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ACTIVE NEOCORTICAL PROCESSES DURING QUIESCENT SLEEP

M. STERIADE

Laboratoire de Neurophysiologie, Faculté de Médecine, Université Laval, Québec, Canada G1K 7P4

PASSIVE SLEEP

Kleitman was a cardinal figure among those who championed the concept of passive sleep which can be traced back to Lucretius' *De rerum natura* and, closer to us, around 1840, to Purkinje. Kleitman (25) thought that sleep is a negation of wakefulness, and what needs to be explained is not sleep but the active state of waking. During the same decade, Bremer (7) made experiments with brainstem transection and postulated that sleep results from the withdrawal of activating impulses. A similar idea is found in the study by Moruzzi and Magoun (32) which led to the conclusion that the presence of a steady background of activity within the brainstem core is an important factor contributing to the maintenance of the waking state, and absence of such activity in it may predispose to sleep. The conception of sleep as a consequence of a fall in the activity of brainstem reticular ascending impulses has the flavor of Bremer's ideas on the *tonus cortical* maintained by afferents acting on brainstem structures, and it is worth mentioning that Moruzzi worked in Bremer's laboratory during the late 1930s. Following some experiments on the lower brainstem in the late 1950s and 1960s, Moruzzi (31) became more inclined towards the theory of active sleep, and the majority of contemporary investigators now think this way. However, the evidence for a compact, homogenous group of inhibitory neurons, inducing sleep through projections to a series of distributed brain structures implicated in the process of falling asleep, is still scarce.

We all hope that the hypnogenic properties of some neuronal aggregates located in the ventrolateral part of the preoptic area will eventually be demonstrated, the more as the inhibitory pathways from that area to the posterior hypothalamus and the presence of sleep-active neurons in the preoptic area and basal forebrain have been reported in some species (20, 37, 63). Even in this case, the hypothesized hypnogenic neurons do not seem necessary and sufficient for sleep induction because, after insomnia produced by excitotoxic lesions of preoptic neurons, sleep is recovered by pharmacological inactivation of the arousing histaminergic tuberoinfundibular neurons (35), as postulated by the concept of passive sleep resulting from removal of activating systems. Thus, this concept is still alive and well. I should say that, along a similar reasoning, no waking "center" is necessary or sufficient because of the presence of multiple such activating systems.

Upon falling asleep, neurons recorded from the cerebral cortex and major activating brainstem modulatory systems display a progressive reduction in their firing

rates, preceding by tens of seconds or a few minutes the overt behavioral signs and physiological correlates of sleep (Fig. 1) (41, 49), suggesting that a cascade of disfacilitatory phenomena occurs during the transition from wakefulness to sleep.

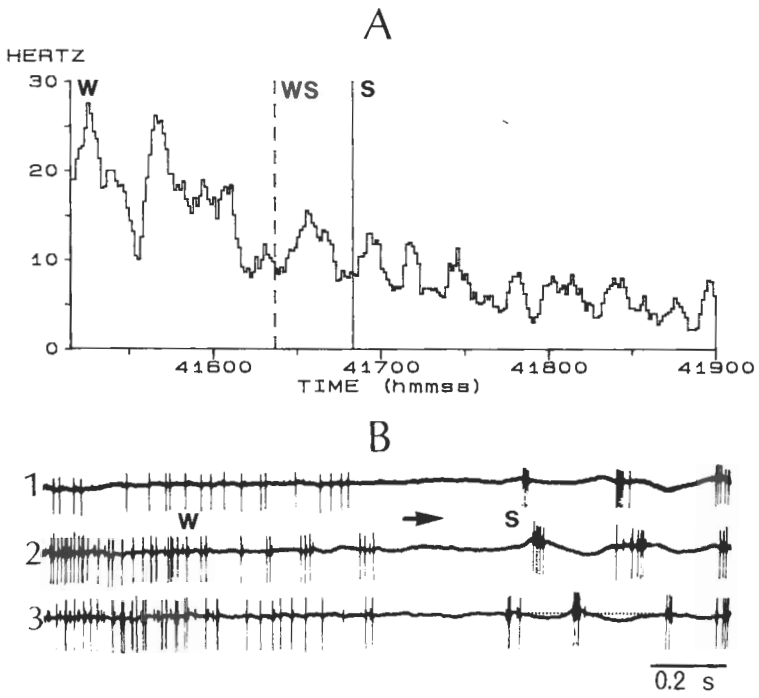

Fig. 1. - *Disfacilitation of brainstem and cortical neurons at sleep onset.*

Extracellular recordings from antidromically identified thalamic-projecting mesopontine cholinergic (*A*) and corticothalamic (*B*) neurons in chronically implanted, naturally sleeping cats. *A*, sequential mean discharge frequency showing progressive reduction in firing rate from waking (W) to the transitional waking-sleep (WS) period, and further to sleep (S). Abscissa indicates real time (hour, minutes, seconds). *B*, cell from cortical area 5 during three transitions from W to S (1 to 3, separated by 70 min.). Note the appearance of focal spindles associated with rhythmic discharges. Modified from Steriade et al. (1990, *A*) and Steriade (1978, *B*).

