In: Michael A. Arbib (Ed.) The Handbook of Brain Theory & Neural Networks, Cambridge, MA: MIT Press 1995

metric representation and tedious parameter estimation, the BPNN probabilistic modeling gives a more relaxed and more reliable solution, as evidenced by successful application to textured image modeling and segmentation of both artificial and real-world textures.

It can be argued that the proposed BPNN modeling approach requires more parameters (interconnection weights of the BPNN) than the number of clique parameters used in a conventional MRF modeling. Even though this challenge is valid, there is no way to increase the number of clique parameters for a better performance without increasing the order of the neighborhood system under the very restricted MRF formulation. More importantly, when the order of the neighborhood system increases in an MRF model, the number of clique parameters increases exponentially, and the parameter estimation performance degrades rapidly. Conversely, the BPNN probabilistic modeling increases its number of interconnection weights linearly (i.e., the input dimension increases), with slight performance degradation observed from our experience.

In spite of its seemingly superior performance based on a limited set of simulations, the proposed BPNN texture modeling can possibly suffer performance degradation in the presence of texture rotation, texture scaling, and also larger number of gray levels. All these difficulties are yet to be overcome before the BPNN texture modeling can be of practical use.

Acknowledgments. This research was partially supported by the National Science Foundation under Grant No. ECS-9014243, and by NASA under Contract No. NAGW-1702.

Road Map: Vision
Background: Markov Random Field Models in Image Processing
Related Reading: Figure-Ground Separation; Regularization Theory and Low-Level Vision

References

Besag, J., 1974, Spatial interaction and the statistical analysis of lattice systems (with discussion), *J. R. Statist. Soc. Ser. B*, 36:192–326.

Besag, J., 1986, On the statistical analysis of dirty pictures, *J. R. Statist. Soc. Ser. B*, 48:259–302.

Chellappa, R., and Jain, A. K., 1993, *Markov Random Fields: Theory and Application*, Orlando, FL: Academic Press.

Cross, G. R., and Jain, A. K., 1983, Markov random field texture modeling, *IEEE Trans. PAMI*, 5:149–163.

Derin, H., and Elliott, H., 1987, Modeling and segmentation of noisy and textured images using Gibbs random fields, *IEEE Trans. Pattern Analysis Machine Intell.*, 9:39–55.

Duda, R. O., and Hart, P. E., 1973, *Pattern Classification and Scene Analysis*, New York: Wiley.

Gelfand, A., Hills, S. E., Racine-Poon, A., and Smith, A. F. M., 1990, Illustration of Bayesian inference in normal data models using Gibbs Sampling, *J. Am. Statist. Assoc.*, 85:972–985.

Geman, S., and Geman, D., 1984, Stochastic relaxation, Gibbs distribution, and the Bayesian restoration of images, *IEEE Trans. Pattern Analysis Machine Intell.*, 6:721–741.

Greenspan, H., Goodman, R., and Chellappa, R., 1991, Texture analysis via unsupervised and supervised learning, in *Proceedings of the International Joint Conference on Neural Networks*, Piscataway, NJ: IEEE, vol. 1, pp. 639–644.

Hwang, J. N., and Chen, T. Y., 1993, Textured image segmentation via neural network probabilistic modeling, in *Proceedings of the International Conference on Neural Networks*, Piscataway, NJ: IEEE, vol. 3, pp. 1702–1707.

Kashyap, R., and Chellappa, R., 1983, Estimation and choice of neighbors in spatial interaction model of images, *IEEE Trans. Inform. Theory*, 29:60–72.

Kollias, S., and Sukissian, L., 1992, Adaptive segmentation of textured images using linear prediction and neural networks, in *Proceedings, Neural Networks for Signal Processing*, Piscataway, NJ: IEEE, pp. 401–410.

Makhoul, J., 1991, Pattern recognition properties of neural networks, in *Proceedings of the IEEE Workshop on Neural Networks for Signal Processing*, Piscataway, NJ: IEEE, pp. 173–186.

Muhamad, A. K., and Deravi, F., 1993, Neural network texture classifiers using direct input coocurrence matrices, in *Proceedings of the International Conference on Acoustics, Speech and Signal Processing*, Piscataway, NJ: IEEE, vol. 5, pp. 117–120.

