bistable Patterns in NREM Sleep: From K-Complexes to Slow Waves

Bistability is a sleep-related neuronal phenomenon appearing in various forms: as K-complexes (KCs) (Campbell et al. 1980; Halász 2005; Cash et al. 2009; Andrillon et al. 2016); or in the form of continual alternation of two components (up- and down-state) in the slow-oscillation (SO) of N3 stage of NREM sleep. KCs have strong autonomic correlates (Johnson and Karpan 1968).

The most detailed studies about bistability can be grouped around KC, carrying the characteristic up- and down-states and the elicitability by sensory stimuli. The pyramidal cell-membrane's down state is related to K^+-dependent hyperpolarisation associated with neuronal silence, which is immediately followed by an up-state (P 900) with membrane depolarisation associated with intense neuronal firing. (Laurino et al. 2014, 2019) (Figure 1).

KCs reflect the bistable slow (<1 Hz) oscillations at the neocortical level, since they survive in cerveau isolé preparations and persist after massive thalamic lesions. (Amzica and Steriade 1997) There is a complex bidirectional traffic between the cortex and thalamus in generating KCs. In fact, although classically considered purely cortical phenomena, the key role of the thalamus in modulating the appearance and morphology of these waves has been evidenced. (Steriade et al. 1993; Compte et al. 2003; Contreras and Steriade 1995). Cortical areas that do not dispose of the complexity of the multi-layered neocortex

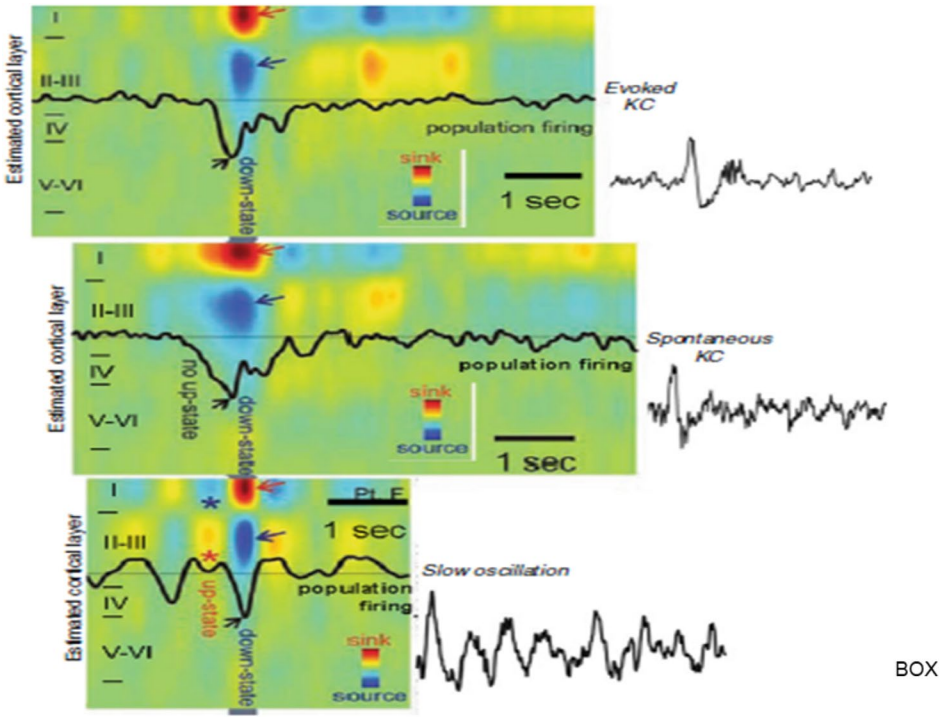


FIGURE 1 | Evoked and spontaneous K-complexes of stage 2 sleep up-down state sequences. Co-registered multiunit activity and current source density with an intracortical multi-microelectrode in human subjects. Red arrow indicates inward currents (sink) in superficial cortical layer and blue arrows show outward currents (source) in layers II–III. Black arrow indicates decreased neuronal firing (Modified from Cash et al. 2009). Note the similarity between spontaneous and evoked K-complexes and slow waves.

and lack a direct thalamic connection (e.g., the pyriform cortex) cannot generate KCs even if they might alternate in up- and down-states (Neske 2016).

