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Inhibitory Processes in the Thalamus

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Three major thalamic inhibitory processes are involved in the genesis of synchronized EEG spindle oscillations during sleep, in spindle disruption upon arousal, and in local synaptic operations within glomeruli that are probably related to discrimination processes during wakefulness. Spindle oscillations are characterized intracellularly by rhythmic (7 to 14 Hz) depolarizations and spike bursts in GABAergic reticular (RE) thalamic neurons, with the consequence of rhythmic hyperpolarization-rebound sequences in thalamocortical neurons. The rebound component triggers postsynaptic events in cortical neurons, within the frequency range of spindles. The absence of spindle rhythms in thalamic nuclei deprived of inputs from the RE nucleus and the preservation of spindle rhythmicity in RE neurons disconnected from the thalamus and cerebral cortex demonstrate that the RE nucleus is the pacemaker of spindle oscillations. The disruption of spindle oscillations upon arousal from sleep is realized by upper brainstem reticular afferents to thalamic and basal forebrain structures. Stimulation of peribrachial (pedunculopontine) nucleus elicits, in addition to an early excitation, a long-lasting hyperpolarization associated with a marked conductance increase in RE neurons. This effectively blocks ongoing spindle sequences in the thalamic pacemaker.

Three types of inhibitory processes operate in the thalamic networks. The first type relates to spindle oscillations during the state of EEG-synchronized sleep. It is generated in GABAergic reticular (RE) thalamic nucleus and acts upon the thalamocortical neurons with the consequence of widespread distribution of spindle rhythms over the neocortex. The second type of inhibition is also a generalized process and it accounts for the blockade of spindling upon arousal from sleep. The origin of this inhibition lies in the brainstem reticular core and its mechanism is a muscarinic effect exerted at the very site of spindle genesis, the RE neurons. The last type of inhibition is produced by GABAergic local-circuit cells which operate on a local basis, within thalamic glomeruli. Whereas the first two inhibitory processes have been investigated in depth (and are, therefore, the matter of this paper), there are very few data bearing on the electrophysiological properties of GABAergic short-axoned cells that represent about 20% to 30% of neurons in various

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thalamic nuclei. As discussed below, the enhanced discrimination in thalamocortical neurons during the waking state may originate, at least partially, in an increased activity of intrinsic interneurons.

The Genesis of Synchronized Spindle Oscillations

Spindle oscillations are high-amplitude, waxing and waning waves at 7–14 Hz, grouped in sequences that last for 1.5–2.5 sec and that recur periodically with a slow rhythm at 0.1–0.3 Hz (see Figure 1). Both these rhythms appear during drowsiness and EEG-synchronized sleep or during barbiturate anesthesia. Spindles originate in the thalamus, as they have been recorded by Morison and Bassett (1945) in the total absence of the cerebral cortex and brainstem. During the 1960's, Andersen and his colleagues proposed that multiple spindle pacemakers are disseminated throughout the thalamus (cf. Andersen and Andersson, 1968). According to this theory, all thalamic nuclei are endowed with the ability of generating spindle rhythmicity through a mechanism of intranuclear inhibition involving recurrent collaterals of thalamocortical axons and local-circuit inhibitory cells. Global synchronization was hypothesized to be realized by the rapid spread of rhythmic activity from one nucleus to another through distributor neurons. However, in view of recent data based on intracellular HRP staining, this theory can no longer be maintained since the intranuclear recurrent collaterals are very few or absent and the internuclear pathways are rather the exception than the rule in the thalamus (cf. Jones, 1985; Steriade and Deschênes, 1984).

We champion the idea of a unique thalamic pacemaker of spindle oscillations, located in the RE nucleus. This proposal is based on the following data. The spindle-related events in thalamocortical neurons consist of long-lasting cyclic hyperpolarizations that can be reversed in sign by hyperpolarizing currents and Cl injection (Deschênes, Paradis, Roy, and Steriade, 1984), hence indicating that they are mainly composed of inhibitory postsynaptic potentials (IPSPs). A pool of inhibitory thalamic neurons with widespread projections to the whole thalamus would be the ideal candidate for inducing rhythmic and synchronous hyperpolarizations in thalamocortical cells. The RE neurons are indeed GABAergic (Houser, Vaughn, Barber, and Roberts, 1980) and project to virtually all thalamic nuclei (Jones, 1975; Steriade, Parent, and Hada, 1984). An implicit assumption of this hypothesis is that those thalamic nuclei which are normally devoid of RE synaptic inputs would not display spindling rhythmicity. Our proposal seems to resist experimental testing.