Richard, M. D., and Lippmann, R. P., 1991, Neural network classifiers estimate Bayesian a posteriori probabilities, *Neural Computation*, 3: 461–483.

Schumacher, P., and Zhang, J., 1994, Texture classification using neural networks and discrete wavelet transform, in *Proceedings of the International Conference on Image Processing*, Piscataway, NJ: IEEE, vol. 3, pp. 903–907.

Visa, A., 1990, A texture classifier based on neural network principles, in *Proceedings of the International Joint Conference on Neural Networks*, Piscataway, NJ: IEEE, vol. 1, pp. 491–496.

Won, C. S., and Derin, H., 1988, Maximum likelihood estimation of gaussian Markov random field parameters, in *Proceedings of the International Conference on Acoustics, Speech and Signal Processing*, Piscataway, NJ: IEEE, pp. 1040–1043.

Thalamocortical Oscillations in Sleep and Wakefulness

Terrence J. Sejnowski, David A. McCormick, and Mircea Steriade

Introduction

The brain spontaneously generates complex patterns of neural activity. As the brain enters slow-wave (quiescent) sleep, the rapid patterns characteristic of the aroused state are replaced by low-frequency, synchronized rhythms of neuronal activity. At the same time, electroencephalographic (EEG) recordings shift from low-amplitude, high-frequency rhythms to large-amplitude, slow oscillations. In what follows, we concentrate primarily on this slow-wave sleep, rather than rapid eye movement (REM) sleep, whose oscillatory properties resemble those of wakefulness. Thus, "sleep" without further qualification will mean "quiescent sleep."

The dramatic reduction in forebrain responsiveness during sleep, the pervasiveness of these changes, and the discovery of the underlying specific cellular mechanisms suggest that sleep oscillations are highly orchestrated and highly regulated. Experimental and modeling studies have shown how sleep rhythms emerge from an interaction between the intrinsic properties of these neurons and the networks through which they interact.

The thalamus and cerebral cortex are intimately linked by means of reciprocal projections. The thalamus is the major gateway for the flow of information toward the cerebral cortex and is the first station at which incoming signals can be blocked by synaptic inhibition during sleep. This mechanism contrib-

utes to the shift that the brain undergoes as it changes from an aroused state, open to signals from the outside world, to the closed state of sleep. The early stage of quiescent sleep is associated with EEG spindle waves, which occur at a frequency of 7–14 Hz; as sleep deepens, waves with slower frequencies (0.1–4 Hz) appear on the EEG recording. This review of these thalamocortical oscillations is adapted from Steriade, McCormick, and Sejnowski (1993).

Delta Oscillations

Delta waves (1–4 Hz) were initially shown to arise between cortical layers 2–3 and 5 (Steriade, Jones, and Llinás, 1990). Intracellular recordings in vivo and in vitro indicate that the thalamus is also involved in the generation of this rhythm (Figure 1*A*, 1*B*). A delta-frequency rhythm can be generated in single cells by the interplay of two intrinsic currents of thalamocortical neurons: the hyperpolarization-activated cation current (I_h) and the transient low-threshold Ca^{2+} current (I_t). A wide variety of other ionic currents (see ION CHANNELS: KEYS TO NEURONAL SPECIALIZATION for a general view of such channels), with different voltage dependencies and kinetics of activation and inactivation, contribute to the shaping of the amplitude and time course of each burst of action potentials, as revealed through biological experiments and computational modeling (Figure 1) (McCormick and Huguenard, 1992; Lytton and Sejnowski, 1992).