DISSOCIATION BETWEEN NEOCORTEX AND THALAMUS DURING SLOW-WAVE SLEEP

Does the first stage of sleep, associated with low-frequency (<15 Hz) oscillations of brain electrical activity, call it slow-wave sleep (SWS) or non-REM sleep, deserve the connotation "inactive", "resting" or "quiescent"? The answer is not simple because, at variance with what I thought until quite recently, the thalamus and neocortex do not display parallel activities during SWS. Thalamocortical neurons undergo a global inhibition during SWS, due to the rhythmic and pro-

longed spike-bursts of GABAergic thalamic reticular neurons (48, 53), and their firing rates decrease dramatically when compared to waking (19). By contrast, the mean discharge frequencies of identified long-axoned neurons from neocortical areas are slightly below those found in both brain-active states, waking and REM sleep, and some neuronal types (large-size, fast-conducting pyramidal neurons) discharge more intensely during SWS than during different epochs of wakefulness (18, 41; see Figs. 3 to 6 in Ref. 47). This was not expected in a "quiescent" or "inactive" state. Such terms were probably coined to contrast SWS with REM sleep and also because, following the antique myth that Hypnos and Thanatos are good brothers, three great scientists of the 20th century thought that a global inhibition irradiates over the cortex (34), thus the brain lies for the most part dark during sleep (38), and sleep is associated with an "abject annihilation of consciousness" (17). Therefore, we are facing the dissociation, during SWS, between the very poor activity of thalamocortical neurons, whose stereotyped spike-bursts cannot reflect the variable codes of incoming signals (45), and the relative rich activity of neocortical neurons. This paradox, since the thalamus is the gate of most sensory impulses to neocortex, is only apparent because corticocortical excitatory connections exceed by far thalamocortical ones.

The core of this paper is based on this dissociation between thalamus and neocortex during SWS. I will propose, on the basis of extracellular and intracellular recordings of neurons from multiple sites in the cerebral cortex and thalamus that neocortical neurons may engage a dialogue of internally-generated signals, despite the generalized inhibition of thalamocortical neurons which contributes to the disconnection of the brain from the outside world. The rhythmic trains of action potentials fired by neocortical neurons during SWS oscillations could exert an important role in the reorganization of the circuitry, leading to plasticity processes and, possibly, to the consolidation of memory traces acquired during the waking state. Some data derive from dual simultaneous intracellular recordings of cortical neurons or of cortical and related thalamic neurons under ketamine-xylazine anesthesia, a state whose electrographic similarity with natural SWS in cats (56, 57) and humans (4) was demonstrated (Fig. 2). Other results derive from the first intracellular recordings, during the whole natural waking-sleep cycle in chronically implanted cats, of various types of electrophysiologically characterized neocortical neurons: pyramidal-shaped of the regular-spiking, intrinsically-bursting and fast-rhythmic-bursting types; and local GABAergic of the fast-spiking type (61, 67).

THE CORTEX AND THALAMUS ARE A UNIFIED OSCILLATORY MACHINE DURING SLOW-WAVE SLEEP

Although neocortical and thalamic neurons display differential pictures during SWS, the former being relatively active and the latter inhibited, they elaborate together sleep oscillations (43, 44). The view that each oscillatory type is generated in a simple, cortical *or* thalamic, circuit became obsolete with the discovery