The RE neurons discharge long-lasting spike bursts which may extend over the whole duration of a spindle sequence during natural sleep (Steriade, Domich, and Oakson, 1986). Intracellularly, the oscillations of RE neurons during barbiturate anesthesia are superimposed on a slowly growing and decaying depolarization (Mulle, Madariaga, and Deschênes, 1986).

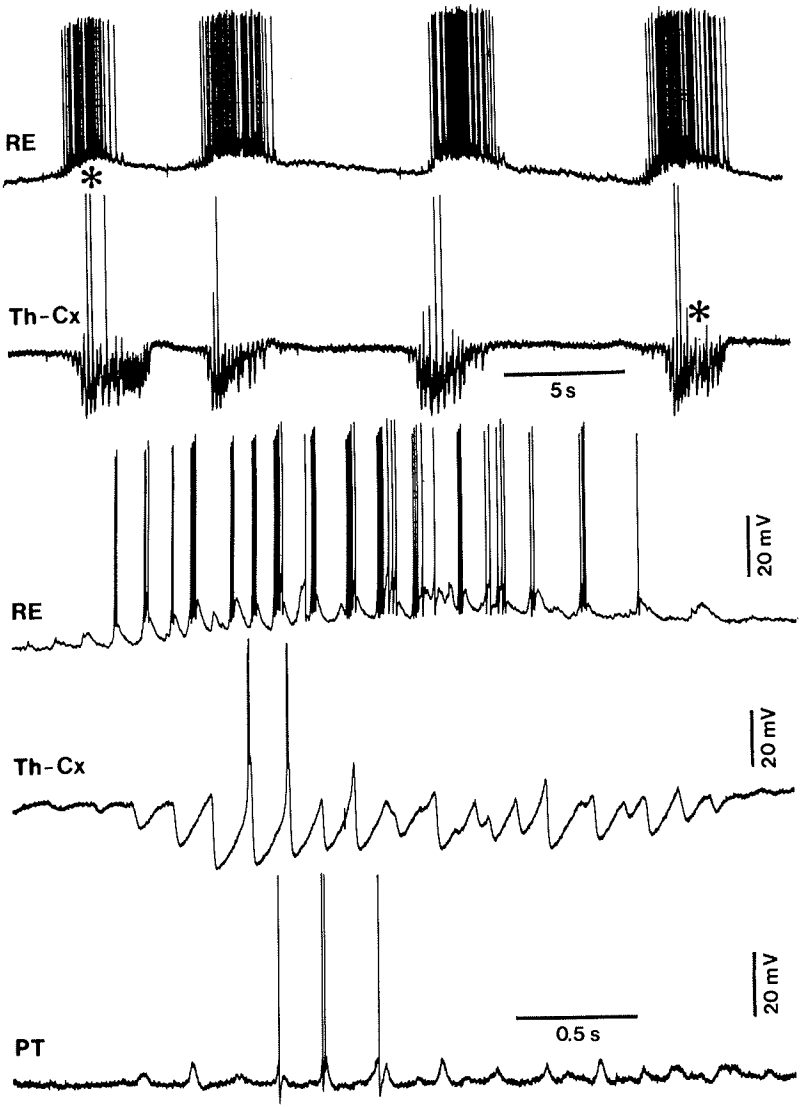

Figure 1: Intracellular aspects of oscillatory activities in reticular (RE) thalamic, thalamocortical (Th-Cx; recorded from ventrolateral nucleus), and pyramidal tract (PT; from precruciate gyrus) neurons of cat under barbiturate anesthesia. In this and following figures, intracellular recordings were made with pipettes filled with K acetate and resting potentials were over -55 mV. The spindle sequences marked by asterisks in top traces of RE and Th-Cx neurons are depicted below at higher speed.