The hyperpolarization of thalamocortical cells is a critical factor for the interplay between I_h and I_t that generates delta oscillation. At the normal resting level in vivo, I_t is inactivated, but a hyperpolarization of 10 mV can lead to spontaneous, self-sustained delta oscillation (Figure 1*A*). The dependence of

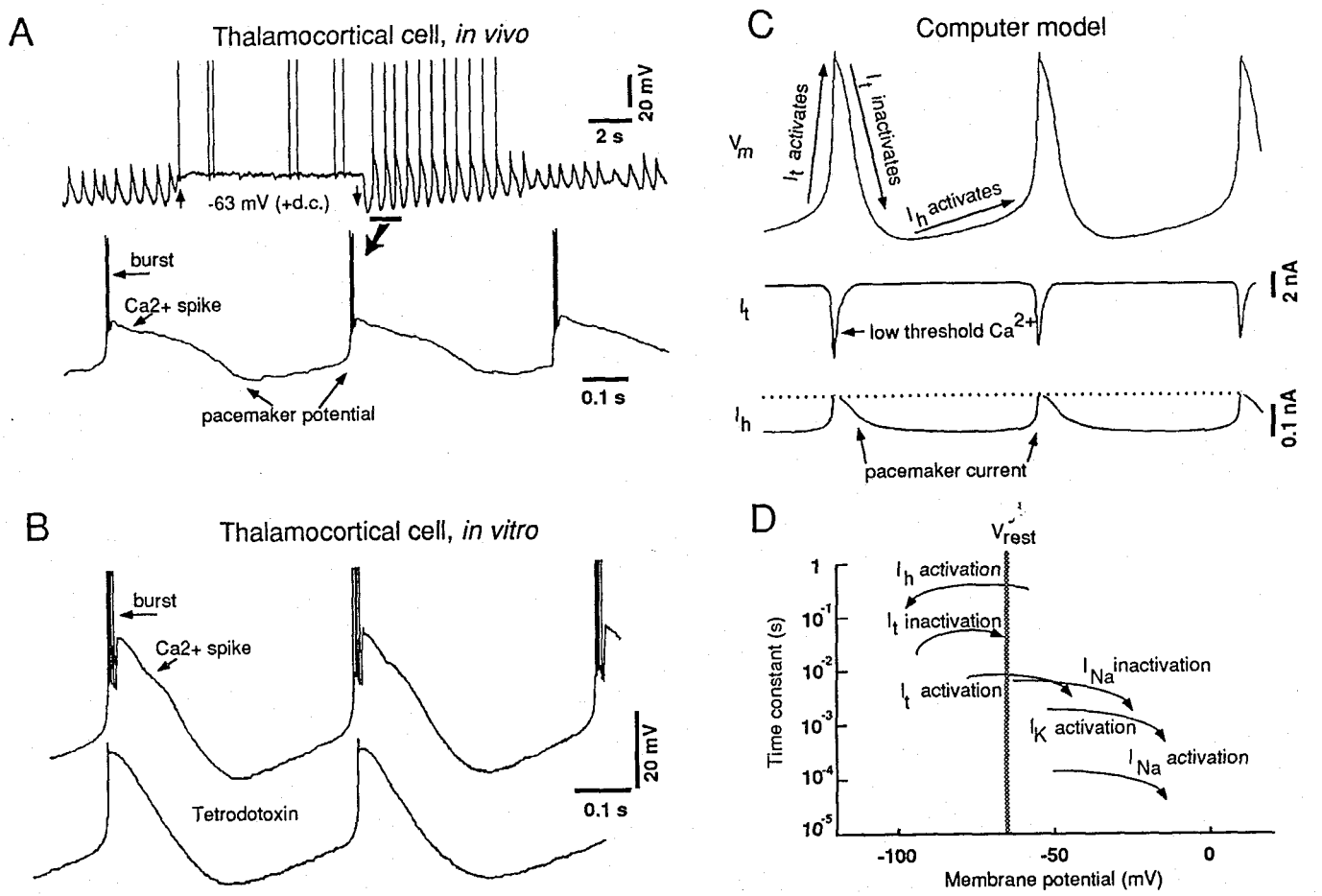

Figure 1. Intrinsic cellular mechanisms of thalamic delta oscillation. *A*, Voltage-dependency of delta oscillation. Intracellular recording in vivo of the lateroposterior thalamocortical neuron after decortication of areas projecting to that nucleus in an anesthetized cat is shown. The cell oscillated spontaneously at 1.7 Hz. A 0.5-nA depolarizing current pulse (between arrows), bringing the membrane potential to −63 mV, prevented the oscillation, and its removal set the cell back into the oscillatory mode. Three cycles marked by the horizontal bar in the upper trace are expanded below. *B*, Spontaneous rhythmic burst firing in a cat lateral geniculate relay cell recorded in vitro before and after block of voltage-dependent Na^+ conductances with application of the Na^+ channel blocker tetrodotoxin. *C*, Computational model of rhythmic generation of I_t as a consequence of interplay between I_t and the pacemaker current I_h. As the membrane becomes depolarized by I_h from hyperpolarized levels, the threshold for I_t is reached, leading to a Ca^{2+} spike. *D*, Diagram of activation and inactivation for the primary ionic currents in thalamocortical cells. Each arc represents the time constant for activation or inactivation of a voltage-dependent current. Most currents begin to activate (or inactivate) on the left side of the arc and are fully activated (or inactivated) on the right side. One exception is the cation current I_h, which activates with hyperpolarization and does not inactivate. Different combinations of currents are active at different membrane potentials. The voltage-dependent Na^+ current, I_{Na}, and the delayed rectifier K^+ current, I_K, are responsible for the fast action potentials; V_{rest} is the resting potential. (Reprinted with permission from Steriade, M., McCormick, D. A., and Sejnowski, T. J., 1993, Thalamocortical oscillations in the sleeping and aroused brain, *Science*, 262:679–685; © AAAS.)

