Spiking neural network for recognizing spatiotemporal sequences of spikes

Dezhe Z. Jin*

Howard Hughes Medical Institute and Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139, USA

(Received 16 October 2003; published 26 February 2004)

Sensory neurons in many brain areas spike with precise timing to stimuli with temporal structures, and encode temporally complex stimuli into spatiotemporal spikes. How the downstream neurons read out such neural code is an important unsolved problem. In this paper, we describe a decoding scheme using a spiking recurrent neural network. The network consists of excitatory neurons that form a synfire chain, and two globally inhibitory interneurons of different types that provide delayed feedforward and fast feedback inhibition, respectively. The network signals recognition of a specific spatiotemporal sequence when the last excitatory neuron down the synfire chain spikes, which happens if and only if that sequence was present in the input spike stream. The recognition scheme is invariant to variations in the intervals between input spikes within some range. The computation of the network can be mapped into that of a finite state machine. Our network provides a simple way to decode spatiotemporal spikes with diverse types of neurons.

I. INTRODUCTION

Sensory neurons in many brain areas spike with precise timing to stimuli with temporal structures. Examples include auditory neurons in cochlear ganglion and auditory brain stem nuclei [1], and ganglion cells in retina [2]. Temporally complex stimuli can thus drive different sensory neurons to spike at different times, forming a spatiotemporal coding of the stimuli. Some temporally stable stimuli are also transformed into spatiotemporal codes. For instance, different odors evolve distinctive spatiotemporal spikes of the projection cells in the locust antennal lobe [3]. Over the years, many aspects of encoding with spatiotemporal spikes have been explored [4–6]. However, relatively few biologically plausible proposals exist for reading out such spike codes.

In this paper, we describe a decoding scheme using a spiking neural network. The network consists of a synfire chain of excitatory neurons [7], and two globally inhibitory interneurons of different types that provide delayed feedforward and fast feedback inhibition, respectively. The network signals recognition of a specific spatiotemporal spike sequence when the last excitatory neuron down the synfire chain spikes, which happens if and only if that sequence was present in the input spike stream. The recognition is invariant to variations in the intervals between input spikes within some range. The sequence recognizing dynamics of the network is characterized by transitions between up states and down states of neuronal membrane potentials, and can be understood in terms of finite state machine, which is a powerful conceptual model widely used for understanding digital computers [8], natural language processing [9], and artificial neural networks [10]. Our network is also a simple example of biologically useful computations arising from the diversity of neuronal properties and the specificity of the synaptic organizations for the inhibitory interneurons observed in experiments [11,12].

The way that our network recognizes the patterns of the input spatiotemporal spikes is different from most of previous biologically plausible proposals, which rely on detecting the time intervals between input spikes. These include internal clocks [13], delay lines [14,15], oscillators [16], short term synaptic plasticity [17], and distributed decaying processes coupled with transient synchrony [18]. Rather than detecting time intervals, the excitatory neurons in our network detect the spatiotemporal orders of the input spikes. This detecting scheme is insensitive to changes of the input interspike intervals within a range. This invariance is a useful feature for processing sensory stimuli such as speech, which can have variable timing between different parts. The length of the recognized sequence is limited only by the number of neurons in the synfire chain. This is markedly different from some of the previous proposals, for which the maximum length of the recognized spike patterns is fundamentally limited by the time scales of the underlying biophysical processes [17,18].

II. RESULTS

A. The network structure and the neuron properties

The network consists of a number of excitatory neurons, labeled N1, N2, etc., and two inhibitory neurons labeled I1 and I2 (see Fig. 1). The excitatory neurons form a synfire chain [7], with each neuron in the chain sending an excitatory connection to its immediate next neighbor down the chain. Each excitatory neuron receives excitatory spike inputs from afferents coming from different sensory neurons. The input afferent to N1 is labeled 1, and that to N2 labeled 2, etc. All excitatory neurons excite interneuron I1, and I1 sends back inhibition to them. I1 provides a global feedback inhibition to the excitatory neurons. Interneuron I2 is excited by all input afferents, and sends inhibition to all excitatory neurons. The excitatory neurons connect back to I2. I1 also sends an inhibitory connection to I2. I2 provides a delayed feedforward inhibition to the excitatory neurons, and is regulated by I1.
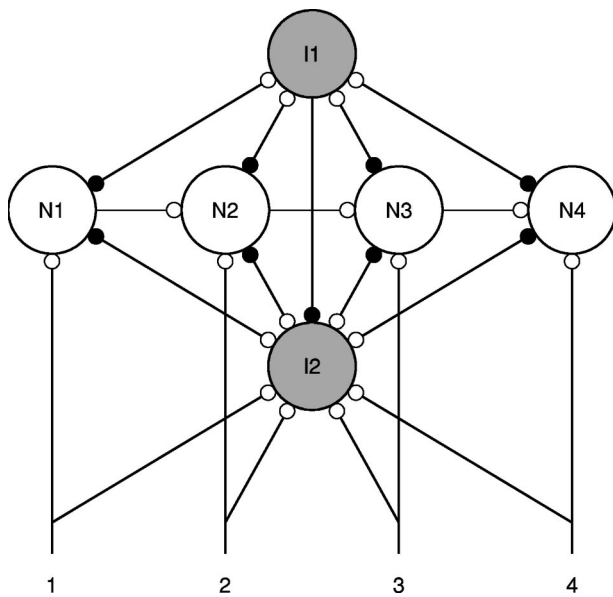
*Electronic address: djin@mit.edu

FIG. 1. Structure of the network. The excitatory neurons are indicated with white circles labeled N1, N2, etc. The two inhibitory neurons are indicated with gray circles labeled I1 and I2. The input afferents are labeled 1, 2, etc. The excitatory synapses are indicated with open circles ending the connection lines, and the inhibitory synapses are indicated with solid circles. The excitatory neurons form a synfire chain, with each neuron exciting its immediate next neighbor down the chain. Interneuron I1 is excited by the excitatory neurons, and sends back inhibition to them, thus proving a feedback inhibition. Interneuron I2 is excited by all input afferents, and inhibits all excitatory neurons. The excitatory neurons send back excitations to I2. I2 is also inhibited by I1. I2 provides a feedforward inhibition to the excitatory neurons, and is regulated by I1.

The spiking properties of the neurons under injection of constant currents are shown in Fig. 2. We model the excitatory neurons as leaky integrate and fire neurons with finite refractory period. Injection of a suprathreshold constant current causes an excitatory neuron to spike regularly. We model interneuron I1 as integrate and fire neuron with Kv3.1-Kv3.2 and Kv1 channels, which are two types of K^+ channels. The model we use is suggested by Lewis and Gerstner [19], and is a modified version of a conductance based model proposed by Erisir *et al.* [20] for the fast spiking inhibitory neurons in the mice neocortex. The modified model replaces the spike generation process in the original model with simple resets of the membrane potential and the conductance parameters, followed by a refractory period. Otherwise the modified model is identical to the original one. There is a range of injected constant current, in which the neuron spikes only once with a small latency. When the magnitude of the constant current exceeds the upper limit of this range, the neuron spikes a regular spike train. The latency to the first spike from the resting state is small. We model interneuron I2 as quadratic integrate and fire neuron with a finite refractory period. The model follows that of Hansel and Mato [21], who tuned the parameters to match the properties of a conductance based model of hippocampal inhibitory neurons proposed by Wang and Buzsaki [22]. Injection of a suprathreshold constant current causes the neuron to
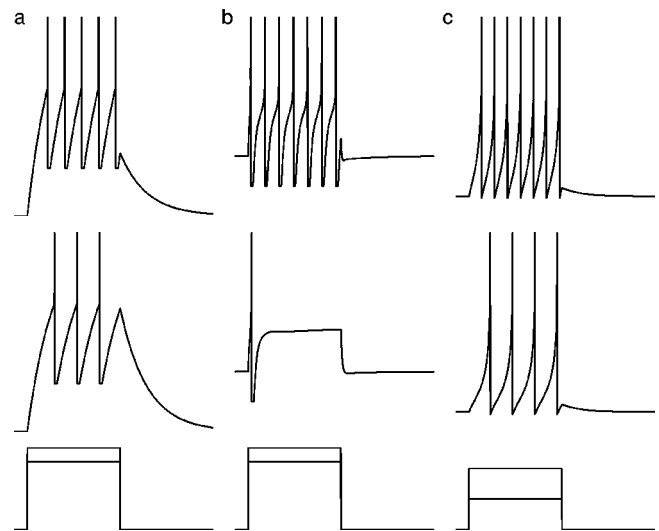

FIG. 2. Spiking properties of the neurons under constant current injections. Three columns of figures are shown. For each column, the upper figure is the voltage trace of the neuron with high current injection, and the middle one is that with low but suprathreshold current injection. The relative magnitudes of the high and low injection currents are shown in the bottom figure. (a) Excitatory neuron. (b) Interneuron I1. (c) Interneuron I2.

produce a regular spike train. Compared to I1, the latency to first spike from the resting state can be quite long for this neuron. Details of the neuron models are given in Appendix A.