Contrastingly, the spindle oscillations of thalamocortical neurons are characterized by rhythmic (7–14 Hz) and prolonged (0.1–0.15 sec) periods of hyperpolarizations that, occasionally, may de-inactivate a low-threshold rebound spike crowned by high-frequency action potentials (Figure 1). The postinhibitory rebound is transferred along thalamocortical axons and triggers postsynaptic events in neocortical neurons within the frequency of spindle waves. These are the cellular bases of EEG spindling.

Do the reciprocal profiles of RE and thalamocortical neurons during spindling (rhythmic depolarizing and hyperpolarizing oscillations, respectively) reflect a causal relation? The answer is yes, since in the absence of RE neurons, following their chemical lesion by kainic acid, the cortically-evoked *rhythmic* hyperpolarization-rebound sequences (as seen in RE-afferented thalamocortical neurons) are replaced by a long depolarizing potential followed by a *single* phase of hyperpolarization that is unaffected by intracellular Cl injection (see Figure 2). The disconnection of cortically-projecting thalamic nuclei from their RE

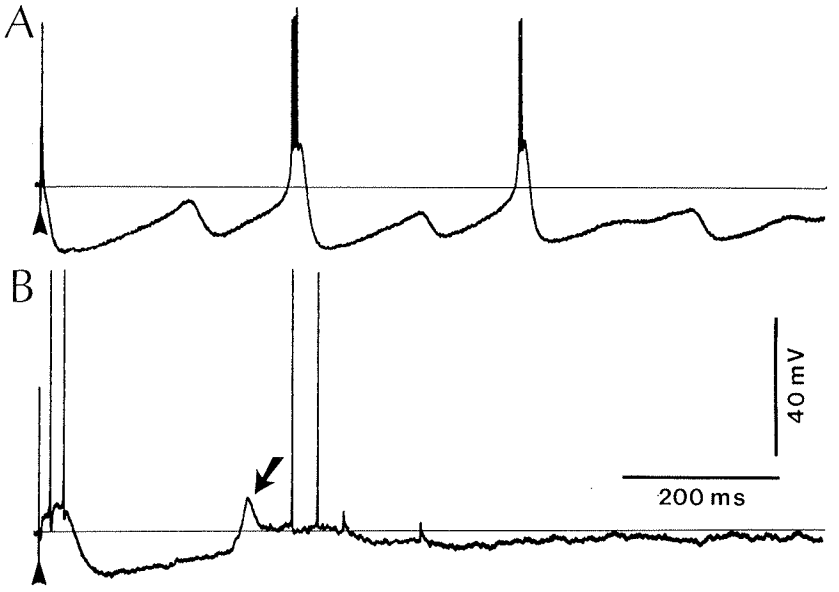

Figure 2: Absence of evoked spindle-like oscillatory response in thalamocortical neurons after kainic acid lesion of the RE nucleus. See the aspect of such lesions in Steriade et al. (1985). *A*: typical response of a ventrolateral thalamocortical neuron to cortical stimulation (arrow head) in an intact preparation. *B*: response of a ventrolateral thalamocortical cell to cortical stimulation after RE lesion; note the absence of oscillations and the presence of a single period of hyperpolarization followed by a low-threshold rebound spike (oblique arrow).

inputs is accompanied not only by the absence of evoked spindles, but also by complete abolition of spontaneous spindling (Steriade, Deschênes, Domich, and Mulle, 1985). The absence of spindle rhythms in RE-deprived thalamic nuclei is associated with an *all-burst* discharge pattern of relay cells, that is, an activity which consists exclusively of high-frequency bicuculline-sensitive bursts. The increased propensity to bursting of thalamocortical cells following RE disconnection indicates that the activity of GABAergic local interneurons is released after RE lesion as if RE cells exerted an inhibitory action upon local interneurons (Steriade et al., 1985). It is now known that RE axons have access not only to projection neurons, but also to short-axoned GABAergic cells (Montero and Singer, 1985).