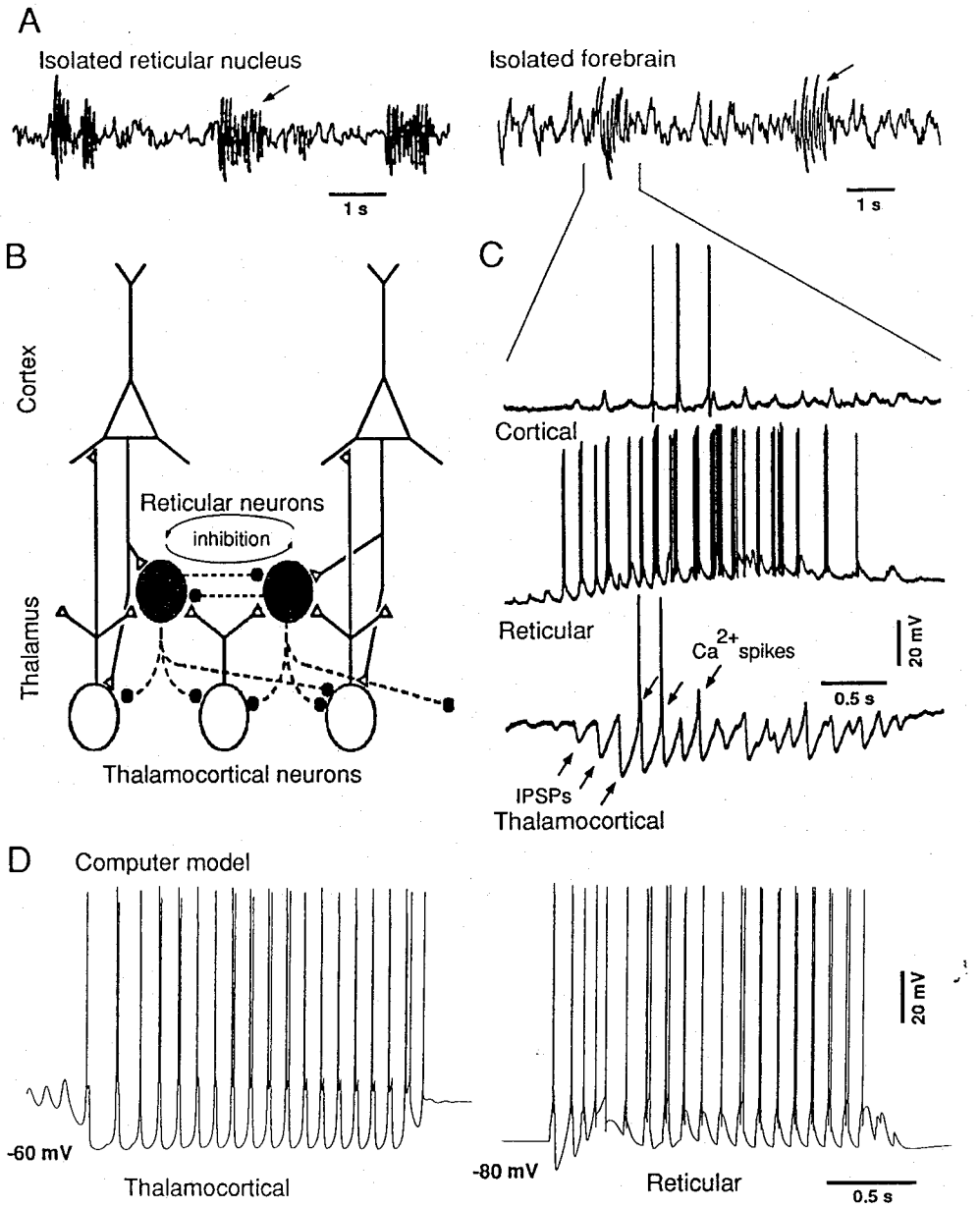

Figure 2. Sleep spindles. *A*, left: Field potentials recorded in vivo through a microelectrode inserted in the deafferented reticular thalamic nucleus of a cat. The arrow indicates one spindle sequence. *A*, right: Spindles recorded in vivo in the intralaminar centrolateral thalamic nucleus of a cat in an isolated forebrain preparation. Two spindle sequences are shown (the second marked by an arrow), and between them are lower-frequency (delta) waves. *B*, Schematic diagram of neuronal connections involved in spindling. *C*, Intracellular recordings of one spindle sequence in three neuronal types (cortical, reticular thalamic, and thalamocortical) of cats in vivo. *D*, Computer model of 8–10-Hz spindling in a pair of interconnected thalamocortical and reticular neurons. A burst of spikes in the thalamocortical cell excites the reticular thalamic cell, which in turn hyperpolarizes and produces a rebound burst in the thalamocortical neuron (as in vivo; compare with part *C*). (Reprinted with permission from Steriade, M., McCormick, D. A., and Sejnowski, T. J., 1993, Thalamocortical oscillations in the sleeping and aroused brain, *Science*, 262:679–685; © AAAS.)

delta oscillation on membrane hyperpolarization can also be demonstrated in simulations of thalamic neurons based on Hodgkin-Huxley-like kinetic models (see SYNAPTIC CURRENTS, NEUROMODULATION, AND KINETIC MODELS) of the ionic currents (Figure 1*C*).