The responses of the interneurons to suprathreshold excitatory spike inputs are displayed in Fig. 3. These properties are very important for the operation of our network. We require that the interneurons spike once for every excitatory spike input. With the time constant small and the conduc-
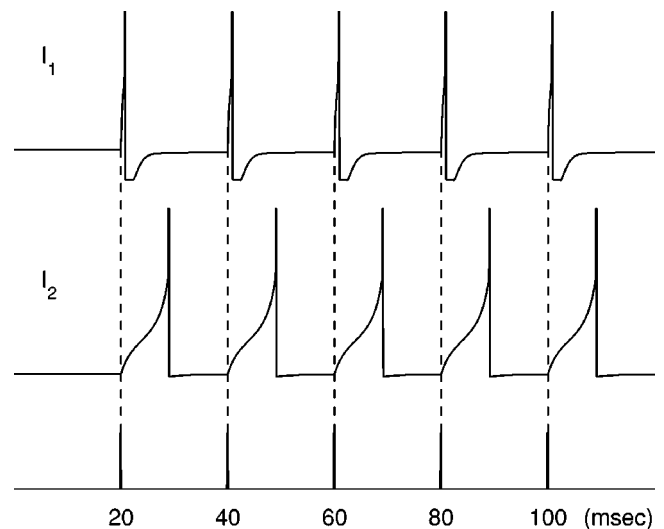

FIG. 3. Spiking properties of the interneurons under suprathreshold excitatory spike inputs. Top: the membrane potential trace of interneuron I1. Middle: the membrane potential trace of interneuron I2. Bottom: the times of the spike inputs. The interneurons spike once for every spike input. The latency of I1 is much smaller than that of I2.

tance large for the excitatory synapses, both neurons can satisfy this requirement. However, because of the different membrane properties, the spike latency is different. I1 spikes with a minimal delay, whereas I2 spikes with much longer delay. With our models of the interneurons, the latency of the feedback inhibition through I1 can be less than 1 msec, and the delay of the feedforward inhibition through I2 can be in the range from 5 msec to 15 msec.

B. Spike sequence recognition

The spatiotemporal input spikes are represented as spike sequences with the interspike intervals assumed to be in between T_{min} and T_{max}. An example is 2431 . . . , which means that afferent 2 carries a spike first, followed by afferents 4, 3, 1, etc. The input spikes drive the spiking of the neurons in the network. In some parameter regimes, spikes in an excitatory neuron indicate the recognition of a specific input sequence. N4 spikes if and only if there is a sequence 1234 in the input stream, N3 if and only if 123, and N2 if and only if 12. In other words, N4 recognizes input spike sequence 1234, N3 recognizes 123, and N2 recognizes 12. N1 is a special neuron; it spikes once every time it gets an input spike. We describe the parameter regimes in the following section. In this section, we describe how the network operates to achieve such selectivity.

Except N1, each excitatory neuron has two states at the times of the input spikes, the down state and the up state. The down state is characterized by a low membrane potential and a net negative or zero synaptic current. An input spike to a neuron in the down state cannot make it spike. This is mainly due to the spiking of interneuron I2: The powerful feedforward inhibition checks the depolarizing action of the input spike before the membrane potential could reach the spike threshold from the down state. The feedforward inhibition further returns the neuron to the down state by the time of the next spike input. Hence, input sequences such as 3333 cannot cause N3 to spike from the down state. Initially, all excitatory neurons are in the down states, and any input sequence without 1 in it will not be able to make any of the excitatory neurons spike. In this case, each input spike depolarizes for short duration the membrane potential of the neuron that receives the input. I2 spikes in response to each input spike, whereas I1 does not spike at all.

The excitatory neuron can also be in the up state, which is characterized by a high membrane potential. Upon receiving an input spike, the neuron in the up state can spike before the arrival of the feedforward inhibition from I2. An excitatory neuron goes from the down state to the up state if it receives a spike from its immediate neighbor preceding it in the synfire chain.

An input to N1 makes it spike once. The spike from N1 causes N2 to jump to the up state; it also induces I1 to spike before I2 could. The spike from I1 inhibits all neurons in the chain, and keeps N2 from spiking before the next input; it also suppresses the spiking of I2, abolishing the feedforward inhibition that would have followed the spike input. This suppression is important for keeping N2 in the up state at the next input. Besides the inhibition from I1, I2 also receives an

excitation from N1, which balances the effect of the inhibition to keep the membrane potential of I2 subthreshold but not hyperpolarized. Without the balancing, the responsiveness of I2 to spike inputs can be compromised.

After the input to N1, there are three possibilities for the next input.

(a) If the input is again 1, N1 spikes again, and N2 stays in the up state without spiking.

(b) If the input is 2, N2 spikes since it is in the up state. The spike from N2 induces spiking of I1, which inhibits all neurons in the chain as well as I2, suppressing the feedforward inhibition. N2 returns to the down state because of the reset, the refractory period, and the feedback inhibition. N3 goes to the up state.

(c) If the input is neither 1 nor 2, no neuron in the chain spikes since N2 is the only one in the up state. The delayed feedforward inhibition is not suppressed, since I1 does not spike. The feedforward inhibition causes N2 to return to the down state. Thus, all neurons are in the down states again, and the network returns to the initial state. In sum, N2 spikes if and only if the input sequence is 12.

We can extend the above scenario to N3 and show that N3 spikes if and only if the input sequence is 123. A different role of the feedback inhibition from I1 is introduced. Suppose that the input is 1231. Before the last input at 1, N4 is in the up state because N3 just spiked. But the input 1 causes N1 to spike and N2 to go to the up state. N4 receives the feedback inhibition via I1, and returns to the down state. This ensures that input sequence such as 12314 will not make N4 spike. The same scenario applies to all other neurons in the chain: neuron n spikes if and only if the spike sequence is 123 . . . n.

In this paper, we only consider the case of each excitatory neuron getting inputs from different sensory neurons. However, this restriction can be lifted by extending the role of the feedback inhibition. Suppose that two neurons are getting the same inputs, with one in the up state and the other in the down state. The neuron in the up state will spike first because it has higher membrane potential. This sends the feedback inhibition to the other neuron, causing it to remain in the down state even though it also receives an excitatory input. Thus, the previous scenario can be still preserved.

Several features of the network are crucial for the operation of the network. The feedforward inhibition is used for preventing the out-of-order input sequences from driving spikes in the excitatory neurons. The feedback inhibition prevents the excitatory neurons from spiking immediately after receiving spikes from upstream neurons; it returns the neuron that just spiked to the down state; finally, it makes every excitatory neuron downstream from N2 to return from the up state to the down state when N1 spikes. The refractory period of the neurons is important for ensuring that the most recently active neuron will not spike multiple times because of the residuals of the excitatory conductance. The inhibition from I1 to I2 is crucial for keeping the up state since it shuts down the powerful feedforward inhibition. For all these to work, the delay of the feedforward inhibition must be longer than the typical delay of the spiking of the excitatory neurons plus the delay of the feedback inhibition. In addition, the

minimum time interval between the input spikes must be longer than the delay of the feedforward inhibition. The synfire chain of the excitatory neurons is the main structure that ensures a sequential spiking of neurons when the recognized input sequence is present.