The abolition of spindle rhythms after thalamic transections and kainic lesions is not attributable to unspecific effects since the same phenomenon is observed, in the absence of any experimental damage, at the level of the anterior nuclei which do not receive connections from the RE nucleus (Steriade et al., 1984). Simultaneous recordings of anterior thalamic and intralaminar thalamic cells show that the former do not display spindles and rhythmic bursts, whereas the latter (which receive powerful projections from the RE nucleus) exhibit spindle oscillations and related spike bursts (Paré, Steriade, Deschênes, and Oakson, 1987). All this, despite the fact that anterior thalamic nuclei appear to be endowed with the same intrinsic membrane properties as disclosed in other thalamocortical neurons (Deschênes et al., 1984; Jahnsen and Llinás, 1984a, 1984b) such as, for example, the low-threshold spike de-inactivated by membrane hyperpolarization (Figure 3). Again, these data stress the importance of thalamic synaptic networks involving the RE neurons in the genesis of spindle rhythms.

Do spindles originate from the activity of reciprocal loops between relay and RE thalamic nuclei or, alternatively, are they generated within the network of RE cells? We tested the latter possibility that points to the pacemaker properties of RE neurons as spindle generators and indeed found that both spontaneous and evoked spindle oscillations can be recorded in the rostral pole of the RE nucleus disconnected by appropriate transections from the thalamic nuclei and cerebral cortex (Steriade, Domich, Oakson, and Deschênes, 1987). The long-lasting bursts of the deafferented RE neurons have a structure which is identical to that of normal RE cells (Domich, Oakson, and Steriade, 1986) and they occur in close time relation with spindle sequences recorded simultaneously, through the same microelectrode, in the RE nucleus (Figure 4).

We presume that the bursts and oscillations in the deafferented RE nucleus are generated by dendritic hyperpolarization through dendro-dendritic synapses of RE neurons (Deschênes, Madariaga-Domich, and Steriade, 1985; Yen, Conley, Hendry, and Jones, 1985). This process would de-inactivate a low-threshold conductance with the consequence of triggering a Ca spike, followed by GABA release and the hyperpolarization of postsynaptic dendrites. Hyper-