Corticothalamic volleys potentiate and synchronize the delta oscillations of simultaneously recorded thalamic cells. In simulations of thalamocortical cells oscillating in the bursting mode at delta frequency, depolarizing cortical inputs are easily able to reset the cell to a new phase of its rhythm. Thalamic synchronization can also be induced by stimulating cortical foci that are not directly connected to the thalamic nuclei where the recordings are performed; this recruitment of thalamic cells may be achieved through the reticular thalamic nucleus, which receives collaterals of layer 6 corticothalamic cells and thalamic neurons that project to the cortex. The reticular cells are exclusively inhibitory and project back to the thalamus, but not to the cerebral cortex, and also innervate other cells of the reticular thalamic nucleus (Figure 2*B*). The reticular nucleus is uniquely positioned to influence the flow of information between the thalamus and the cerebral cortex (see THALAMUS).

Spindle Waves

Spindle oscillations consist of 7–14-Hz waxing and waning field potentials, grouped in sequences that last for 1–3 s and recur once every 3–10 s (see Figure 2*A*). The EEG spindles are the epitome of brain electrical synchronization at sleep onset, an electrographic landmark for the transition from waking to sleep that is associated with loss of perceptual awareness. These oscillations are generated in the thalamus as the result of synaptic interactions and intrinsic membrane properties of inhibitory neurons of the reticular thalamic nucleus and excitatory thalamocortical cells, and their interaction with cortical pyramidal neurons (see Figure 2*B*).

