COMPARTMENTAL models of thalamic reticular (RE) neurons were investigated based on current-clamp and voltage-clamp data. Spontaneous oscillations in the model arise from the interaction between inhibitory synaptic conductance and the membrane time course of RE cells. These oscillations,  

Modeling the control of reticular thalamic
current, both based on current clamp data.\textsuperscript{5,6} \( I_{\text{Na}}, I_{\text{K}} \) are the fast Na\(^+\) and K\(^+\) currents responsible for the generation of action potentials (taken from Ref. 17). Details of the kinetics of these currents, based on estimates from physiological data, are given in Ref. 10.

The synaptic current \( I_{\text{GABA}} \) represents the intra-RE inhibitory current, mediated by \( \gamma \)-aminobutyric acid (GABA). The receptors on R cells are of GABA\(_A\) type with a very weak GABA\(_B\) component. Only the GABA\(_A\) synaptic currents were modeled here, using a kinetic scheme for the binding of neurotransmitter to postsynaptic receptors.\textsuperscript{18} The current is described by the first-order equation:

\[
I_{\text{GABA}} = g_{\text{GABA}} m (V - E_C) \tag{2}
\]

\[
g_{\text{KL}} = \tilde{g}_{\text{KL}} m \tag{4}
\]

\[
\frac{dm}{dt} = \alpha [S] m - \beta (1 - m) \tag{5}
\]

where \( \tilde{g}_{\text{KL}} (= 1 \text{nS}) \) is the maximal leak conductance for K\(^+\) and \([S]\) represents the concentration of second messenger. For RE neurons, the only experiments available for noradrenergic and serotoninergic depolarization are from delivery of agonists \textit{in vitro}:\textsuperscript{21} the response lasted up to several minutes. Comparable data are not available for electrical stimulation of noradrenergic and serotoninergic receptors. Brief stimulation of peribrachial cholinergic nuclei evoked a short lasting (about 2 s) hyperpolarization in RE neurons\textsuperscript{23} and there are many indications that these muscarinic receptors have the same G protein-based activation mechan-
Control of reticular thalamic oscillations

oscillations. This behavior was extremely robust to changes in parameters such as the reversal potential of GABAergic currents ($E_{\text{Cl}}$), the values of the synaptic conductances or the amount of leak $K^+$ current affected by NE/5HT synapses. Typically, $E_{\text{Cl}}$ and the resting level were varied in a range of 5 mV around the present values; the simulations showed that sustained oscillations arose only if there was a sufficient ‘driving force’, of at least several millivolts (around 10–15 mV, depending on the maximal conductance of GABAergic synapses), between the resting membrane potential.
FIG. 2. Dependence of RE oscillatory behavior on the membrane potential. Simulation of a network of 100 RE cells interconnected with their neighbors through GABAergic synapses. The top 10 traces represent the activity of 10 neurons in the network and the bottom trace is the average membrane potential. 20% of NE/5HT synapses were initially activated (as in Fig. 1b). In these conditions, the network showed self-sustained oscillations at a frequency of 10–16 Hz and the average membrane potential displayed waxing and waning fluctuations of amplitude. After 2 s (first arrow), all NE/5HT synaptic activity was suppressed; the resulting hyperpolarization prevented the network from sustaining oscillations. Depolarizing (second arrow) or hyperpolarizing (third arrow) current pulses injected simultaneously in all neurons (with random amplitude) could not restore spontaneous oscillations. These conditions might correspond to the membrane potential of RE cells in vitro.

The model predicts that spontaneous sustained oscillations could be observed in slices of the RE nucleus if the resting level of RE cells be brought to more depolarized values. This could be achieved by using bath application of NE/5HT agonists in weak concentration such as to depolarize all RE neurons to the – 60 to – 70 mV range. Another prediction is that NE/5HT antagonists should suppress oscillatory spontaneous oscillations if their resting levels were brought to more depolarized values, such as those seen in vivo. Such a depolarization might be provided by noradrenergic or serotonergic agonists, or by glutamate metabotropic receptor agonists. More generally, this model suggests that neuromodulatory systems cannot only regulate the firing mode of RE cells, but they can also control the degree of information.