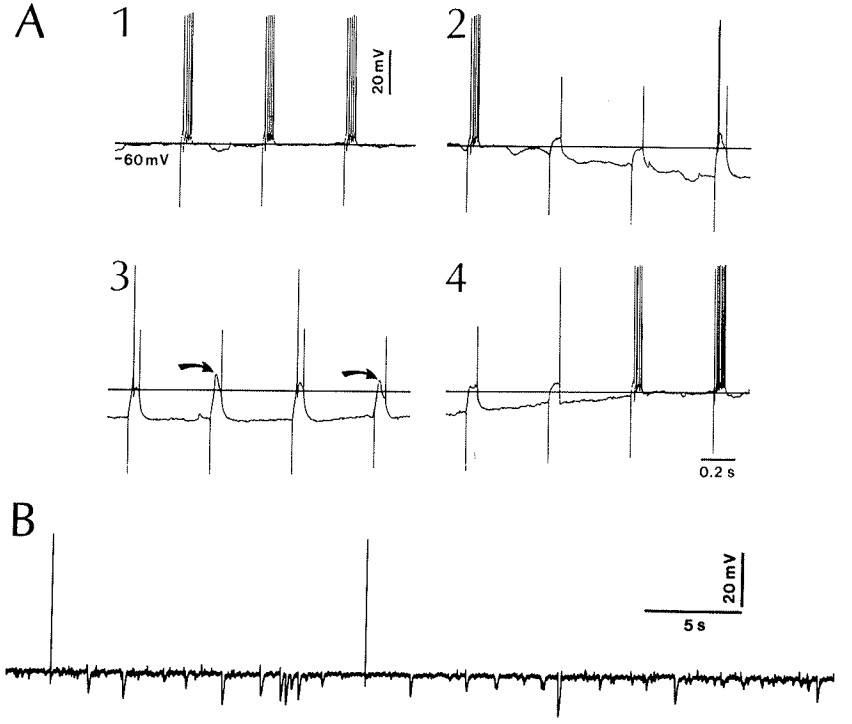

Figure 3: Absence of spindle oscillations in neurons of anterior thalamic nuclei, a group devoid of inputs from the RE nucleus. *A*: effect of membrane potential on firing mode of anteromedial thalamic neuron. In *A1*, tonic firing induced by a depolarizing pulse at resting membrane potential (-60 mV); the same pulse triggered a low-threshold slow spike crowned by a burst of fast action potential during hyperpolarization of cell membrane at about -72 mV (right part of *A2*, and *A3*; slow spikes in isolation are indicated by oblique arrows in *A3*); recovery of tonic mode in *A4*. *B*: absence of spindle oscillation in an anterior thalamic neuron; compare with the oscillations of other thalamocortical cells depicted in Figure 1.

polarization in the latter dendrites would then de-inactivate the Ca conductance and thus hyperpolarize synaptically coupled dendrites. In this manner, oscillations could start at any point in the network and spread to adjacent elements. As to the question whether any extrinsic factor is necessary to trigger the spindle sequences in the RE network, this could only be answered by investigating the occurrence of oscillations in RE slices. Since the rodent RE nucleus lacks dendro-dendritic synapses (Ohara and Lieberman, 1985), *in vitro* studies should be performed in cat. The presence of spindle oscillations in the deafferented RE nucleus does not exclude that, in the normal condition of an intact thalamus, hyperpolarizations and spike bursts in discrete thalamic foci