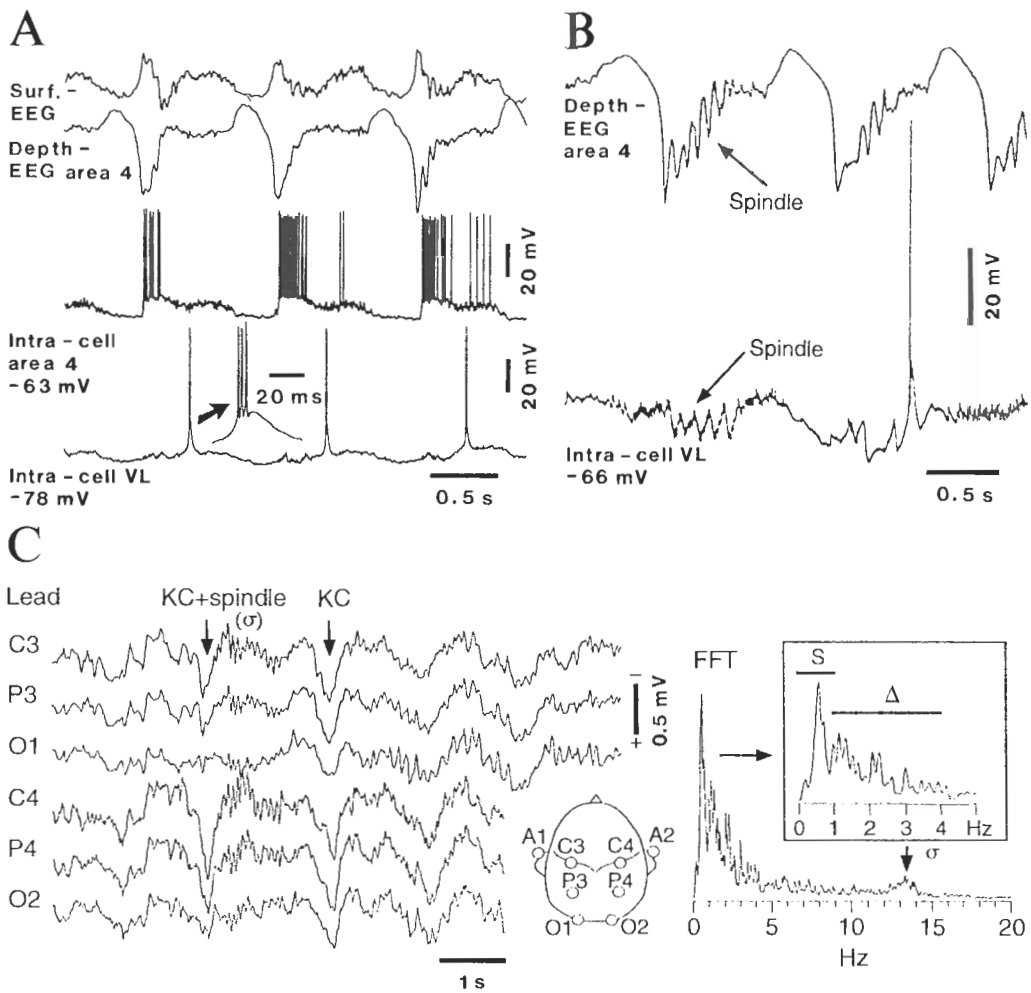

Fig. 2. - *Similar patterns of neocortical slow oscillation in cats under ketamine-xylazine anesthesia and during natural SWS in humans.*

A, the slow oscillation (about 0.9 Hz) in dual simultaneous intracellular recordings from regular-spiking cell in cortical area 4 and thalamocortical cell in the ventrolateral (VL) nucleus, together with depth-EEG from area 4. Cat under ketamine-xylazine anesthesia. Note three cycles of slow oscillation. Arrow points to a low-threshold spike-burst in VL neuron. *B*, intracellular recording of thalamocortical neuron from VL nucleus, together with EEG waves from the depth of cortical area 4 where VL nucleus projects. Cat under ketamine-xylazine anesthesia. The excitatory component (depth-negative, downward deflection) of the slow cortical oscillation (0.9 Hz) is followed by a sequence of spindle waves at ~10 Hz (arrows), generated in the thalamus. This combination gives rise to what are called K-complexes in human sleep (see below, *C*). *C*, the K-complex (KC) in natural human sleep. Scalp monopolar recordings with respect to the contralateral ear are shown (see figurine). Traces show a short episode from a stage 3 SWS. The two arrows point to two K-complexes, consisting of a surface-positive wave, followed (or not) by a sequence of spindle (sigma) waves. Note the synchrony of K-complexes in all recorded sites. At right, frequency decomposition of the electrical activity from C3 lead into three frequency bands: slow oscillation (S, 0 to 1 Hz), delta waves (Δ, 1 to 4 Hz) and spindles (σ, 12 to 15 Hz). Modified from Steriade (1997, *A*), Timofeev and Steriade (1997, *B*) and Amzica and Steriade (1997, *C*).

of a slow oscillation (generally 0.5-1 Hz, mainly 0.7-0.8 Hz) that arises in neocortical networks but has the virtue of grouping other sleep oscillations, including those generated in the thalamus, in complex wave-sequences. This is illustrated in Fig. 2 showing that each cycle of the cortical slow oscillation leads to a short sequence of thalamically generated spindles. Such a combination between the slow oscillation and spindles is termed K-complex in clinical EEG (Fig. 2C) (4).