In intracellular recordings of reticular and thalamocortical cells as well as from computational modeling, these two neuronal classes behave inversely during spindles (Figure 2*C*). In reticular cells, rhythmic (7–14 Hz) bursts are generated by low-threshold Ca^{2+} spikes and are superimposed on a slowly rising and decaying depolarizing envelope. The bursts of reticular cells inhibit large numbers of thalamocortical cells through their divergent GABAergic axons, leading to the appearance of rhythmic (7–14 Hz) inhibitory postsynaptic potentials (IPSPs) in thalamocortical neurons (Figure 2*C*). Some of these IPSPs result in enough removal of inactivation of the low-threshold Ca^{2+} current to be followed by a rebound Ca^{2+} spike and an associated burst of action potentials (Figure 2*C*). These periodic bursts in thalamocortical cells converge onto reticular neurons and facilitate their rhythmic oscillation.

A simple model consisting of a thalamocortical cell reciprocally interacting with a reticular cell already demonstrates the essential features of spindling (Destexhe, McCormick, and Sejnowski, 1993). The waxing and waning of the spindling in this two-neuron model is controlled by the intracellular calcium level in the thalamocortical neuron, which increases with each Ca^{2+} spike; calcium binding to the I_h channels changes their voltage dependence and eventually terminates the spindle, as shown in Figure 2*D* (Destexhe, Babloyantz, and Sejnowski, 1993).

Isolation of the reticular nucleus from the rest of the thalamus and cerebral cortex abolishes spindle oscillations in thalamocortical systems, but the deafferented reticular thalamic nucleus can generate oscillations at spindle frequencies (Steriade et al., 1987). Axonal and, in some species, dendrodendritic interconnections between reticular cells may allow the coupling and interaction of these endogenous oscillators, thereby generating oscillations in an isolated nucleus. Models of simplified reticular thalamic neurons with full connectivity and slow mutual inhibition exhibit synchronous oscillatory activity, but the frequency is below the range of the spindling rhythm (Wang and Rinzel, 1993; Destexhe et al., 1994a). An array of model reticular neurons with fast inhibition between locally connected neurons exhibits 8–10-Hz oscillations in the local field potential in the model (based on the average membrane potential for a cluster of nearby neurons) that wax and wane in a fashion similar to what has been observed in vivo (Destexhe et al., 1994a).

Spindling has been observed in thalamic slice preparations (von Krosigk, Bal, and McCormick, 1993). However, when the reticular cells were isolated from the thalamocortical cells, spindling was abolished. The modeling suggests that that may occur because a larger and more intact collection of reticular thalamic cells is needed to generate spindle waves autonomously. Another possible reason is that the presence of neuromodulators in vivo keeps the resting levels of reticular cells more depolarized than in vitro; in the model, the oscillations in the reticular network are abolished at resting levels that are too hyperpolarized (Destexhe et al., 1994b).

Traveling spindle waves have been observed in vitro (McCormick, unpublished data) and in thalamic models based on sheets of interacting thalamocortical and reticular neurons (Destexhe and Sejnowski, unpublished modeling).

Absence Seizures

The spindles of natural sleep are related to the development of a peculiar pattern of oscillatory activity, the spike-and-wave EEG complexes, which are associated with absence (petit mal) epileptic seizures. Because the reticular thalamic nucleus is central to the genesis of spindles, decreasing or abolishing the inhibitory efficacy of reticular neurons on thalamocortical cells would also decrease the incidence of epileptic spike-and-wave discharges. This hypothesis is supported by recent experiments showing that, in animals with genetic absence epilepsy, thalamic injections of a selective agonist of $GABA_B$ receptors increase the incidence of spike-and-wave discharges, whereas injections of a $GABA_B$ antagonist decrease these seizures in a dose-dependent manner.

The activation of $GABA_B$ receptors in thalamocortical neurons produces a slow increase in K^+ conductance and a deep hyperpolarization and also enhances the removal of inactivation of the low-threshold Ca^{2+} spike. As a consequence, there is a larger than usual rebound burst discharge in a greater than usual proportion of thalamocortical cells. These facilitated rebound bursts further excite reticular cells, quickly resulting in the generalization of paroxysmal activity. Further support for the $GABA_B$ hypothesis derives from a model of spindling in which the frequency of spindling could be shifted from 8–10 Hz to 2–4 Hz by slowing the kinetics of the inhibitory synaptic potentials from that of $GABA_A$ (5–25 ms) to that of $GABA_B$ (100–250 ms) (von Krosigk, Bal, and McCormick, 1993; Destexhe, McCormick, and Sejnowski, 1993).