The mean occurrence of KCs is 1–2/min. During deep slow wave sleep, singular KCs can be shown by averaging (Sanchez-Vives et al. 2008).

They appear spontaneously or can be elicited during NREM sleep. In the latter scenario, the human brain's response to thalamic inputs results in three types of waves constituting the KC: the positive P200 (not always seen in routine EEG records), the giant negative N550, and the P900 (respectively creating a sequence of positive–negative–positive components on the scalp EEG), while N550 and P900 belong only to NREM sleep. Low-voltage P200 appears in waking as well, increasing from wakefulness to deeper sleep. The brain's proneness to bistability can change, depending on how synchronously K^+ channels open during slow waves' transition to down-state (hence the high variability of the K-complexes). KC can be characterised by the parameters of its slopes and its components (Campbell et al. 1980; Halász 2005; Laurino et al. 2014) (Figure 2). The bistable alternation of KCs is easily reproducible by sensory stimulation (acoustic, tactile, visual or respiratory). Spontaneous and evoked KCs stem from the same cellular dynamics (Laurino et al. 2019).

Although KCs might seem locally generated patterns, they represent, or are part of a complex large-scale network, largely propagating (travelling) within the brain. This travelling wave is key to the bistable cortical response, which needs to quench sensory processing (preserving sleep continuity with the down-state)

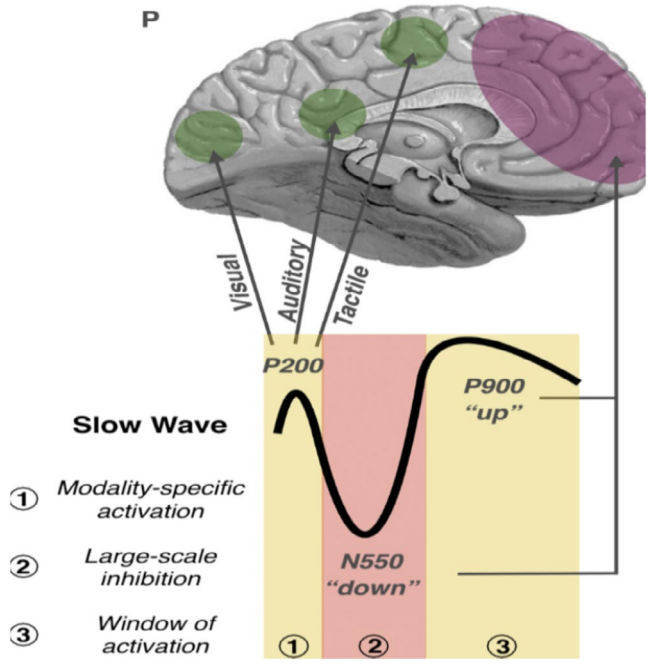


FIGURE 2 | Localisation, polarity and activation/inhibition relations of K-complexes. P220: Modality specific activation (seen in waking and sleep); N550/P900 down- and up-states (window for hyper- and depolarisation during sleep; frontal convexity) (After Andrillon and Kouider 2020).

and at the same time consolidate newly acquired information triggering the thalamo-cortical spindle activity during the up-state (Halász et al. 2004).

Although data collected from microelectrode array channels show that the excitatory output of slow waves (SWs) originates from the supra-granular layers I-III of pyramidal cells (Massimini et al. 2004), the triggering mechanisms of the slow oscillation is far from clear.

Alike to KC, the SW are bistable, too, with an increasing occurrence rate with sleep deepening. They originate from the anterior cortices and travel posteriorly (Csercsa et al. 2010).

The stimulation of the sleeping brain can elicit different responses such as N300 associating with vertex waves in early sleep-stages and N550 associating with KCs in later stages (Murphy et al. 2009).

2 | Bistability and the Influence of External Stimulation

The parameters of evoked KCs change with the type of sensory stimuli. The latency of P200 is low in primary sensory cortices and it follows a wake-like pattern, while it increases significantly in other cortical regions. It changes with sleep-deepening as well: shorter during N3 compared to N2 sleep. Its direction of travelling varies with the types of stimuli, too (Colrain et al. 2000).