The slow oscillation was first described intracellularly under anesthesia (54, 55) and was also found, with the same characteristics, using extracellular recordings from naturally sleeping animals (56), as well as scalp EEG (1, 4; see Fig. 2C) and magnetoencephalographic (39) recordings in humans. The cortical origin of the slow oscillation was demonstrated by its survival after extensive thalamectomy (55), disappearance in the thalamus of decorticated animals (64), and disruption of its synchronization process after disconnection of intracortical synaptic linkages (2). The major types of neocortical neurons (pyramidal regular-spiking and local GABAergic fast-spiking), formally identified by intracellular staining and responses to depolarizing current pulses (11), display the slow oscillation with the same characteristics, namely, discharging during the depolarizing phase associated with depth-negative field potentials and being silent during the hyperpolarizing phase associated with depth-positive field potentials. The slow cortical oscillation is mainly initiated by the progressive deafferentation of the forebrain following decreased activities in ascending activating systems and is obliterated by setting into action either the brainstem-thalamic-cortical system or the cortically-projecting basal forebrain system (51).

The synchronization of all types of neocortical neurons during the slow oscillation and, in particular, their synchronous discharges during the depolarizing component of this oscillation explains the impact of neocortical neurons on thalamic reticular and relay neurons, with the consequence of triggering sleep spindles at 7-to-14 Hz (Fig. 2). Although spindles may be generated in the absence of cortex (29) and can also be investigated in thalamic slices (68), the synchronization of this sleep rhythm is different in the absence or the presence of neocortex. Indeed, spindles propagate systematically in visual thalamic slices (24), whereas they are nearly simultaneous in widespread territories of the thalamus and cortex in the intact cortex of both cats and humans (12, 14). The enhanced excitability of neocortical neurons (16), their synchronization among distant areas (3), and the projection of these coherent activities to the thalamus, all contribute to the virtually simultaneous occurrence of spindle waves. After decortication, the thalamic synchronization of spindles is disorganized or completely lost (12) and what remains is accounted for by the synchronizing power of the thalamic reticular nucleus (14).

The appearance of delta waves (1-4 Hz) on the EEG is also controlled by neocortex. This is not only the case of cortically generated delta waves, which survive thalamectomy (55), but also of thalamic clock-like delta oscillation which results from the interplay between two hyperpolarization-activated inward currents of thalamocortical neurons (15, 26, 28). Although intrinsic, the thalamic delta

rhythm is reflected at the cortical EEG level because the cortex drives thalamic reticular neurons that, by virtue of their inhibitory actions and widespread projections to the dorsal thalamus, set the membrane potential of thalamocortical neurons at the hyperpolarized level required for the generation of delta waves and synchronize pools of thalamocortical cells that impose their activity to cortex (50).

In sum, although the separate description of the three different (spindles, delta and slow) sleep oscillations had an heuristic value and may still be used for didactic purposes, the intact brain reveals concerted activities of neocortical and thalamic neurons leading to grouping of oscillations, which are otherwise defined as distinct frequency bands. While corticothalamic neurons similarly exert excitatory actions when thalamic reticular and thalamocortical neurons are considered in isolation, the real effects of synchronous cortical volleys are a powerful excitation of thalamic reticular neurons and, conversely, cyclic IPSPs in thalamocortical neurons because of the actions of GABAergic reticular and local-circuit neurons (44). The cortical slow sleep oscillation provides these synchronous volleys leading to activation of thalamic oscillatory properties. This is all the more important when considering that a newly described class of deeply lying corticothalamic neurons fires high-frequency spike-bursts (43, 59) and thus has a great impact on thalamic neurons. Some of these cortical neurons were antidromically identified from rostral intralaminar thalamic nuclei (59) that project over wide neocortical territories (22, 58) and could, thus, generalize oscillatory activities generated in corticothalamocortical loops through the reentrant pathways. The search for the neuronal bases of the brain rhythms, using intracellular recordings in acute experiments (Fig. 2A-B), and validating these results by means of extracellular and intracellular recordings during natural sleep of chronically implanted animals (see Fig. 3 in the next section) as well as through scalp recordings from humans (Fig. 2C), has provided a new view of sleep oscillations. The complex processes resulting from reciprocal connections between cortical and thalamic neurons, under the control exerted by brainstem and forebrain modulatory systems, can only be investigated in the intact brain. This explains my choice of working *in vivo*.