Arousal

Electrical activation of certain brainstem and hypothalamic regions, including the reticular activating system, causes a variety of neurotransmitters, including acetylcholine (ACh), norepinepherine (NE), serotonin (5-HT), histamine (HA), and glutamate to be released though diffuse ascending axonal arborizations. These neuromodulators mimic arousal by suppressing sleep spindles, delta waves, and slow cellular rhythms and by replacing these low-frequency oscillations with activity similar to that of the awake, attentive animal. In cortical pyramidal neurons, ACh, NE, 5-HT, HA, and glutamate can reduce three distinct K^+ currents, thereby resulting in a significantly enhanced responsiveness to depolarizing inputs and changes in the neuronal firing mode (McCormick, 1992). Adenosine and GABA can reduce excitability by increasing membrane K^+ conductance.

These neurotransmitter systems abolish the low-frequency rhythms in thalamocortical systems during waking and rapid eye movement (REM) sleep and also promote more tonic activity or the appearance of high-frequency oscillation. The changes in firing between sleep and arousal in thalamic neurons are accomplished by depolarization of the membrane potential by 5–20 mV, which inactivates the low-threshold Ca^{2+} current and therefore inhibits burst firing. These results have been simulated in models of thalamocortical and reticular neurons.

High-Frequency Oscillations

Changes in the activity pattern generated by cortical neurons and circuits are less stereotyped than those of thalamic cells and circuits, although some common features exist. The low-frequency oscillations of the cortical EEG disappear on arousal and are replaced by higher-frequency (20–80 Hz, mainly around 40 Hz) rhythms. As in the thalamus, these alterations in cortical activity occur, at least in part, through the depolarization of pyramidal cells, presumably through the reduction of specialized K^+ conductances by ACh, NE, and other neuromodulators.

The high-frequency (20–80 Hz) oscillations in the EEG occur during some behaviors, such as immobility during hunting and focused attention to stimuli during complex sensory or motor tasks. Neurons throughout the nervous system (e.g., the

retina, lateral geniculate nucleus, and cortex) have the ability to generate repetitive trains of action potentials in the frequency range of 20–80 Hz, although the synchronization of this activity into behaviorally relevant subgroups of widely spaced neurons has only been demonstrated in the cerebral cortex (Gray, 1994).

The diversity of cortical cells and their complex interactions make it difficult to model cortical networks with the same confidence with which thalamic networks have been modeled. However, it is not difficult to generate oscillatory activity in the 20–80-Hz range with networks of simplified neurons (Koch, 1993). These models reveal the need to regulate the tendency of recurrent networks to oscillate. The excitability of neurons can be controlled by inhibition. However, inhibition is also an efficient mechanism for synchronizing large populations of pyramidal neurons because of voltage-dependent mechanisms in their somas and the strategic location of inhibitory boutons on the somas and the initial segments of axons, where action potentials are initiated (Lytton and Sejnowski, 1991). Realistic simulations of cortical neurons show that sparse excitatory connectivity between distant populations of neurons can produce synchronization within one or two cycles, but only if the long-range connections are made on inhibitory as well as excitatory neurons (Bush and Sejnowski, in press).

Discussion

This article has focused on the events that occur during the transition from wakefulness to sleep and on the rhythms of deep, slow-wave sleep. Dreams occur during another sleep state, REM sleep. This sleep state is characterized by an abolition of low-frequency oscillations and an increase in cellular excitability, much like wakefulness, although motor output is significantly inhibited. Despite great interest, there is no generally accepted function for dreams or, for that matter, for the sleep state itself.

During spindling and slow-wave sleep, the thalamus excites the cortex with patterns of activity that are more spatially and temporally coherent than normally would be encountered in the awake state. Depolarizing pulses of Ca^{2+} that enter the thalamic and cortical neurons may influence enzyme cascades and regulate gene expression, homeostatically adjusting the balance of ionic currents and regulatory mechanisms. This widespread activity could be used to reorganize cortical networks after learning occurs during the awake state (Wilson and McNaughton, 1994).