Also, the relevance of information influences the sleeping brain's responses. For instance, cortical response to unfamiliar voices differs from that to familiar ones; the former associating with more KCs and microarousals, larger evoked potentials, more precise time-locking in the delta (1–4 Hz) band and stronger activity in the high frequency (> 16 Hz) range. In other words, even the low-level sensory processing of sleep can discriminate stimuli by relevance (Velluti 1997; Ameen et al. 2022; Oswald et al. 1960).

While the bistable alternation is highly reproducible by sensory stimulation in KC, this reactive nature is dubious in the case of the slow oscillation (SO); albeit rhythmic acoustic stimulation can entrain it. The possibility of boosting SO has been shown in several studies. It was first shown by Marshall et al. (Blume et al. 2018) inducing SW-like field potentials by transcranial application of polarising current through bilateral fronto-lateral anode and mastoid cathode–electrodes during early nocturnal NREM sleep (SW frequency: 0.75 Hz). The evoked SW induced slow wave sleep and entrained slow spindling over the frontal cortex. Similarly, transcranial magnetic stimulation has reliably triggered SO resembling spontaneous SO, leading to sleep deepening on one hand and facilitating mnemonic retention of hippocampus-dependent declarative memories on the other (Massimini et al. 2007). Spindling entrained by the evoked SO resembled the spindling occurring on the up states of spontaneous SO, and it also travelled along the cortex as the spontaneous form did (Marshall et al. 2006).

The elicited SO was state-dependent; appearing exclusively during NREM sleep and not in wakefulness. Stimulation in N2 sleep led quickly to a transition to deeper sleep stages (Massimini et al. 2007).

Also, other sensory stimuli can elicit SWs (Ngo et al. 2013). For instance, non-lemniscal acoustic stimulation enhanced them, especially when administered at their up states (Pereira et al. 2017). In fact, while auditory stimuli were ineffective in the modulation of SW when administered out of phase, tones delivered with the up states could evoke a resonating response in the network, associating with sleep-dependent memory consolidation (Bellesi et al. 2014). These findings are congruent with a previous study on rats, showing a refractory period of about 2 s after the neurons' down state (Bellesi et al. 2014) The possibility of inducing a SW is strongly influenced by the proximity of the preceding spontaneous one. Immediately after that, none could be elicited. (Vyazovskiy et al. 2009) studied the effect of intracortical stimulation with brief (0.1 msec) electric pulses during sleep in rat; the elicited potentials were indistinguishable from spontaneous ones, and they were followed by spindling, alike in natural circumstances.

Overall, many data suggest that the effect of incoming stimuli is strongly influenced by the phase of cortical SO. The ability of acoustic stimuli to trigger a SW relies both on the sufficient activation of the ascending sensory pathways and the phase of the thalamocortical oscillation (27, 31). The parameters of evoked SWs change also as a function of homeostatic sleep pressure. When it is high, sleep-like responses are frequent and the slopes of evoked SWs are steeper.

During NREM sleep, in both animals and humans, KC and SW can be elicited both by physiological and artificial stimuli (TMS, polarising current, or direct cortical stimulation). The stereotypical and modality-dependent KC response could be considered an elementary building stone of sleep bistability.

NREM sleep, especially SO, is profoundly influenced by sensory input. Challenging the traditional view, sensory stimulation can deepen sleep both locally and generally; it does not necessarily elicit an arousal; on the contrary, it may evoke sleep-like (hyperpolarizing) responses (Compte et al. 2003).

Several studies have demonstrated that'the fate of incoming stimuli during NREM sleep is determined by spindles and the phase of the slow oscillation. Sensory transmission is blocked at the thalamic level during sleep spindles; likely due to the recurrent inhibition of thalamocortical neurons by reticular thalamic cells. On the other hand, across NREM sleep, the brain appears receptive to the environment at the up states of SO. The specific up-state dependency of incoming stimuli in influencing spindles and SW is restricted to the association cortex suggesting the role of bistability in the higher processing of information (Vyazovskiy et al. 2009).