C. Tuning the parameters

In order for the network to operate as proposed, the connection strength of the synapses on the excitatory neurons cannot be arbitrary. The main parameters that need to be tuned are the feedforward excitatory conductance G_1, the lateral excitatory conductance G_2, the delayed feedforward inhibitory conductance G_3, and the feedback inhibitory conductance G_4. Related parameters are the time constant τ_E of the excitatory synapses, the time constant τ_I of the inhibitory synapses, the membrane time constant τ of the excitatory neurons, the range $(\Delta_{min}, \Delta_{max})$ of the delay of the feedforward inhibition, the range $(\delta_{min}, \delta_{max})$ of the delay of the feedback inhibition, the maximum delay to spike λ of the excitatory neurons, and the refractory time τ_R of the excitatory neurons. The feedforward excitatory conductance G_0 to N1 is different from that to other excitatory neurons, since N1 is special in the sense that it spikes to every input. The reversal potential of the excitatory synapses is 0 mV. The relative relationship of the potentials is $E_I < L < R < \Theta < 0$. Here E_I is the reversal potential of the inhibitory synapses, and L, Θ, and R are the resting membrane potential, the spike threshold, and the reset potential of the excitatory neurons, respectively.

The feedforward excitation conductance G_1 is bounded in a finite range. It cannot be too large, otherwise the sequence selectivity is lost since the neuron can spike even from the down state before the delayed feedback inhibition could prevent it from spiking. The conductance G_1 cannot be too small; otherwise the input spike cannot cause the neuron to spike even from the up state.

The lateral excitatory conductance G_2 is also bounded within an interval. The conductance G_2 cannot be too large; otherwise the spiking of an excitatory neuron will cause an immediate cascade of spiking of the neurons in the chain even without the spike inputs, since the feedback inhibition could not react fast enough to prevent this cascade. The conductance G_2 cannot be too small; otherwise neurons in the chain cannot jump to the up states when the upstream neurons spike.

The delayed feedforward inhibitory conductance G_3 must be large enough to prevent the neuron in the down state from jumping to the up state. It should also be large enough to return a neuron in the up state to the down state when the next input spike is not to the neuron in the up state. The feedback inhibitory conductance G_4 is also bounded from below. It must be large enough to counteract the excitatory effect of G_2 to ensure that the neuron receiving the lateral excitation does not spike immediately. It also must be large enough to make the neuron just spiked to return to the down state. Finally, it should be large enough to return any neuron in the up state, if it is not N2, to the down state when N1 spikes.
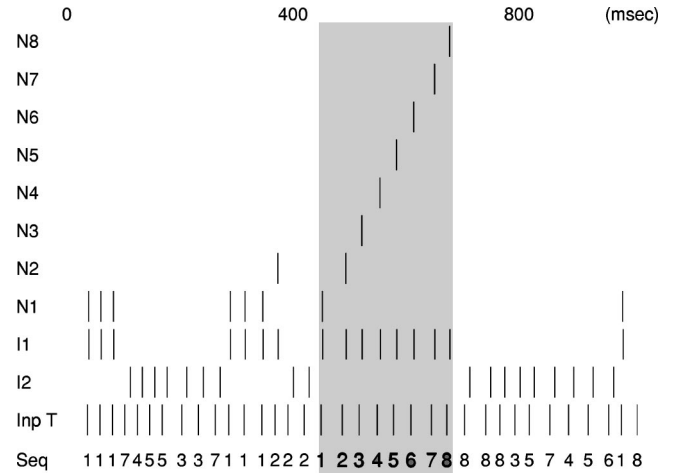

FIG. 4. Spike sequence recognition. The spike raster of neurons N1 to N8 and the interneurons I1 and I2 is plotted. The input spike sequence is indicated in the bottom row, and the input spike times are indicated with the vertical lines in the row next to the bottom row. N8 spikes if and only if the input stream contains a subsequence 12345678, as highlighted with the gray rectangle in the figure; therefore, N8 recognizes this particular subsequence.

The inhibitory conductance G_3 and G_4 also have upper bounds. The upper bound of G_3 is mainly due to the residual effect of the conductance. Too large G_3 causes the inhibitory effect to linger beyond one input spike, and suppresses the neurons from transiting to the up states. Similarly, G_4 has an upper bound because of the residual effects. Another upper bound for G_4 is determined by the requirement that the combined effect of G_2 and G_4 should leave the neuron in the up state. Too large G_4 cannot achieve this.

The lower bound T_{min} of the time intervals of the input spikes must be larger than the maximum Δ_{max} of the delays of the feedforward inhibition, as discussed before. Because of the finite synaptic time constants, the neurons need finite time to make transitions between the up and down states. Therefore, T_{min} should be sufficiently larger than Δ_{max}. In practice, setting $T_{min} = \Delta_{max} + 2\tau_I$ works quite well. The maximum T_{max} of the input spike intervals cannot be too large. This is because the natural decay of the neuron's membrane potential degrades the up states to the down states if long intervals are present in the input sequence. In this case, a correct sequence may not be able to spike the relevant neurons.

The above description of the bounds of the synaptic conductance can be put into mathematically rigorous forms. The details of the derivations and the results are given in Appendix B.

D. Performance of the network

To illustrate the network performance, we show a simulation result in Fig. 4. The network consists of eight excitatory neurons. The excitatory and inhibitory synapses on the excitatory neurons have time constants of 3 msec. The lower limit of the delay of the feedforward inhibition is 5 msec, and the upper limit is 7 msec. The membrane time constant of the excitatory neurons is 20 msec. The lower limit of the

input spike time intervals is 13 msec, and the upper limit is 32 msec. The values of the synaptic conductance are $G_1 = 2.4$, $G_2 = 4.8$, $G_3 = 20.2$, $G_4 = 22.8$, $G_0 = 4.4$, in the unit of the leak conductance of the excitatory neurons. Other relevant parameters are $E_I = -75$ mV, $L = -70$ mV, $R = -64$ mV, and $\tau_R = 2$ msec. The input spike sequences are generated to include a subsequence running from 1 to 8, a block of 111, a block of 888, a block of 11222, as well as two blocks of randomly selected sequences. The time intervals between the input spikes are selected randomly from the interval 13 msec and 32 msec, except for 111, whose intervals are 13 msec. As can be seen from the figure, N8 spikes only to the subsequence 12345678. A sequential spiking of neurons in the synfire chain leads to the spiking of N8. All other subsequences fail to spike N8. Extensive simulations with randomly generated input sequences confirm that the N8 spikes if and only if the subsequence 12345678 is present in the input stream. The spiking of I1 and I2 is complementary. When a subsequence is recognized, I1 spikes, and I2 is silent. When the subsequence contains no recognized sequences, I2 spikes, and I1 is silent. These spiking properties of the interneurons reflect their roles in shaping the sequence recognition function of the network.

In Fig. 5, we show the membrane potentials of the excitatory neurons in response to the spike inputs. Focus on N4 as an example. The membrane potential is in a hyperpolarized state most of the time. This is because the reversal potential of the inhibition is more negative than the resting membrane potential, and the neuron receives frequent inhibition from I1 or I2. When the neuron gets an out-of-order direct input, the membrane potential shows a brief depolarization. In the span of the recognized input sequence, the membrane potential stays in a depolarized state (up state) following the spike of N3. Subsequent direct input spikes the neuron, and the membrane potential returns to the hyperpolarized state.