ACTIVE NEOCORTICAL PROCESSES DURING QUIESCENT SLEEP

The characteristic feature of neocortical neurons during SWS is the presence of prolonged (up to 0.5-0.6 s), high-amplitude (10-20 mV) hyperpolarizations that correspond to depth-positive (surface-negative) field potentials (Fig. 3), as is the also the case during the slow oscillation under ketamine-xylazine anesthesia (11). These long-lasting hyperpolarizations are not mediated by GABAergic events as all recorded fast-spiking, aspiny or sparsely spiny basket-type inhibitory interneurons discharge, like pyramidal neurons, during the depolarizing component of the slow oscillation and are silent during the hyperpolarizing phase (11). Testing the apparent input resistance of cortical neurons under ketamine-xylazine anesthesia through

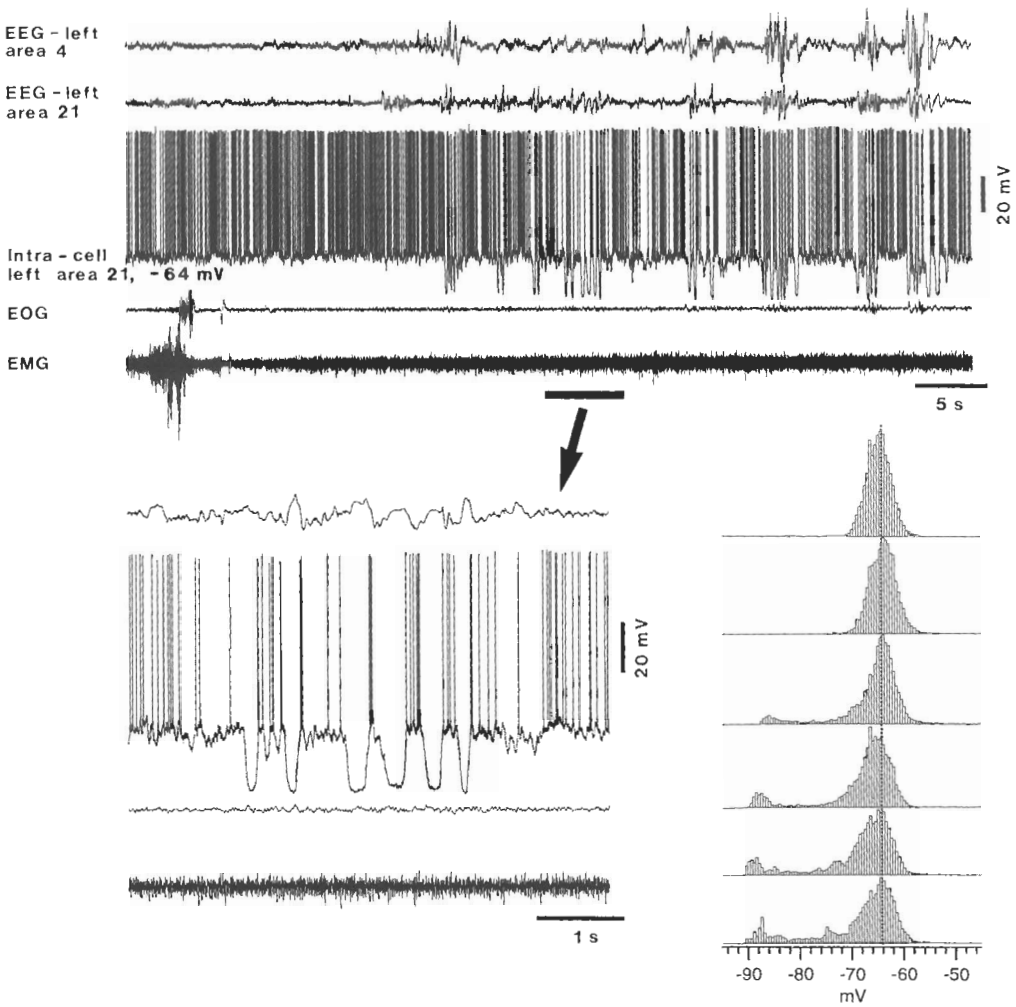

Fig. 3. - *Natural slow-wave sleep is characterized by prolonged hyperpolarizations in neocortical neurons, but rich spontaneous firing during the depolarizing epochs.*