3 | Bistability and the Homeostatic Process

The homeostatic regulation of KCs had been early confirmed (Schabus et al. 2012; Wauquier 1993; Nicholas et al. 2002; Peszka and Harsh 2002; Crowley et al. 2002; Zadra et al. 2008; Parapatics et al. 2015; Halász and Bódizs 2013), but it had been evidenced only for the first cycles of slow wave sleep. Later, it has turned out that it is difficult to expect a uniform behaviour of KCs, because their activity is under double regulation such as the upcoming outer

stimuli reaching the brain; and the propensity of SW-production closely related to homeostasis. Rather than following a consistent homeostatic down-regulation, the amount of KC varies with the descending and ascending slopes as well as the smaller up- and down-oscillations of SW within cycles and stages of slow wave sleep. At the same time, a close interrelation between CAP A1 phase and homeostatic decay across the night has been shown (Colrain et al. 2010). Frequent KCs reflect a high homeostatic pressure at the beginning of NREM sleep, and their frequency augments again in sleep's last part; when the homeostatic pressure is low but environmental inputs multiply.

4 | The Morphology of Bistable Elements Depends on Their Topology and the Timing in Sleep

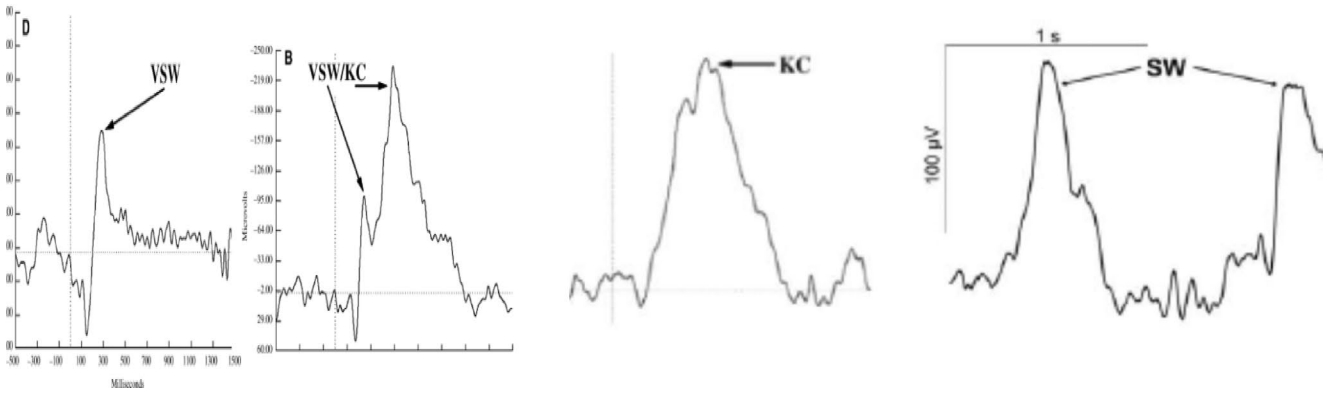
Research has confirmed in several ways the 'local' nature of sleep. Instead of involving the brain homogeneously, sleep occurs and is regulated locally; different vigilance levels co-exist. Besides prior use as a confirmed regulator of local sleep, the neuronal proneness to bistability might also impact on sleep-deepening and reactivity to external stimuli.

Alike to SW, the sensory input propagating toward the frontal lobe via the cingulate highway might first ignite the fronto-medial cortex and evoke a vertex wave. KC appears later on the frontal convexity when the augmentation of homeostatic pressure increases the proneness to bistability.

In this perspective, vertex waves seem to be'fore-runners' of KCs in time and localization; and KCs are the fore-runners of slow waves (Terzano Terzano et al. 2000) (Table 1).

Murphy et al. (Murphy et al. 2009) characterised the source, site, propagation, and role of SWs. They have found that SW preferentially originate from areas adjacent to the lateral sulci (insula, superior temporal gyri) propagating via the cingulate cortices, involving the anterior and posterior cingulate gyrus, the middle, medial and anterior frontal gyri and, the precuneus (Bernardi et al. 2018). These areas overlap with the major connectional backbone of the cortex and with many hubs of the resting-state network (DMN); that is associated with daydreaming. This topographical correspondence of the DMN and SO suggests that the latter might participate in self-organised sleep functions. It is known that the connectivity profile of the DMN changes

TABLE 1 | From left to right: Vertex wave (vsw); K-complex (KC); sleep slow waves (SW).