E. Mapping into a finite state machine

The sequence recognizing dynamics of the network can be mapped into that of a finite state machine. We illustrate this point in Fig. 6 for the case of four excitatory neurons in the synfire chain. At the times of the spike inputs, the excitatory neurons as a system can have only four possible states. The first state is S1, in which all neurons are in the down state (except N1, for which the distinction of the up and down states cannot be made because it spikes for every input at afferent 1). Any input other than 1 will not make the system to leave S1, since all neurons stay in the down states. This is indicated in the diagram with an arrowed arc that starts from and returns to S1. An input to 1 causes N1 to spike and N2 to be in the up state. All other neurons are in the down states. This state of the system, S2, is characterized with N2 as the only neuron in the up state. The transition from S1 to S2 is indicated with a straight line arrow starting from S1 and ending at S2. From S2, there are three possibilities. If the input is 1, N1 spikes again, N2 remains in the up state, and all other neurons stay in the down state. In other words, the system stays in S2. This is indicated with the
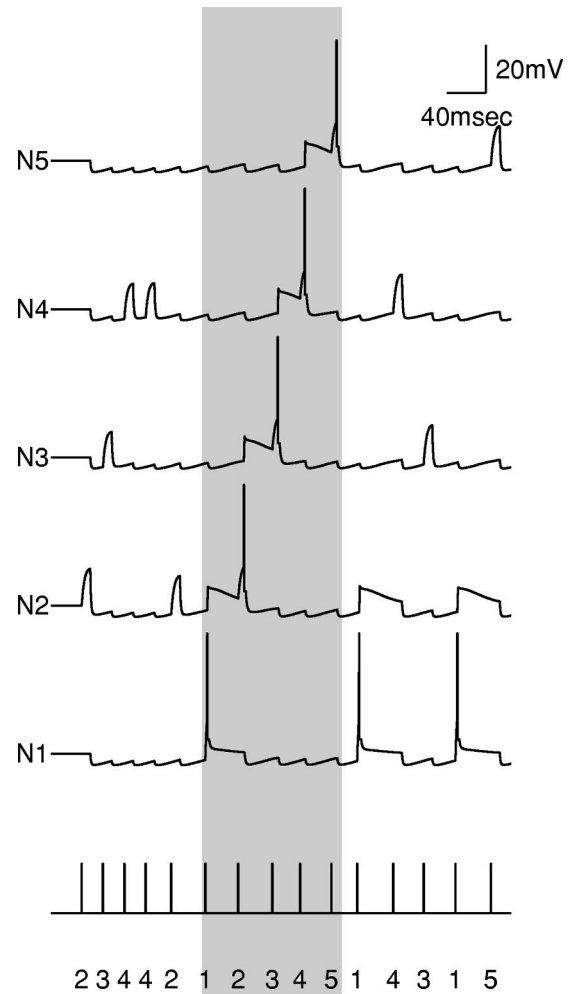

FIG. 5. Dynamics of the membrane potentials of the excitatory neurons. The bottom row of the figure shows the input spike sequence. The vertical lines in the row above the bottom row indicate the input spike times. The time period that contains the recognized sequence is indicated by the gray rectangle. Input spikes to neurons in the down states cause a short duration depolarization of the membrane potentials. Input spikes to the neurons in the up states cause spiking of the neurons. The down states are characterized by low membrane potentials with frequent inhibitory postsynaptic potentials, and the up state is characterized by the extended period of high membrane potentials.

arrowed arc starting from and returning to S2. If the input is neither 1 nor 2, no neuron spikes, and the feedforward inhibition makes N2 to return to the down state. Therefore, the system returns to S1 with all neurons in the down states. This is indicated with the arrowed arc starting from S2 and ending at S1. Finally, if the input is 2, N2 spikes and returns to the down state, while N3 jumps into the up state. This is a new state of the system, S3, with N3 as the only neuron in the up state. This transition from S2 to S3 is indicated with the straight arrow starting from S2 and ending at S3. Similarly, at S3, there are three possibilities. If the input is 1, N1 spikes, N2 jumps to the up state, and N3 returns to the down state because of the feedback inhibition. The system returns to S2. If the input is neither 1 nor 3, N3 returns to the down state, and the system returns to S1. Input 3 makes N3 to
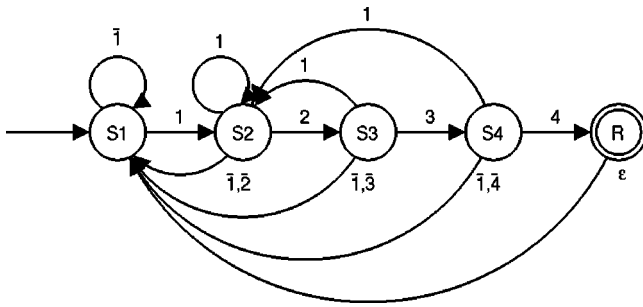
FIG. 6. The finite state machine corresponding to the dynamics of the network with four excitatory neurons. There are four states for the system of the excitatory neurons. S1 corresponds to the state of all neurons in the down states, S2 to that of only N2 in the up state, S3 to that of only N3 in the up state, and S4 to that of only N4 in the up state. The arrowed lines indicate the transitions between the states with the spike inputs. The inputs corresponding to each transition are printed near the lines. Here a bar on a number means "input other than." A special state R is indicated as the recognizing end state, with N4 spiking. From R, the system returns to S1 without any input (denoted with ϵ). There is a unique input sequence that can lead to each of the states S2, S3, S4, and R. For example, S4 can be reached only by input sequence 123. The spiking of N3 indicates the system has reached S4; meanwhile, it signals that the sequence 123 is recognized.

spike and N4 to jump to the up state. This leads to a new state of system, S4, with N4 as the only neuron in the up state. The same choices are there for S4, with the only difference being that for input 4, there is no next neuron in the chain to jump to the up state, so N4 spikes and the system returns to S1. The spiking of N4 signals the recognition of the input sequence 1234, so an end state R is added to show this fact. This end state is inserted to distinguish input 4 from other inputs that are neither 1 nor 4. These other inputs also cause the system to return to S1, but without causing N4 to spike.

In general, there are N states for the network with N excitatory neurons in the synfire chain. One state is S1, with all neurons in the down states. In all other states, only one neuron is in the up state. The sequence recognition is done through transitions between these finite states with the spike inputs. As shown in Fig. 6, the key for the sequence recognition is that a unique input sequence leads to each state (except S1). Reaching each state thus signals the detection of a specific input sequence.

III. DISCUSSION

A. Selectivity of spatiotemporal spikes

Neurons responding to specific spatiotemporal sensory inputs are found in various brain areas of animals. Cells in primate auditory cortex are selective to sound patterns with complex temporal and spectral context [23]; neurons in macaque nonprimary auditory cortex discriminate monkey vocalizations [24]; species-specific and bird's own songs evoke strong responses in neurons of songbird forebrain and premotor areas [25]; and specific spatiotemporal deflections of multiple whiskers produce strong responses in cells of rat

barrel cortex [26]. It is most likely that there are many biologically plausible mechanisms for such spatiotemporal selectivity. Our network provides one.

Two features of our network are noteworthy. First, the maximum length of the recognized spike sequence equals the number of the excitatory neurons in the chain, and can be arbitrarily long. Thus, the duration of the recognized sequence is not limited by biophysical time scales of the neurons such as the synaptic time constant and the membrane time constant. This is quite different from some other mechanisms of spatiotemporal selectivity, in which biophysical time scales constrain the maximum duration of the recognized spike patterns [17,18]. Second, time warping of the sequence does not affect recognition. Our mechanism only requires the correct ordering of the input spikes, not their exact timings. As long as the intervals between input spikes lie within a specific range, which can be quite broad, recognition is intact. Invariance to time warping is beneficial for tasks such as speech recognition [18].

B. Implementing a finite state machine with spiking neurons

Our network implements a finite state machine for spike sequence recognition. A finite state machine processes sequential inputs by transitions among a finite number of discrete states. For some finite state machines, certain states may be reached if and only if a specific sequence is present in the input stream. These states are thus selective to particular input sequences. Finite state machines are conceptually simple but functionally powerful, and have been used for analyzing digital computers [8] and the processing of natural languages [9]. Artificial neural networks such as discrete-time rate-model neural networks have long been mapped into finite state machines [10]. Our network shows that biologically plausible spiking neural networks can also be understood in terms of finite state machines.

The neurons and their connectivity in our network closely resemble structures found in various cortical areas. There are diverse types of interneurons in the brain, and different interneurons often participate in different subsets of connections [11,12]. For example, there are two common types of inhibitory interneurons in rat somatosensory neocortex, fast spiking neurons and low threshold spiking neurons. Fast spiking neurons get strong feedforward inputs from the thalamocortical neurons, whereas low threshold spiking neurons only get weak feedforward inputs [11]. Both types of neurons reciprocally connect with high probability to regular spiking excitatory neurons [11]. This synaptic organization of interneurons is quite similar to that of the two interneurons in our network. The synfire chain structure of excitatory neurons may also be quite common in some cortical areas, where the connection probability between the excitatory neurons is low [11].