Chronically implanted cat. Five traces in top panel depict EEG from the depth of left cortical areas 4 (motor) and 21 (visual association), intracellular recording from area 21 neuron (resting membrane potential is indicated), electro-oculogram (EOG) and electromyogram (EMG). Part marked by horizontal bar is expanded below left (arrow). Note relation between the hyperpolarizations and depth-positive EEG field potentials. Below right, histograms of membrane potential (10-s epochs) during the period of transition from waking to slow-wave sleep depicted above. Note membrane potential around -64 mV during the 20 s of waking and progressively increased tail of hyperpolarizations, up to -90 mV, during sleep. From Steriade et al. (1999).

their responses to short hyperpolarizing current pulses revealed that the highest values occur during the prolonged hyperpolarization and the lowest during the depolarizing phase (13). We then proposed that disfacilitation is the main mechanism of hyperpolarizations during sleep. Network disfacilitation may occur in

avalanche following a short IPSP leading to silenced firing in target neurons, as recordings with chloride-filled pipettes revealed a decreased steepness and amplitude in the very onset of hyperpolarizations (see Fig. 8 in Ref. 54). However, because of the short duration of $GABA_A$-mediated IPSPs and the very long duration of hyperpolarizations during the slow sleep oscillation, the former can hardly be determined in all instances. Recordings with cesium-filled pipettes produced a significant reduction in the amplitude of hyperpolarizations (54, 67), thus indicating that some potassium currents are responsible for the hyperpolarizations. Most likely, these are calcium-dependent potassium currents as setting in action ascending activating cholinergic systems obliterates the hyperpolarization phase of the slow oscillation (51). It is known that activation of muscarinic receptors diminishes or suppresses those potassium currents (36). Thus, the mechanisms of sleep hyperpolarizations are a mixture of disfacilitation and potassium currents.

Although neocortical neurons are cyclically hyperpolarized during the natural state of SWS, their discharges during the depolarizing phase of the slow sleep oscillation are as high and sometimes higher than during the tonically depolarized states of waking and REM sleep (see Fig. 3). This accounts for the only modest reduction in discharge rates of neocortical neurons during SWS.

Why are neocortical neurons so busy during a behavioral state in which the brain is disconnected from the outside world? We hypothesized that the rich activity of neocortical neurons may reorganize/specify the neuronal circuitry and consolidate memory traces acquired during wakefulness. Thus, the prolonged and rhythmic trains of action potentials fired by neocortical neurons during SWS may lead to plastic processes and increased synaptic excitability in target cortical and thalamic neurons. This hypothesis was advanced on the basis of work in corticothalamic circuits (52) and a similar idea was proposed (8) and tested experimentally (69) in the hippocampus.

A major sleep oscillation, spindles, can be mimicked by stimulating the thalamus or the neocortex with trains of electrical pulses within the frequency range of spindles (~10 Hz). We asked whether the responses evoked by rhythmic stimulation are followed by self-sustained activity associated with increased synaptic excitability in the stimulated network. The responses to pulse-trains at around 10 Hz grow progressively in size, from the second stimulus in the train, and are therefore termed augmenting or incremental. They have been known since the 1940s (30) but the mechanisms have been studied intracellularly only recently (9, 10, 46, 60). We used *in vivo* simultaneous intracellular recordings from different types of neurons in the cortex or in thalamic nuclei and related cortical areas (60). Realistic models of the augmentation phenomenon provided insights into the mechanisms of augmenting responses and made predictions that could be further tested experimentally (5, 6).

Two aspects should be briefly discussed in relation to the possibility that augmenting responses, mimicking spindles, may play a role in short-term plasticity processes during sleep. The first point is whether these responses depend on the thalamus (at which level spindles are generated) or on the neocortex (where

spindles produce active processes that may eventually lead to self-sustained activities). The second point is the occurrence of enhanced excitability and self-sustained oscillatory activities in the neocortex despite the fact that thalamocortical neurons remain under the hyperpolarizing pressure from the thalamic reticular neurons. To anticipate, *although augmentation occurs in the thalamus of decorticated animals* (46) *and in the cortex of athalamic preparations* (55), *the full development of augmenting responses, leading to self-sustained activities, requires interacting thalamic and cortical networks.* This corroborates data showing that, although spindles are generated in the thalamus, their full synchrony and simultaneous appearance over widespread territories requires the cortical feedback (see above).