Reactive graphoelements	Vertex wave (vs)	K-complex (kc)	Slow waves + cyclic alternating pattern (CAP) A1
Reactivity	Spontaneous/ acoustic evoked	Spontaneous/evoked	Spontaneous/evoked
Electrophysiology	Enlarged evoked potential	Homeostasis-dependent reactive cortical down state; connection with arousal	Homeostasis-dependent travelling wave of frontal origin; up/down state. Connection with plastic functions
Typical occurrence on the slopes of sleep-cycles; in the first part of night sleep	Descending and ascending	Descending dominant	Descending
Functional MRI bold activation	In sensory cortical areas	On frontal convexity	Frontal (on slow wave-convexity); CAP A1 (fonto-medial)

Note: The continuum of spontaneous and elicitable EEG patterns during the first cycles of NREM sleep: Vertex wave is the fore-runner of K-complex, K -complex is the fore-runner of slow waves. The table briefly summarises the reactive sleep EEG elements and their features.

with sleepiness and sleep-deepening; with decreasing contribution of the posterior cingulate cortex/retrosplenial cortex, parahippocampal gyrus and medial prefrontal cortex (De Gennaro et al. 2000).

The homeostatic increase of sleep SW wave is more prominent in the left hemisphere and the frontal lobes; likely related to specific inputs (e.g., a preferential involvement of left-side areas due to the demand of the speech network) or to a greater sensitivity (restitution-need) of certain cortical areas (e.g., those areas responsible for human cognitive neo-functions). In any case, one can notice the prominent role of the frontal lobes, which are under the double regulation of both short- and long-term homeostatic processes (Sämann et al. 2011) as well as by use-dependent plasticity processes (Halász et al. 2014).

The dynamics of bistable sleep elements change also with ageing. In particular, SW originate centro-parietally in children and move more frontally in adolescence. Additional elements of sleep macro- and microstructure change the dynamics of brain maturation (Tononi and Cirelli 2014).

5 | Cyclic Alternating Pattern and Bistability

Another aspect alighting bistability during NREM sleep originates from studies of the CAP, recognised in the mid-eighties by the Parma sleep research team (Mutti et al. 2022). CAP is a mini-cyclicity incorporating different phasic elements; mainly micro-arousals of the so-called micro-structure of NREM sleep (Terzano et al. 1985, 2002). The Parma sleep research team described an alternating sequence of activated (phase A) and background (phase B) within a 2–60 (mean 20–40 s) time-span. The absence of CAP for more than 60 s is defined as non-CAP. Within phase A, A1, A2, and A3 subtypes were differentiated. A1 contained slow waves and K-complexes boosting slow-wave sleep; an anti-arousal, sleep-promoting function. A3 overlapped with traditional AASM arousals that were more intrusive to sleep continuity and prepared the sleeping brain for sleep-lightening or REM sleep. A2 was a mixture of A1 and A3. These subtypes related to the level of homeostatic pressure: A1 linked to high, while A2 and A3 linked to lower homeostatic pressure, as reflected by their night distribution. There were more A1 types in the first half of the night and the descending slopes of sleep cycles; A2 and A3 dominated the last part of the night and the ascending slopes when sleep pressure was low (Parapatics et al. 2015). (Figure 3) Also CAP has a double nature, occurring both spontaneously and in response to stimuli. A stimulus during stable non-CAP sleep can induce an A1 type response. Notably, the reaction of the sleeping brain to external stimuli varies according to sleep stages; slow-wave sleep is the most perturbed by acoustic stimuli and presents the steepest increase in the CAP rate compared to lighter sleep stages, which are more resilient.

The features of CAP A1, KCs and SWs reflect an important difference between arousal responses in sleep, versus low homeostatic periods and waking; each proportional to the homeostatic pressure. The traditional arousals in wakefulness and CAP A3 present with fast EEG frequencies—desynchronization; while microarousals in high homeostatic periods of sleep are characterised by hyperpolarisation with delta-like frequencies and carry an antiarousal, sleep-protecting function.

On a different (longer) time scale, CAP seems to repeat the sequence of bistability seen in KCs and SWs. A1 promotes sleep, while A3 is consistent with the classic awake-type arousal response representing swings toward awakening or REM sleep.