C. Extension to more complex networks

The operation of our network relies on potent synapses that can reliably induce transitions between the down and the up states. The resulting changes of the postsynaptic membrane potentials can be as much as 15 mV, as evident in Fig.

5. The synapses are quite strong compared to those commonly observed in cortex. A possible remedy is to use synchrony. We can replace each excitatory neuron in the network with a pool of interconnected excitatory neurons that can transit collectively between the down states and the up states and spike synchronously from the up states. We can substitute the two interneurons with two groups of interneurons that spike synchronously. We can replace the input spike sequences with sequences of synchronized spikes from groups of sensory neurons. Such spatiotemporal sequences of synchronized spikes are observed in projection neurons in the locust antennal lobe [6]. Synchronous spikes can induce large changes of the postsynaptic potentials. Operating with synchrony should also make the network more robust against noise.

There are other biophysically plausible ways of implementing a sequence recognizing finite state machine. One possibility is to substitute each of the excitatory neurons in our network with a group of interconnected excitatory and inhibitory neurons that supports bistable persistent activity [27]. In this case, the down and up states of the neurons are replaced with the quiescent and persistent states of the neuron groups. This implementation may be more relevant for sequence selectivity in the prefrontal cortex. Other possibilities include using synaptic and cellular mechanisms such as N-methyl-D-aspartate receptors, voltage dependent conductance, rebound excitation from inhibition, etc., for creating the up and down states.

Our simple network recognizes a single spike sequence. Ultimately, such network should be embedded in more complex networks to expand the capacity of spatiotemporal spike processing. Viewed as devices that transform input spatiotemporal spikes to output spatiotemporal spikes, more complex spiking networks may be analyzed as finite state machines. Adding noise may turn the spiking networks into Markov models or related hidden Markov models, which are probabilistic versions of the finite state machines.

D. Searching for the network in the brain

Several properties of our network can be used to find it in the brain. First, the network structure is orderly. The excitatory neurons form a synfire chain, and two types of interneurons mediate fast feedback and delayed feedforward inhibition, respectively. Second, the spiking of the two interneurons is anticorrelated. Each input evokes spiking of either I1 or I2, but not both. A span of repeated spiking of I1 is complemented with a period of silence of I2, signaling the recognition of a long input spike sequence (see Fig. 4). Third, the complexity of the input patterns that drive the excitatory neurons is distributed. N1 spikes to every input to it, while neurons further down in the synfire chain are more selective and harder to drive with random inputs. Finally, the membrane potentials of the excitatory neurons (except N1) show up and down states and quick transitions between them. Neurons spike only at the end of up states. The neurons can also return to the down state from up state without spiking.

APPENDIX A: MODELS OF THE NEURONS AND THE SYNAPSES

We model the excitatory neurons as leaky integrate and fire neurons with finite refractory time after spike. The membrane potential of an excitatory neuron evolves according to

$$\tau \frac{dV}{dt} = L - V - g_E V - g_I (V - E_I). \quad (A1)$$

Here τ is the membrane time constant, L is the resting membrane potential, E_I is reversal potential of the inhibitory synapse, g_E is the excitatory synaptic conductance, and g_I is the inhibitory synaptic conductance. The synaptic conductance is scaled with the leak conductance of the neuron. If the membrane potential reaches the threshold Θ, the neuron spikes, and the membrane potential is reset to the reset potential R. After spike, the membrane potential stays at the reset potential for a refractory period τ_R. In the simulations of the paper, we use the following values for the parameters: $\tau = 20$ msec, $L = -70$ mV, $\Theta = -54$ mV, $R = -64$ mV, $E_I = -75$ mV, and $\tau_R = 2$ msec.

We model interneuron I1 as a multicurrent integrate and fire neuron. The model is modified from the conductance based fast spiking interneuron model of Erisir *et al.* [20], which contains fast deactivating Kv3.1-Kv3.2 K^+ channels. The modified neuron retains all subthreshold conductance currents, but replaces the spike generating process with simple threshold and reset mechanism. The neuron spikes if its membrane potential exceeds the threshold value, and the membrane potential and the gating variables are reset to fixed values after spike. The modification is proposed by Lewis and Gerstner [19]. The following is the model used in our simulations. The membrane potential V of the neuron satisfies the following equation:

$$C \frac{dV}{dt} = -I_L - I_{Na} - I_{K1} - I_{K3} - g_E V - g_I (V - E_I). \quad (A2)$$

Here $C = 1$ $\mu F/cm^2$ is the membrane capacitance; $I_L = g_L (V - E_L)$ is the leak current with the leak conductance $g_L = 1.25$ $\mu S/cm^2$ and the resting membrane potential $E_L = -70$ mV; $I_{Na} = g_{Na} m^3 h (V - E_{Na})$ is the Na^+ current with conductance $g_{Na} = 112.5$ $\mu S/cm^2$ and Na^+ reversal potential $E_{Na} = 74$ mV; $I_{K1} = g_{K1} n^4 (V - E_K)$ is the Kv1.3 current with conductance $g_{K1} = 0.225$ $\mu S/cm^2$ and K^+ reversal potential $E_K = -90$ mV; $I_{K3} = g_{K3} p^2 (V - E_K)$ is the Kv3.1-Kv3.2 current with conductance $g_{K3} = 225$ $\mu S/cm^2$; g_E is the excitatory conductance; g_I is the inhibitory synaptic conductance with reversal potential $E_I = -75$ mV. The gating variables satisfy the equation

$$\frac{dy}{dt} = \alpha_y(V)(1-y) - \beta_y(V)y, y = m, h, n, p, \quad (A3)$$

with

$$\alpha_m = (3020 - 40V)/\{\exp[-(V-75.5)/13.5]-1\}, \tag{A4}$$

$$\beta_m = 1.2262/\exp(V/42.248), \tag{A5}$$

$$\alpha_h = 0.0035/\exp(V/24.186), \tag{A6}$$

$$\beta_h = -(0.8712 + 0.017V)/\{\exp[-(V+51.25)/5.2]-1\}, \tag{A7}$$

$$\alpha_n = -(0.616 + 0.014V)/\{\exp[-(V+44)/2.3]-1\}, \tag{A8}$$

$$\beta_n = 0.0043/\exp[(V+44)/34], \tag{A9}$$

$$\alpha_p = (95-V)/\{\exp[-(V-95)/11.8]-1\}, \tag{A10}$$

$$\beta_p = 0.025/\exp(V/22.222). \tag{A11}$$

The spike threshold is -30 mV, and the refractory period after spike is 1.5 msec. After spike, the membrane potential is reset to -85 mV, and the gating variables m, h, n, p are reset to 0, 0.16, 0.88, 0.2, respectively.

We use the quadratic integrate and fire neuron model for interneuron I2. We follow Hansel and Mato [21] and set the parameters to match the properties of the Wang-Buszaki model of fast spiking hippocampal interneurons [22]. In the model, the membrane potential evolves according to

$$C\frac{dV}{dt} = A(V-V^*)^2 - I_c - g_E V - g_I(V-E_I). \tag{A12}$$

Here $C = 0.9467$ μF/cm^2 is the membrane capacitance; the other parameters have values $V^* = -59.5462$ mV, $A = 0.012\ 875 mS/cm^2/mV$, and $I_c = 0.1601\ mA/cm^2$; g_E is the excitatory synaptic conductance; g_I is the inhibitory synaptic conductance with reversal potential $E_I = -75$ mV. The spike threshold is -26.3462 mV, and the reset potential after spike is -64.1462 mV.

We use the "kick-and-decay" model of the synaptic conductance. Upon receiving an excitatory spike, the excitatory conductance of the neuron jumps a finite value G_E, which is the maximum conductance of the synapse that receives the spike:

$$g_E \rightarrow g_E + G_E. \tag{A13}$$

In between spikes, the conductance decays exponentially with time:

$$\tau_E \frac{dg_E}{dt} = -g_E. \tag{A14}$$

Here τ_E is the synaptic time constant of the excitatory synapse. Similarly, upon receiving an inhibitory spike, the inhibitory conductance of the neuron jumps a finite value G_I, the maximum conductance of the synapse that receives the spike; and in between spikes the conductance decays exponentially with time:

$$g_I \rightarrow g_I + G_I, \tag{A15}$$

$$\tau_I \frac{dg_I}{dt} = -g_I. \tag{A16}$$

The excitatory and inhibitory synapses on the excitatory neurons have time constants typically at 3 msec. The excitatory synapses on interneuron I1 have a time constant 2.5 msec, and inhibitory synapses have a time constant 1 msec. The excitatory and the inhibitory synapses on interneuron I2 have a time constant 1 msec.