After hemidecortication, ipsilateral thalamocortical neurons display two types of augmenting responses to local thalamic stimulation at 10 Hz (46). One type is associated with progressively decreased IPSPs elicited by successive stimuli in the train and with progressive depolarization of neurons leading to high-threshold spike-bursts, with increasing number of action potentials and spike inactivation (Fig. 4A). The other type of intrathalamic augmenting responses is based on progressively increased postinhibitory rebounds, low-threshold Ca2+ spikes (LTSs), an intrinsic property of thalamic cells (27). The latter form of augmentation (increasing hyperpolarization with increasing LTSs) is due to the parallel excitation in a pool of thalamic reticular GABAergic neurons, whereas the former type (high-threshold augmenting) is due to the decremental responses in a pool of thalamic reticular neurons (66). Thus, the incremental responses in GABAergic reticular neurons produce a progressive hyperpolarization in related thalamocortical neurons, with the consequence of progressively de-inactivating the LTS and increasing the postinhibitory rebound spike-bursts, whereas the decremental responses of thalamic reticular cells are the cause of augmenting responses in thalamocortical neurons occurring at depolarized levels, probably due to Ca^{2+}-dependent high-voltage currents.

On the other hand, the neocortex is capable of displaying augmentation, with progressive depolarization and increased responsiveness to successive volleys, in animals with ipsilateral thalamectomy (Fig. 4B) (55).

Despite the fact that the thalamus is capable of producing the augmenting responses in decorticated animals and the cortex displays the same type of frequency-dependent increased responses after thalamectomy, we further investigated this phenomenon in intact thalamocortical systems by means of dual intracellular recordings of thalamic and related neocortical neurons *in vivo* (60). This study revealed that the augmentation in neocortical neurons is expressed by a selective increase in the secondary depolarizing component of thalamically-evoked responses. The cortical secondary depolarization invariably follows by about 3 ms the LTSs crowned by rebound spike-bursts in simultaneously recorded thalamocortical neurons. Thus, in intact-brain preparations, the thalamocortical augmenting responses primarily depend upon the LTS-type of augmentation and related spike-bursts in thalamic relay neurons. However, after augmenting responses tha-

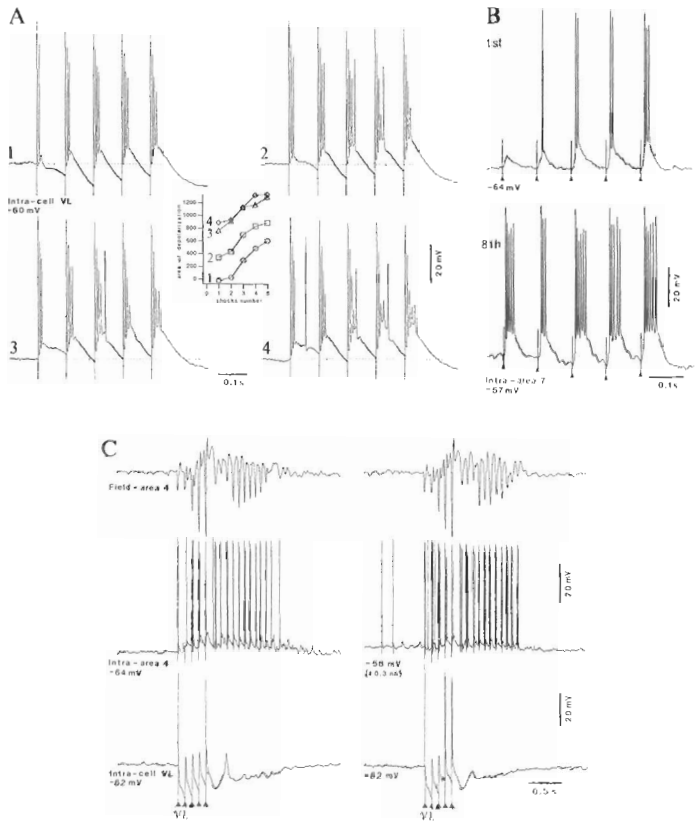

Fig. 4. - *Progressive depolarization, increased synaptic excitability and self-sustained oscillatory activity during and after augmenting responses mimicking sleep spindles.*