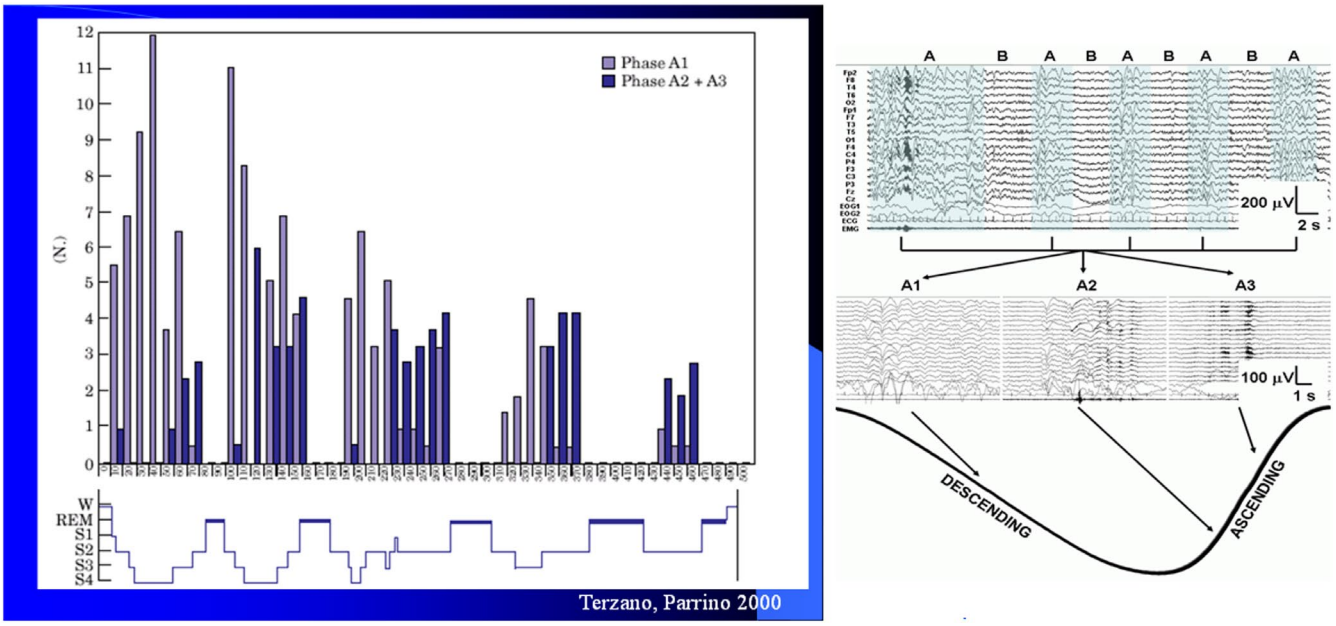


FIGURE 3 | Left: Bottom: Hypnogram, Top: Distribution of A1 (grey) and A2-3 (dark) phases of cyclic alternating pattern (CAP). Right: Top: Alternating activation (A) and background (B) patterns of the NREM sleep EEG; bottom: Typical electromorphology of A1, A2, and A3 sub-phases. Arrows show the typical EEG patterns along the descending and ascending slopes of the first sleep cycle. Note the characteristic distribution of A1 phase through the night with exponential decay paralleling the decay of homeostatic pressure from evening to morning.

A2 is a mixture of A1 and A3. The tyme-span of those CAP responses varies within some seconds compared to the milliseconds' order of SWs' up and down states and the seconds' order of KCs' slow waves (2–4 Hz with fronto-medial localisation) (Halász 1998). CAP provides a frame overarching microarousals.

With the recognition of CAP A1, we have learned that the flow of SWs is not continuous; rather, they appear in input-related staccato bouts. Depending on the degree of sleep-perturbation, 30%–40% of sleep time is spent in CAP A1, characterised by recurrent bouts of reactive slow activity (0.25–2.5 Hz). SW-power augments significantly in N3, leading to an increase of the rate of CAP A1. Early experiments (Terzano et al. 1990) have shown that the spectral power of A1 and A2 phases increased proportionally under increasing sound pressure levels when 45–75 dB white noise had been applied, while the spectral power of phase A3 remained unchanged. Thus, the increase of reactive SW-rate seems to be proportional to the degree of sleep perturbation by external stimulus.