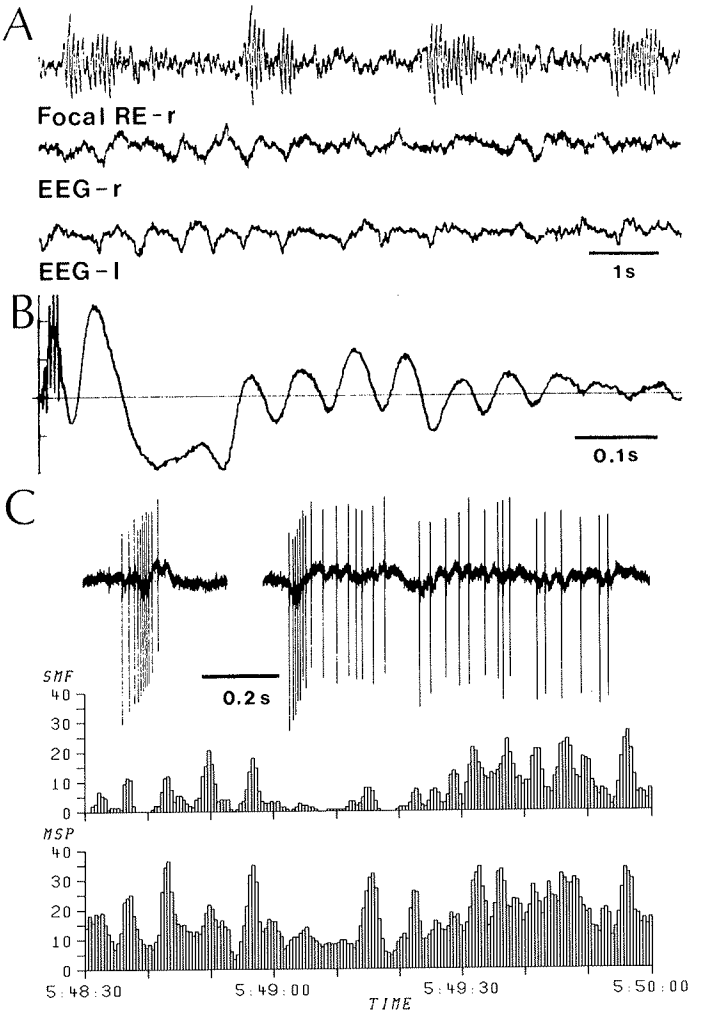

Figure 4: Spontaneous and evoked spindle oscillations in the RE nucleus disconnected from its thalamic and cortical inputs. See the histological aspect of transections that deafferented the rostral pole of the RE nucleus in Steriade et al. (1987). *A*: spindle sequences recorded focally in the RE nucleus and absence of spindle rhythmicity (but presence of slow waves) in the cortical EEG after bilateral thalamic transections. *B*: evoked spindle-like oscillations in the deafferented RE nucleus (thalamic and cortical transections) by stimulating the white matter overlying the caudate nucleus; 50 averaged traces. *C*: slow rhythm of spindle sequences and related burst oscillations in the deafferented RE nucleus by thalamic and cortical transections. At top, a short and a long spike burst from this period. Below, a computer-generated graph showing the sequential mean frequency (SMF) of the neuron and the normalized amplitude of focal waves filtered for spindle waves (MSP) recorded simultaneously through the same microelectrode. Abscissa indicates real time.

(as they occur through disfacilitation at sleep onset) may be decisive factors in triggering the RE oscillator through the collaterals of thalamocortical axons. Focal spindles in given thalamic zones would develop, however, into generalized and synchronous spindling only by spread of oscillations throughout the RE nuclear complex which, by virtue of its generalized thalamic projections, will induce cyclic hyperpolarization-rebound sequences in almost all thalamoneocortical systems.

The function of the long-lasting periods of hyperpolarizations in thalamocortical neurons during EEG-synchronized epochs is probably to ensure forebrain deafferentation during resting behavioral states. The thalamus is the first relay station where the incoming messages are blocked from the very onset of sleepiness. This was shown by testing the input-output operations of thalamic nuclei in behaving animals, in response to a stimulus applied to prethalamic afferent fibers. While the presynaptic component does not change across the shifts in the states of vigilance, there is a progressively diminished amplitude, up to the complete disappearance, of the postsynaptic response as the animal passes from waking to the sleepy state (Steriade, Iosif, and Apostol, 1969). This obliteration of the transfer function at sleep onset is a key thalamic operation that deprives the cerebral cortex of the input required to elaborate a response and can be regarded as a prelude for falling asleep.

Why, then, instead of a state with an uninterrupted hyperpolarization, are the postinhibitory rebounds beating cyclically during sleep spindles? One reason may be that the periodic rebound spikes would prevent the metabolic inertia—which may result from complete absence of cell discharges during tens of minutes or even hours of EEG-synchronized sleep, and would therefore favor the quick passage from this resting period to activated states, such as waking or REM sleep. It is also possible that the rhythmic spike bursts in thalamocortical axons would create a favorable condition for synaptic efficacy and dendritic growth in neocortical neurons. Rather than a period of brain rest and abject mental annihilation, the state of EEG-synchronized sleep may serve to consolidate the synaptic circuitry and to facilitate the storage of information acquired during wakefulness.

The Passage from the Oscillatory Mode to Brain Activation

The substrate of spindle blockade and generalized EEG desynchronization upon arousal from sleep must be somewhere in the brainstem reticular core (Moruzzi and Magoun, 1949) since a preparation with a high collicular transection displays continuous spindle sequences and ocular signs of sleep whereas a preparation with a bulbo-spinal cut exhibits alternating EEG patterns of waking and sleep (Bremer, 1937). During the 1960's, Purpura and his colleagues investigated intracellularly the effects of brainstem reticular stimulation and suggested that EEG desynchronization results from inhibition

of some inhibitory processes (Purpura, McMurtry, and Maekawa, 1966). Purpura's conclusions have been essentially confirmed (cf. Singer, 1977). However, the anatomical substrate, the chemical transmitterrs and the detailed electrophysiological mechanisms involved in these brainstem-thalamic effects remained to be clarified—beginning with the disclosure of the involved pathways since experiments using autoradiography and antidromic invasion techniques failed to reveal brainstem reticular projections to many relay and associational thalamic nuclei.