Inhibitory neurons in the thalamus and cortex are of particular importance in producing the synchrony and controlling the spatial extent of the coherent populations. Synchrony and other network properties could be used to control the flow of information between brain areas and to decide where to store important information. Synchronization enhances the strength of signals, but also reduces the amount of information that can be encoded.

The ascending neuromodulatory transmitter systems delicately tune the state and excitability of the different parts of the nervous system so that it is appropriate for the analysis of sensory information, the cognitive processing and storage of this information, and the subsequent performance of the appropriate neuronal and behavioral responses. Uncovering and modeling the cellular mechanisms of these dynamic changes may provide important clues to long-standing questions ranging from the functional role of sleep to the nature of cognitive representations.

Road Map: Biological Networks
Background: Ion Channels: Keys to Neuronal Specialization
Related Reading: Neuromodulation in Invertebrate Nervous Systems; Oscillatory and Bursting Properties of Neurons; Synchronization of Neuronal Responses as a Putative Binding Mechanism

References

Bush, P., and Sejnowski, T. J., in press, Inhibition synchronizes sparsely connected cortical neurons within and between columns of realistic network models, *J. Computat. Neurosci.*

Destexhe, A., Babloyantz, A., and Sejnowski, T. J., 1993, Ionic mechanisms for intrinsic slow oscillations in thalamic relay neurons, *Biophys. J.*, 65:1538–1552.

Destexhe, A., McCormick, D. A., and Sejnowski, T. J., 1993, A model for 8–10 Hz spindling in interconnected thalamic relay and reticularis neurons, *Biophys. J.*, 65:2473–2477.

Destexhe, A., Contreras, D., Sejnowski, T. J., and Steriade, M., 1994a, A model of spindle rhythmicity in the isolated thalamic reticular nucleus, *J. Neurophysiol.*, 83:803–818.

Destexhe, A., Contreras, D., Sejnowski, T. J., and Steriade, M., 1994b, Modeling the control of reticular thalamic oscillations by neuromodulators, *NeuroReport*, 5:2217–2220.

Gray, C., 1994, Synchronous oscillations in neuoronal systems: Mechanisms and functions, *J. Computat. Neurosci.*, 1:11–38.

Koch, C., 1993, Computational approaches to cognition: The bottom-up view, *Curr. Opin. Neurobiol.*, 3:203–208.

Lytton, W. W., and Sejnowski, T. J., 1991, Simulations of cortical pyramidal neurons synchronized by inhibitory interneurons, *J. Neurophysiol.*, 66:1059–1079.

Lytton, W. W., and Sejnowski, T. J., 1992, Computer model of ethosuximide's effect on a thalamic neuron, *Ann. Neurol.*, 32:131–139.

McCormick, D. A., 1992, Neurotransmitter actions in the thalamus and cerebral cortex and their role in neuromodulation of thalamocortical activity, *Prog. Neurobiol.*, 39:337–388.

McCormick, D. A., and Huguenard, J. R., 1992, A model of the electrophysiological properties of thalamocortical relay neurons, *J. Neurophysiol.*, 68:1384–1400.

Steriade, M., Domich, L., Oakson, G., and Deschênes, M., 1987, The deafferented reticular thalamic nucleus generates spindle rhythmicity, *J. Neurophysiol.*, 57:260–273.

Steriade, M., Jones, E. G., and Llinás, R. R., 1990, *Thalamic Oscillations and Signaling*, New York: Wiley-Interscience.

Steriade, M., McCormick, D. A., and Sejnowski, T. J., 1993, Thalamocortical oscillations in the sleeping and aroused brain, *Science*, 262: 679–685.

von Krosigk, M., Bal, T., and McCormick, D. A., 1993, Cellular mechanisms of a synchronized oscillation in the thalamus, *Science*, 261: 361–364.

Wang, X.-J., and Rinzel, J., 1993, Spindle rhythmicity in the reticularis thalami nucleus: Synchronization among inhibitory neurons, *Neuroscience*, 53:899–904.

Wilson, M., and McNaughton, B., 1994, Reactivation of hippocampal ensemble memories during sleep, *Science*, 265:676–679.