The network dynamics is simulated with fourth-order Runge-Kutta method with time step 0.01 msec.

APPENDIX B: TUNING THE PARAMETERS

1. The pulse coupling limit

In this section, we use the δ pulse coupling limit of the synaptic conductance for tuning the parameters. This limit is sufficiently simple for analytical treatment, yet complex enough to retain the essence of the spiking dynamics. Previously, this limit was used to prove that the dynamics of a general class of recurrent networks of leaky integrate and fire neurons converge to spike sequence attractors [5]. In this limit, the synaptic time constant is set to zero, but the charge transfer at each spike is kept finite. More precisely, the synaptic conductance is modeled as

$$g_E = G_E \tau \delta(t - t_{spike}), \tag{B1}$$

$$g_I = G_I \tau \delta(t - t_{spike}). \tag{B2}$$

Here t_{spike} is the spike time, G_E and G_I are the maximum conductance of the excitatory and inhibitory synapses receiving the spike, and τ is the membrane time constant. An excitatory spike causes the membrane potential of the postsynaptic neuron to jump from its initial value V to

$$V^+ = Ve^{-G_E}. \tag{B3}$$

An inhibitory spike causes the membrane potential to jump to

$$V^+ = E_I(1 - e^{-G_I}) + Ve^{-G_I}. \tag{B4}$$

Simultaneous spikes of both inhibition and excitation cause the membrane potential to jump to

$$V^+ = E_{eff}(1 - e^{-G_E - G_I}) + Ve^{-G_E - G_I}. \tag{B5}$$

Here E_{eff} is the effective reversal potential, and is defined as

$$E_{eff} = \frac{G_I E_I}{G_E + G_I}. \tag{B6}$$

The combined effect of an excitation and an inhibition is an effective inhibitory synapse with the effective reversal potential. Details of deriving the above results can be found in our previous work [5].

With above results on the effects of the δ pulse coupling limit, we now derive the relationships between the excitatory feedforward connection strength G_1, the excitatory lateral connection strength G_2, the delayed feedforward inhibition

connection strength G_3, and the feedback inhibition strength G_4. For simplicity, we neglect the delays of the feedback inhibition, and assume that the lateral excitation and feedback inhibition are simultaneous. The delay of the feedforward inhibition is assumed to be a constant value Δ. We require the minimum T_{min} of the intervals of the input spikes to be larger than Δ.

A neuron is in the down state if its membrane potential is less than a critical value V_c; it is in the up state otherwise. We assume the following ordering of the voltages:

$$E_I < L < V_c < \Theta < 0. \qquad (B7)$$

To ensure that the neuron spikes only in the up state, we must have the following relation for G_1:

$$\Theta = V_c e^{-G_1}. \qquad (B8)$$

Since $L < V_c < \Theta$, we have

$$G_1 < \ln\left(\frac{L}{\Theta}\right). \qquad (B9)$$

The delayed feedforward inhibition G_3 should be large enough to accomplish two tasks: bring the neuron to the down state when it is excited from the down state by the feedforward excitation; bring the neuron to the down state from the up state when the input sequence is out of order. In other words, the neuron should return from any state to the down state upon receiving the feedforward inhibition. A sufficient lower bound is found by requiring the membrane potential to go below V_c from Θ, since this is the most demanding case for the action of the feedforward inhibition. This leads to the following inequality for G_3:

$$E_I(1 - e^{-G_3}) + \Theta e^{-G_3} < V_c. \qquad (B10)$$

It is easy to see that a sufficiently large G_3 should satisfy the above equation.

When a neuron in the chain spikes, the neuron immediately next to it down the synfire chain receives both lateral excitation and feedback inhibition. The combined effect of the excitation and the inhibition is such that the membrane potential of the postsynaptic neuron should be in the up state without going over the threshold. One necessary condition is that the effective reversal potential of the combined excitation and inhibition should be larger than V_c but smaller than Θ. This is given by

$$V_c < \frac{G_4 E_I}{G_2 + G_4} < \Theta. \qquad (B11)$$

Another requirement is that the membrane potential should jump beyond V_c from the lowest possible initial value, and remain there for at least the minimum T_{min} of the time intervals between the input spikes. Since the membrane potential can be no lower than E_I, the inhibitory reversal potential [because of Eq. (B7)], it is sufficient to require that the up state can be achieved and sustained for T_{min} from a membrane potential at E_I:

$$V_d \equiv \frac{G_4 E_I}{G_2 + G_4}(1 - e^{-G_2 - G_4}) + E_I e^{-G_2 - G_4} > V_c e^{T_{min}/\tau}$$
$$- L(e^{T_{min}/\tau} - 1). \qquad (B12)$$

Since $V_d < \Theta$, the above inequality leads to a lower limit on G_1:

$$G_1 > \ln\left(\frac{L}{\Theta} + \frac{\Theta - L}{\Theta} e^{-T_{min}/\tau}\right). \qquad (B13)$$

Together with Eq. (B9), we have the upper and lower bounds for G_1. Equation (B11) requires that the ratio between G_2 and G_4 should be in a bounded range. It is easy to see that with a ratio of G_2 and G_4 such that the effective reversal potential is close to Θ, a sufficiently large sum of G_2 and G_4 guarantees that Eq. (B12) can be satisfied. Therefore, solutions to Eqs. (B11) and (B12) can always be found. Since the effective reversal potential of the excitation and inhibition is less than the threshold, repeated firing of N1 will not cause N2 to spike.

If a neuron other than N2 is in the up state, spiking of N1 should bring the neuron back to the down state through the feedback inhibition. The feedback inhibition should also be strong enough to ensure the neuron just spiked to return to the down state upon receiving the feedback inhibition. All these requirements can be satisfied if G_4 is large enough to bring the membrane potential down below V_c from Θ:

$$E_I(1 - e^{-G_4}) + \Theta e^{-G_4} < V_c. \qquad (B14)$$

The above equation can be satisfied as long as G_4 is large enough.

From the up state, the membrane potential decays before the next input spike can come in. If the interval between the input spikes is too large, the neuron can decay into the down state before the arrival of the next input. This will lead to no response even if the input sequence is correct. Therefore, there is a maximum value T_{max} for the allowed time intervals of the input spikes for correct operation of the network. The sufficient upper limit of T_{max} is derived by requiring the neuron starting from E_I to stay in the up state by the time T_{max} after receiving the lateral excitation and the feedback inhibition. This is expressed in the following inequality:

$$T_{max} < \tau \ln\left(\frac{L - V_d}{L - V_c}\right). \qquad (B15)$$

Here V_d is the membrane potential of the neuron after receiving the lateral excitation and the feedback inhibition starting from E_I, and is defined in Eq. (B12).

A final condition is for G_0, the feedforward excitation strength to N1, which should spike following every input spike. This is achieved by requiring that N1 can spike even from the lowest possible membrane potential:

$$E_I e^{-G_0} > \Theta. \qquad (B16)$$

As long as all above equations are satisfied, the network will perform the sequence recognition task as proposed.

These bounds are sufficient but not necessary. Tighter bounds can be derived with more detailed analysis.

2. The case of finite synaptic time constants

With finite synaptic time constants, we cannot write down the explicit analytical conditions for the parameters. Nevertheless, we can prescribe numerical procedures for finding the bounds of the parameters much in the same way as in the δ pulse coupling case. The main impact of the finite synaptic times is the finite rise time of the membrane potentials and the temporal summation effects of the conductance. We no longer assume that the lateral excitation and the feedback inhibition arrive at the postsynaptic neuron at the same time. A delayed feedback inhibition imposes an upper bound for the strength G_2 of the lateral excitation, since the postsynaptic neuron should not spike before the arrival of the inhibition. This upper limit on G_2 imposes an upper limit on the strength G_4 of the feedback inhibition, since too large inhibition will not be able to leave the neuron at the up state given that the excitation strength is upper bounded. As we have seen in the δ pulse coupling case, there is a lower limit for G_4 since it needs to be large enough to return the neuron from the up states to the down states when N1 spikes or return the neuron to the down states after spiked. Therefore, the parameter regime for G_4 will be limited. There are also upper limits for G_3 and G_4 coming from the constraints imposed from the temporal summations of the conductance. Too large G_3 and G_4 will cause large accumulation of the inhibitory conductance during the operation, which can suppress the neurons from ever going to the up states.