In recent studies, we combined choline acetyltransferase (ChAT) immunohistochemistry with retrograde tracing after injections of horseradish peroxidase conjugated with wheat germ agglutinin (WGA-HRP) confined within the limits of various thalamic nuclei of cat and monkey to reveal the contribution of different cholinergic and non-cholinergic cell aggregates of the upper brainstem reticular formation in the afferentation of the thalamus. These experiments disclosed that all major sensory and motor thalamic nuclear complexes, as well as intralaminar and RE thalamic nuclei, receive cholinergic projections from the peribrachial (pedunculopontine) and laterodorsal tegmental nuclei at the midbrain-pontine junction and from non-cholinergic neurons located at more rostral levels of the midbrain reticular formation.

Stimulation of the midbrain reticular formation prevents the periodic recurrence of intracellularly recorded spindle sequences in thalamocortical neurons (see Figure 5) and cuts off an ongoing spindle sequence (see Figure 6A).

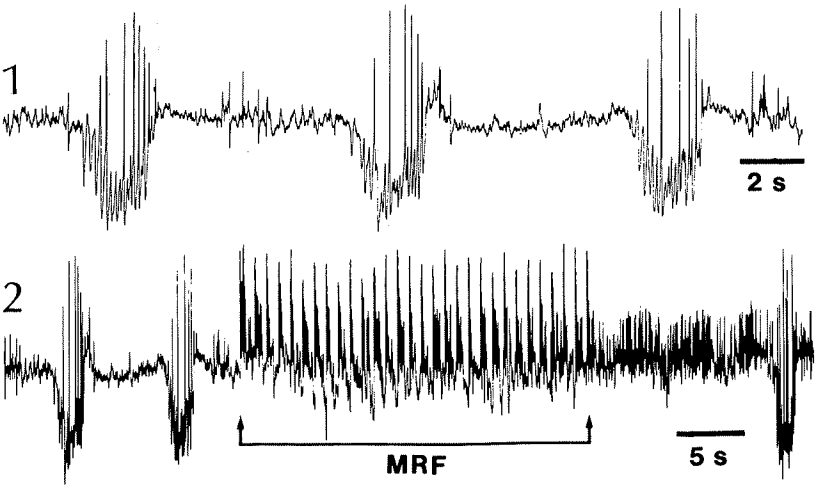

Figure 5: The slow rhythm of hyperpolarizing spindle sequences and its suppression during midbrain reticular formation (MRF) stimulation. Intracellular ink-written (Brush) recording of a ventrolateral thalamic neuron. In 2, MRF stimulation (a shock-train at 300 Hz, lasting for 60-ms) was applied every second.

This is the cellular correlate of the classical reticular-elicited desynchronizing reaction. Since spindles originate in the RE nucleus, we recorded the brainstem reticular effect at the very site of spindle genesis (Hu, Steriade, and Deschênes, 1986). Stimulation of the peribrachial nucleus elicits, after an initial depolarization at a latency of 7 to 10 msec, a prolonged (0.2–0.5 sec) hyperpolarization accompanied by a marked conductance increase that effectively blocks ongoing spindle sequences (see Figure 6B). The hyperpolarization is a muscarinic effect since it does not appear in scopolamine-treated animals, in which the initial excitation may extend for