CAP is an instant homeostatic device; not only reflecting sleep instability as the balance between sleep and wake promoting systems, but representing a protective, short-term homeostatic mechanism of slow wave sleep as well (Ferri et al. 2005).

Near bifurcations, complex systems start to oscillate in order to prepare a successful transition from the current attractor to the new set point. During sleep, CAP always heralds the onset of stage REM and accompanies the descending slope of the sleep cycle from light to deep NREM sleep. In normal conditions, it takes approximately 20–25 min to reach the stable attractor of stage N3 non-CAP. According to recent studies, non-CAP reflects a subcritical state and CAP a critical state (Terzano et al. 1990). In practice, whether we deliver one or more stimuli during non-CAP, the output will always be, within given thresholds, a soft, limited response; the system will remain in the previous state. Accordingly, during NREM sleep, criticality is strongly correlated to stage N3 and to CAP phase A1. The most interesting subtype of CAP is the A1. It exerts an anti-arousal reaction and it is made up of SWs and KCs. CAP A1 implements the paradigm of bistability as a sleep-like down-state, an anti-arousal device, ensuring sleep continuity during NREM sleep (Scarpetta et al. 2023).

We can see that sleep SO seems to be under instant short-term and long-term homeostatic regulation. In the case of the instant one, SW response comes immediately after the incoming stimulus, while long-term regulation clamps the input occurring during wakefulness, with a postponed response developing in the next sleep period (Sämann et al. 2011).

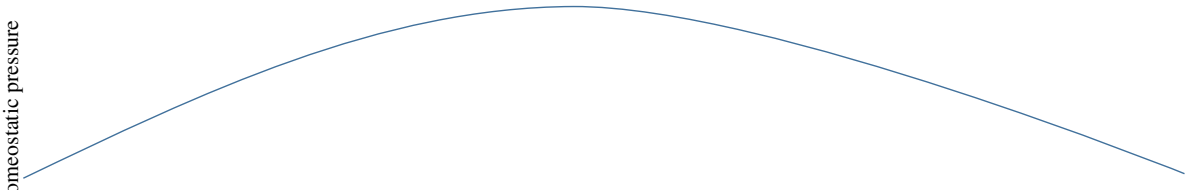
6 | Bistability May Provide an Interface Between the Brain and the Environment Throughout the Night

Micro-shifts of vigilance are fuelled by phasic microarousals within every stage of sleep (Parapatics et al. 2015). The fine-tuned regulation of microarousals originates from the environment and from the brainstem (e.g., the adrenergic afferentation from the locus coeruleus Andrillon et al. 2016). NREM sleep contains reactive bistable elements.

The sleep-process of the night can be divided to three parts (Table 2) such as falling asleep; deep sleep dominated by a continuity of SW with up- and down states; and a preparatory phase to awakening in the end of night.

TABLE 2 | It is a schematic view about slow wave bistability characteristics (mode, anatomy, function) during the three parts of night sleep (falling asleep, deep sleep, preparation to awakening/REM sleep). The relationship of bistability with sleep homeostasisis shown (the curves of the line represent high or low homeostatic pressure).

BISTABILITY IN THE THREE PARTS OF NREM SLEEP ACROSS THE NIGHT

	Falling asleep	Deep slow wave sleep	Preparation for arousal and REM sleep
Homeostatic pressure			
Mode of bistability	recurrent singular evoked and spontaneous	continouos oscillating spontaneous	recurrent singular evoked and spontaneous
Key anatomy	fronto-medial association cortex and anterior cingulate gyrus	cortex involving the talamus	fronto-medial association cortex and anterior cingulate gyrus
Function	promoting sleep	maintaining sleep	promoting awakening and REM sleep

Night-sleep is organised in 4–6 cycles. We have very little knowledge about the roles and functions of this cyclicity and of the descending and ascending slopes manifesting in each cycle. Some continuous changes shown by EEG measures overarch the stages and cycles of sleep (Ioannides et al. 2019).

KCs occur from the onset to the end of sleep both on the descending and ascending slopes of cycles; having different parameters. They reflect a NREM sleep-promoting role at the beginning of the night, and an arousal- and REM-promoting effect later. Sleep onset periods are especially rich in KCs, even appearing in abundance in young persons.