Cats under ketamine-xylazine (*A*), urethane (*B*) and barbiturate (*C*) anesthesia. In *A*, intracellular recording from the thalamic ventrolateral (VL) nucleus after ipsilateral hemidecortication and callosal cut. Pulse-trains consisting of five stimuli at 10 Hz were applied to VL nucleus every 2 seconds. The type of augmenting illustrated here is high-threshold, occurring at a depolarized level. In this case, the responses to a 5-shock train consisted of an early antidromic spike, followed by orthodromic spikes displaying progressive augmentation and spike inactivation. Responses to four pulse-trains (*1-4*) are illustrated (*1* and *2* were separated by 2 seconds; *3* and *4* were separated by 2 seconds and followed 14 seconds after *2*). With repetition of pulse-trains, IPSPs elicited by preceding stimuli in the train were progressively reduced until their complete obliteration and spike-bursts contained more action potentials with spike inactivation. The graph depicts the increased area of depolarization from the first to the fifth responses in each pulse-train as well as from pulse-train *1* to pulse-trains *3* and *4*. Panel *B* illustrates changes in properties of a neocortical neuron after repetitive callosal stimulation of the homotopic point in the contralateral hemisphere. The ipsilateral thalamus was excitotoxically destroyed by means of kainic acid. Intracellular recording of neuron at a depth of 1.5 mm in area 7. Responses to pulse-trains (each consisting of 5 stimuli at 10 Hz), repeated every 3 s, applied to contralateral area 7. The intracortical augmenting responses to the 1st and 8th pulse-trains are illustrated. Note depolarization by about 7 mV and increased number of action potentials within bursts after repetitive stimulation. Panel *C* shows self-sustained, post-augmenting oscillation in cortical neuron. Dual intracellular recording from motor area 4 and thalamocortical neuron in the VL nucleus, in conjunction with field potentials from the depth of area 4. Stimulation at 10 Hz (5 stimuli) to the VL nucleus. Two values of membrane potential in cortical neuron (left, -64 mV; right, under +0.3 nA, -58 mV). Note persistent, spindle-like oscillation at the same frequency of augmenting responses in area 4, contrasting with a single low-threshold rebound in the VL cell. Modified from Steriade and Timofeev (1997, *A*), Steriade et al. (1993, *B*), and Steriade et al. (1998, *C*).

lamocortical neurons remain under the hyperpolarizing pressure exerted by thalamic reticular neurons, whereas cortical neurons display self-sustained oscillatory activities, within the same frequency range as that of responses during the stimulation stage (Fig. 4C). Our data show that *intracortical circuits have a major influence on the incoming thalamocortical inputs and can amplify oscillatory activity arising in the thalamus.* This is consistent with the fact that, although spindles are generated in the thalamus, they are not passively reflected in cortex and cortical synaptic circuitry has a major role in modifying and amplifying thalamocortical volleys (23).

The importance of an intact, reciprocal thalamocorticothalamic loop for the development of self-sustained activities results from the effects of prolonged and rhythmic stimulation of neocortex, which lead to responses in bursting thalamic neurons and, at later stages of stimulation, to spontaneous spike-bursts that begin to appear and progressively develop in periods free of stimulation, with the same pattern and frequency as stimulus-locked responses, as in "memory" processes of reciprocal circuits (42, 44). The fact that the self-sustained activity strikingly resembles the responses elicited during the final stage of stimulation was also observed in amygdalo-hippocampal circuits (40). Taken together, these data suggest that such resonant phenomena, both evoked and self-sustained, use the same circuitry.

The repeated circulation of impulses in reverberating circuits, especially when considering the corticothalamic and thalamocortical neurons which are able to discharge rhythmic spike-bursts, could lead to synaptic modifications in target structures, which favor alterations required for memory processes. These data and hypothesis imply that the state of resting sleep is not associated with a complete suspension of processes leading to cognition or consciousness. In fact, contrary to the usual thinking that dreaming only occurs in paradoxical sleep, and that the memory of dreams is erased if a period of slow-wave sleep intervenes after paradoxical sleep, dreams with a peculiar content and dissimilar to those in paradoxical sleep also occur in late stages of slow-wave sleep (21) and the recall rate from quiet sleep dreaming seems to be quite high (33).

SUMMARY

The neocortex and the thalamus constitute a unified oscillatory machine during different states of vigilance. The cortically generated slow sleep oscillation has the virtue of grouping other sleep rhythms, including those arising in the thalamus, within complex wave-sequences. Despite the coherent oscillatory activity in corticothalamic circuits, on the functional side there is dissociation between thalamus and neocortex during sleep. While dorsal thalamic neurons undergo inhibitory processes induced by prolonged spikebursts of GABAergic thalamic reticular neurons, the cortex displays, periodically, a rich spontaneous activity and preserves the capacity to process internally generated signals. Simultaneous intracel-

lular recordings from thalamic and cortical neurons show that short-term plasticity processes occur after prolonged and rhythmic spike-bursts fired by thalamic and cortical neurons during slow-wave sleep oscillations. This may serve to support resonant phenomena and reorganize corticothalamic circuitry.

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