As in the δ pulse coupling case, we find the sufficient bounds for the parameters by considering the extreme conditions of the membrane potentials and the synaptic conductance when neurons make transitions between the down and the up states. With this approach, some parameter regimes will be missed; however, the derivations of the conditions are simple. A number of parameters are useful to define. We define the lower and upper limits of the delay of the feedforward inhibition as Δ_1 and Δ_2, and those of the feedback inhibition as δ_1 and δ_2. With a finite excitatory synaptic time constant and a finite membrane time constant, it takes a finite time for the excitatory postsynaptic potential to reach the peak. We denote the upper limit of this time to peak as λ. We assume that these delays satisfy the following inequality:

$$\lambda + \delta_2 < \Delta_1. \tag{B17}$$

In other words, the minimum of the delay of the feedforward inhibition must be larger than the maximum delay of the feedback inhibition. This makes it possible for interneuron I1 to prevent interneuron I2 from spiking. We also assume the following relationship between the synaptic time constants:

$$\tau_E \lesssim \tau_I. \tag{B18}$$

This condition simplifies some of the arguments in the derivations of the bounds. As before, we denote the minimum time between the input spikes as T_{min}. We must have a relationship

$$\Delta_2 < T_{min}. \tag{B19}$$

In practice, we find that making $T_{min} > \Delta_2 + 2\tau_I$ is useful for getting solutions with the procedures we derive below. A T_{min} too close to Δ_2 tends to limit the solution space.

We start with the definition of the down state. A neuron is in the down state if its membrane potential is less than V_c, the critical membrane potential, and the net synaptic current on the neuron is negative, i.e.,

$$V < V_c, \tag{B20}$$

$$I_s(V, g_E, g_I) \equiv -g_E V - g_I(V - E_I) < 0. \tag{B21}$$

Here I_s is the synaptic current of the neuron. Because the synaptic time constant of the inhibition is longer than that of the excitation, a neuron in the down state at one time will remain in the down state without any spike inputs. The value of V_c is randomly selected from the range (L, Θ).

The amount of the feedforward excitatory conductance G_1 is such that a neuron receiving the input spike should not spike from the down state before the delayed inhibition can arrive. To calculate this, we set the initial condition at the most depolarized down state, apply the excitatory spike input, and integrate for the maximum delay of the feedforward inhibition. The membrane potential should stay subthreshold during this integration. We set the maximum of the feedforward excitatory conductance that satisfies this condition as G_1. The most depolarized down state is the one with membrane potential at the critical value, and the residual synaptic conductance equal to zero. The following is the procedure for calculating G_1.

Procedure I: calculate G_1.
Initial conditions:

$$V = V_c,$$

$$g_E = G_1,$$

$$g_I = 0.$$

Integration time:

$$t = (0, \Delta_2).$$

Condition:

$$V(t) < \Theta.$$

Iteration:
Decrease G_1 until the condition is met.

With G_1 determined, we calculate a lower limit of the strength G_3 of the feedforward inhibition. The inhibition should be strong enough to ensure a neuron in the down state to remain in the down state before the arrival of the next input spike. To get this lower limit, we consider the extreme condition of the neuron that requires a large inhibition. This condition is realized for the neuron receiving the feedforward excitation from the most depolarized down state. Here, the membrane potential can be at most near the threshold, and the excitatory conductance is at most $G_1 e^{-\Delta_1/\tau_E}$. The least

possible time for the inhibition to do its job is the minimum possible time span between the feedforward inhibition and the next input spike. So here is the procedure for finding a lower limit of G_3.

Procedure II: calculate a lower limit of G_3.

Initial conditions:

$$V = \Theta,$$

$$g_E = G_1 e^{-\Delta_1/\tau_E},$$

$$g_I = G_3.$$

Integration time:

$$t = (0, T_{min} - \Delta_2).$$

Condition:

$$V(t) < \Theta,$$

$$V(T_{min} - \Delta_2) < V_c,$$

$$I_s(T_{min} - \Delta_2) < 0.$$

Iteration:

Increase G_3 until the condition is met.

Here, $I_s(T_{min} - \Delta_2)$ represents the residual synaptic current at the end of the integration.

We now consider the transition from the down state to the up state due to the lateral excitation and the feedback inhibition. This transition imposes an upper limit on the lateral excitation strength G_2 and a lower limit on the feedback inhibition strength G_4. These limits come from the requirement that the postsynaptic neuron does not spike because of the lateral excitation. The condition that imposes the strongest constraints on these limits is the case of N1 repeatedly spiking with the smallest possible time intervals. This happens when the input sequence is 1111 . . . . N2 should not spike under these repeated lateral excitations, even starting from the most depolarized down state. We find the limits in two steps. First, we find an upper limit of G_2 by requiring that N2 does not spike from the most depolarized down state before the arrival of the feedback inhibition.

Procedure III: calculate an upper limit of G_2.

Initial conditions:

$$V = V_c,$$

$$g_E = G_2,$$

$$g_I = 0.$$

Integration time:

$$t = (0, \delta_2).$$

Condition:

$$V(t) < \Theta.$$

Iteration:

Decrease G_2 until the condition is met.

Once we find the upper limit of G_2, we randomly set G_2 from 0 to this maximum allowed value. We then decide the lower limit of G_4. The feedback inhibition should be strong enough to keep N2 from spiking. This is done in the following procedure that contains a two-step loop.

Procedure IV: calculate a lower limit of G_4.

Initial conditions:

$$V = V_c,$$

$$g_E = 0,$$

$$g_I = 0.$$

Integration:

Loop
Step 1: $g_E \to g_E + G_2$. Time: $t_1 = (0, \delta_2)$.
Step 2: $g_I \to g_I + G_4$. Time: $t_2 = (0, T_{min} - \delta_2 - \lambda)$.
Condition:

$$V(t_1) < \Theta, V(t_2) < \Theta.$$

Iteration:

Increase G_4 until the condition is met.

The loop is stopped if the steps converge to a periodic orbit. The neuron state at the end of step 2 after the loop converges is the most depolarized up state at the times of the spike inputs. This state will be useful for calculations of other limits. We denote the membrane potential at this state as V_1, the residual excitatory conductance as g_{E1}, and the residual inhibitory conductance as g_{I1}.

Neurons in the up states must return to the down states if the input spikes are out of order and the delayed feedforward inhibition arrives. This gives another lower limit of G_3. To calculate this, we start with the most depolarized up state. The integration has two steps. First, the neuron state decays from the most depolarized up state before the arrival of the delayed inhibition. Second, the feedforward inhibition arrives, and returns the neuron to the down state before the next input comes. The procedure is as follows.

Procedure V: calculate a lower limit of G_3.

Initial conditions:

$$V = V_1,$$

$$g_E = g_{E1},$$

$$g_I = g_{I1}.$$

Integration:

Step 1: Time: $t_1 = (0, \Delta_1)$.
Step 2: $g_I \to g_I + G_3$. Time: $t_2 = (0, T_{min} - \Delta_2)$.
Condition:

$$V(t_2 = T_{min} - \Delta_2) < V_c,$$

$$I_s(t_2 = T_{min} - \Delta_2) < 0.$$

Iteration:

Increase G_3 until the condition is met.

A neuron in the up state should return to the down state if it is not N2 and the next input is to N1. In this case, the transition is due to the feedback inhibition induced by the spiking of N1. This gives a lower limit on the strength G_4 of the feedback inhibition. Since the neuron in the up state is not N2, it must arrive at the up state from a down state. Therefore, the most demanding situation for the feedback inhibition is that the neuron arrived at the up state from the most depolarized down state. So here is the procedure for calculating this lower limit.

Procedure VI: calculate a lower limit of G_4.