The first two–three cycles of NREM sleep-EEG are dominated by KCs and bouts of SW inter-mixed with sleep spindles (CAP A1). Sleep deepening is marked by the amount of SW and the steepness of the descending slopes within cycles.

During the process of falling asleep, the connection between the sleeper and environment is very rich; both vertex waves and KCs are reactive; pointing to intensive information processing (Andrillon et al. 2016).

KCs are coupled with autonomic activation (Johnson and Karpan 1968) heart rate acceleration, electro-dermographic swings, as well as the activation of the anterior cingulate cortex (Osorio-Forero et al. 2021) one of the hubs of the salience network. This is congruent with the idea of KCs' sentinel function, related to its bottom-up, arousal-dependent mechanism (Ioannides et al. 2019; Osorio-Forero et al. 2021; Jahnke et al. 2012). According to the "predictive brain" theory (Destexhe et al. 2007), the environmental information reaching the brain constitutes a complex picture of the world, and the incoming inputs are compared with the expectations of the individual. Based on this theory, KC may have a predictive sampling function along with continuous monitoring of the environment; protecting the sleep process.

The behaviour of KC varies; its spontaneous occurrence rate as well as its elicitability is high on the ascending slopes possibly due to the brain's higher input sensitivity in those periods. The last third of sleep gives way to a shift toward arousal and REM sleep preparation "cholinergic phase" (Timofeev and Chauvette 2017); SW power decreases, the cycles become shallower, and the arousal-fuelled traffic between sleeper and environment increases again.

Deep sleep is dominated by ~1 Hz SO (Amzica and Steriade 1997). This is the most enigmatic part of slow wave sleep, with many question-marks regarding mechanism, function, distribution and timing within NREM-3 sleep.

The electrophysiological characteristics of down and up states—the bistability phenomenon—is far from clear. There is an obvious similarity in the alternation of down- and up-states of SO versus single KCs. Despite intensive research, the igniting factors of SO remain unclear. It is a cortical rhythm with secondary thalamic involvement. (Contreras and Steriade 1995) The up-states (depolarisations) represent a near wake-level cortical activity with associated activity oscillations of frequencies above 20 Hz (Massimini et al. 2024), opening a window for incoming input ensuring the exchange with the environment (Clark 2024).

There are some hints that down states give place for certain self-organised plastic processes. During periods of high homeostatic pressure, short periods of depolarised phases alternate with long periods of hyperpolarised phases. Conversely, under low homeostatic pressure, long depolarised phases are interrupted by short hyperpolarised ones; showing the close relation of the up-and-down state dynamics with homeostatic regulation (Destexhe et al. 2007).

7 | Conclusions

In this review we broaden the scope of bistable phenomena during slow wave sleep, outlining common features and analysing the dynamics of vigilance-level fluctuations related to micro-shifts within sleep. Bistability appears in NREM sleep, when (within the frame of circadian rhythmicity) the propensity to SW production increases due to the augmentation of homeostatic drive.

Three patterns show the features of bistability; such as the KC, the slow oscillation of NREM sleep, and—on a longer time scale—the CAP. An antiarousal mechanism is inbuilt in NREM sleep. When an inner or outer sleep-disturbing stimulus arrives, a hyperpolarised delta-response appears. It quenches the arousing effect of the stimulus, maintaining sleep continuity and assessing (unconsciously) the importance of the input at the same time.

Bistability reflects a continuous oscillation between sleep and arousal-promoting micro-oscillations regulated by homeostatic and environmental forces. Bistability seems to mediate (providing an interface) between the sleeper and theenvironment.

Recently an interesting new work of Massimini et al. (Timofeev and Chauvette 2017) evidenced a compensating process with bistable elements after stroke-related brain injuries; appearing in NREM sleep, and penetrating into wakefulness and REM sleep as well. Similar findings were reported by the Timofeev group (Massimini et al. 2024; Avramescu and Timofeev 2008) in the development of posttraumatic epilepsy. These studies confirm the important role of SW bistability in homeostatic healing processes.

Author Contributions

Péter Halász: conceptualization, writing – original draft, supervision. **Anna Szűcs:** writing – original draft, writing – review and editing, conceptualization. **Carlotta Mutti:** writing – review and editing. **Liborio Parrino:** writing – review and editing, supervision.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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