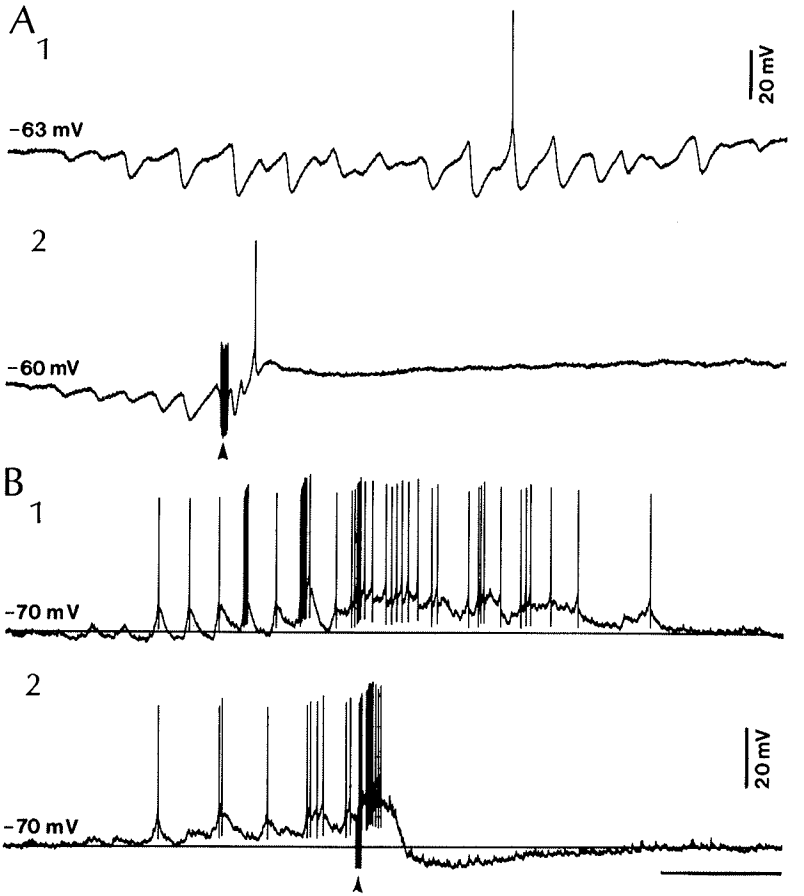

Figure 6: Blockade of ongoing spindle sequences of geniculostriate (in A) and in perigeniculate (in B) neurons by a 6-shock-train to the brainstem peribrachial nucleus (arrow heads in *A2* and *B2*).

50–200 msec. Both components of the biphasic depolarizing-hyperpolarizing RE-cell response to brainstem peribrachial stimulation are direct effects. The early excitation can be obtained under deep barbiturate anesthesia (a condition that abolishes the short-latency depolarization of thalamocortical cells) and after large lesions of dorsolateral thalamic nuclei. On the other hand, the increased conductance during the long-lasting hyperpolarization is probably due to a K current, in view of McCormick and Prince's (1986) studies *in vitro*. It is improbable that this biphasic response sequence evoked by stimulating the brainstem reticular core is due to co-activation of passing fibers issuing from locus coeruleus since an early excitation with spike discharges and a subsequent period of suppressed firing were obtained in RE neurons by stimulating the peribrachial nucleus in behaving animals, after chronic bilateral lesions of locus coeruleus (Steriade et al., 1986).

Further studies are needed to elucidate the role of the pathways linking the cholinergic brainstem cell-groups to the basal forebrain neurons whose widespread cortical projections probably play an important role in the cholinergic activation of the cerebral cortex. Besides, cholinergic and non-cholinergic neurons of the diagonal band nuclei and substantia innominata project to RE and some medial thalamic nuclei (Steriade, Parent, Paré, and Smith, 1987), where their modulatory actions converge with those of brainstem reticular neurons.

During the EEG-desynchronized behavioral states a tonic firing mode replaces the rhythmic burst discharges that are characteristic of quiet sleep. During wakefulness, the tonic discharge pattern is associated with enhanced excitability of long-axoned thalamic and cortical neurons and also with short-lasting inhibitory processes (Steriade and Deschênes, 1974) which subserve an accurate discrimination of incoming messages and a fine control of performances. These and other (Livingstone and Hubel, 1981) data congruently suggest an enhanced activity, during the waking state, of the progenitors of inhibitory operations related to discriminatory functions. As yet, there is no direct evidence of midbrain reticular or natural arousal effects upon identified GABAergic local-circuit cells. Studies by Sillito, Kemp, and Berardi (1983) suggest that the effect of ACh, a major transmitter of the ascending brainstem reticular system, is tripartite: an inhibition of the oscillations' pacemaker (the RE nucleus), a facilitation of excitatory responses of thalamocortical cells to an optimal stimulus, and an enhancement of stimulus-specific inhibitory influences on the relay cells. The complete (electrophysiological, morphological and immunohistochemical) identification of short-axoned inhibitory cells and the investigation of their behavior upon arousal from sleep are among the most tantalizing tasks in the future.

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