Initial conditions:

$$V = V_c,$$

$$g_E = 0,$$

$$g_I = 0.$$

Integration:
Step 1: $g_E \rightarrow g_E + G_2$.
Time: $t_1 = (0, \delta_2)$.
Step 2: $g_I \rightarrow g_I + G_4$.
Time: $t_2 = (0, T_{min} - \lambda - \delta_2 + \delta_1)$.
Step 3: $g_I \rightarrow g_I + G_4$.
Time: $t_3 = (0, T_{min} - \lambda - \delta_2)$.

Condition:

$$V(t_3 = T_{min} - \lambda - \delta_2) < V_c,$$

$$I_s(t_3 = T_{min} - \lambda - \delta_2) < 0.$$

Iteration:
Increase G_4 until the condition is met.

There is yet another lower limit of G_4. After an excitatory neuron spikes, it must return to the down state before the arrival of the next spike input. This is done through the combined effects of the reset of the membrane potential, the refractory time period, and the feedback inhibition. The most demanding situation for the inhibition is for the case of N2. Repeated spiking of N1 can cause N2 to be in the most depolarized up state. The residuals of the synaptic conductance from this up state work against the action of the feedback inhibition after N2 spikes. A long refractory period helps, since the residuals decay during the refractory period. We assume that the refractory time period is longer than the maximum delay of the feedback inhibition. A large reset of the membrane potential also helps to reduce the burden of the feedback inhibition. The following is the procedure to calculate the limit.

Procedure VII: calculate a lower limit of G_4.

Initial conditions:

$$V = R,$$

$$g_E = G_1 e^{-\tau_R/\tau_E} + \frac{G_2 e^{-(T_{min} - \lambda + \tau_R)/\tau_E}}{1 - e^{-T_{min}/\tau_E}},$$

$$g_I = G_4 e^{-(\tau_R - \delta_1)/\tau_I} + \frac{G_4 e^{-(T_{min} - \lambda - \delta_2 + \tau_R)/\tau_E}}{1 - e^{-T_{min}/\tau_I}}.$$

Integration time:

$$t = (0, T_{min} - \lambda - \tau_R).$$

Condition:

$$V(T_{min} - \lambda - \tau_R) < V_c,$$

$$I_s(T_{min} - \lambda - \tau_R) < 0.$$

Iteration:
Increase G_4 until the condition is met.

We need one more condition for a lower limit of G_4 if we want to extend the network for recognizing sequences with repeated inputs from the same source, for example 1234537, in which input 3 is fed to both N3 and N6. In this case, when N3 spikes because it is in the up state and the input is 3, N6 gets the excitatory input as well (this is not possible if all excitatory neurons get spikes from different input sources). But N6 should return to the down state. This can only be done with the feedback inhibition since the spiking of N3 blocks the delayed feedforward inhibition. We will not discuss this case in detail.

The procedures II, IV, V, VI, and VII give the lower limit of G_3 and G_4 given G_1 and G_2. There are also upper limits of G_3 and G_4. The upper limits come from the requirement that the excitatory neurons can transit from the down state to the up state; furthermore, the neuron can spike from the up state if it receives a spike input. There is also an upper limit for the time intervals between the input spikes, since a neuron cannot stay in the up state indefinitely due to the decaying of the membrane potential and the synaptic conductance. Large inhibitory conductance tends to have adversary effects on this requirement, since the residuals of the inhibition conductance can be too large for the excitation to overcome. The most demanding condition for an excitatory neuron to go to the up state is when it is in the most hyperpolarized down state. Here the membrane potential is at the most possible negative value, which is the reversal potential of the inhibition. The maximum possible inhibitory residuals can also be calculated from the case of repeated feedforward inhibition or feedback inhibition with minimal time intervals of the input spikes. From this most hyperpolarized state, we calculate the effect of the lateral excitation and feedback inhibition and make sure that the neuron is in the up state. We then check if a feedforward excitation coming with input interval T can cause the neuron to spike. This procedure enables us to get upper limits of the feedforward inhibition, the feedback inhibition, and the time intervals of the spike inputs.

Procedure VIII: calculate upper limits of G_3, G_4, T.

Initial conditions:

$$V = E_I,$$

$$g_E = 0,$$

$$g_I = \frac{\max(G_3, G_4)e^{-(T_{min} - \Delta_2)/\tau_I}}{1 - e^{-T_{min}/\tau_I}}.$$

Integration:

Step 1: $g_E \to g_E + G_2$. Time: $t_1 = (0, \delta_2)$.

Step 2: $g_I \to g_I + G_4$. Time: $t_2 = (0, T - \delta_1)$.

Step 3: $g_E \to g_E + G_1$. Time: $t_3 = (0, \Delta_1)$.

Condition:

$\max[V(t_3)] > \Theta$, i.e., the neuron spikes during step 3.

Iteration:

Decrease G_3, G_4, and/or increase T until the condition is met.

For given G_1 and G_2, this procedure may produce upper limits of G_3 or G_4 that are not larger than the lower limits produced in the previous procedures, or the upper limit of T that is not larger than T_{min}. This means that we failed to find that the parameter regime for the network to operate as proposed. We need to select new G_1 and G_2, and start over, until we find the parameters satisfy all the limits required from the procedures.

[1] L.O. Trussell, Annu. Rev. Physiol. **61**, 477 (1999).

[2] M.J. Berry, D.K. Warland, and M. Meister, Proc. Natl. Acad. Sci. U.S.A. **94**, 5411 (1997).

[3] M. Stopfer and G. Laurent, Nature (London) **402**, 664 (1999).

[4] W. Bialek, F. Rieke, R.R. de Ruyter van Steveninck, and D. Warland, Science **252**, 1854 (1991).

[5] D.Z. Jin, Phys. Rev. Lett. **89**, 208102 (2002).

[6] G. Laurent, Science **286**, 723 (1999).

[7] M. Abeles, *Corticonics* (Cambridge University Press, Cambridge, U.K., 1991).

[8] M. Sipser, *Introduction to the Theory of Computation* (PWS, Boston, 1997).

[9] D. Jurafsky and J.H. Martin, *Speech and Language Processing* (Prentice-Hall, New Jersey, 2000).

[10] R.C. Carrasco, M.L. Forcada, M.A. Valdes-Munoz, and R.P. Neco, Neural Comput. **12**, 2129 (2000).

[11] J.R. Gibson, M. Beierlein, and B.W. Connors, Nature (London) **402**, 75 (1999).

[12] A. Gupta, Y. Wang, and H. Markram, Science **287**, 273 (2000).

[13] M. Treisman, Psychol. Monogr. **77**, 1 (1963).

[14] V. Braitenberg, Prog. Brain Res. **25**, 334 (1967).

[15] D.W. Tank and J.J. Hopfield, Proc. Natl. Acad. Sci. U.S.A. **84**, 1896 (1987).

[16] E. Ahissar, S. Haidarliu, and M. Zacksenhouse, Proc. Natl. Acad. Sci. U.S.A. **94**, 11633 (1997).

[17] D.V. Buonomano and M.M. Merzenich, Science **267**, 1028 (1995).

[18] J.J. Hopfield and C.D. Brody, Proc. Natl. Acad. Sci. U.S.A. **98**, 1282 (2001).

[19] T. J. Lewis and W. Gerstner, in *Spiking Neuron Models: Single Neurons, Populations, Plasticity* (Cambridge University Press, Cambridge, UK, 2002).

[20] A. Erisir, D. Lau, B. Rudy, and C.S. Leonard, J. Neurophysiol. **82**, 2476 (1999).

[21] D. Hansel and G. Mato, Neural Comput. **15**, 1 (2003).

[22] X.J. Wang and G. Buzsaki, J. Neurosci. **16**, 6402 (1996).

[23] R.C. deCharms, D.T. Blake, and M.M. Merzenich, Science **280**, 1439 (1998).

[24] J.P. Rauschecker, B. Tian, and M. Hauser, Science **268**, 111 (1995).

[25] M.S. Lewicki and B.J. Arthur, J. Neurosci. **16**, 6987 (1996).

[26] S. Shimegi, T. Akasaki, T. Ichikawa, and H. Sato, J. Neurosci. **20**, 6241 (2000).

[27] D. Hansel and G. Mato, Phys. Rev. Lett. **86**, 4175 (2001).