Neural Synchrony Through Controlled Tracking

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Abstract

We present a model for generating a kind of neural synchrony in which the individual spike trains of one neuron or group of neurons closely match the spike trains of another. This kind of neural synchrony has been observed in animals performing auditory, visual and attentional information processing tasks. Our model is realized in a system of functionally identical, refractory spiking neurons. Larger systems with more sophisticated information processing capabilities can be constructed from aggregated instances of the basic network.

Introduction

Recently researchers ranging from neurobiologists (e.g. Ritz & Sejnowski, 1997) to computer scientists (e.g. Shastri, 1999) to psychologists (e.g. Hummel & Holyoak, 1997; Sougné, 2000) have studied the thesis that the synchrony of neural activity is one means by which bits of information are aggregated into the larger wholes necessary for complex information processing. In brief, neural synchrony is thought to implement the 'dynamic binding' of certain representations in the brain. It has also been suggested that neural synchrony is part of the process by which consciousness emerges from distributed brain activity.

But which neural dynamics are actually being synchronized? The answers provided by a decade of intensive research have generally all been variations on three basic themes. The first approach to synchrony intends the term to refer to coherence between 'oscillations' observed in the brain. In these cases it is the aggregated neural activity generated by the whole of a particular neural population that is deemed to be oscillating, not individual cortical neurons. Ritz and Sejnowski (1997) provide an excellent review of the conditions under which this mode of synchrony occurs, as well as its reputed significance in information processing tasks. Two representative examples include the observation of oscillatory synchrony in the brains of cats completing a sensorimotor task (Roelfsema, Engel, König & Singer, 1997), and its generation in an artificial neural network modeling the brain's solution of a figure-ground segregation problem (Sporns, Tononi & Edelman, 1991). Engel et al. have even speculated that oscillatory synchrony may be instrumental in bringing representations to consciousness, based on results showing that this mode of synchrony correlates with perceptual awareness in cats (1999).

Synchrony is understood elsewhere as correlated quasiperiodic activity occurring at a constant phase offset with respect to a persistent background oscillation. Different groups of neurons periodically become active during different phases of the background oscillation; neurons that become coactive during the same subperiods are said to exhibit phase synchrony. Phase synchrony allows the expression of phase-coded representations: representations in which information is coded in the relative timing of quasiperiodic neural activity. Gerstner, Kempter, van Hemmen and Wagner (1999) use empirical evidence for phase-coding's involvement in the sound source localization task in barn owls to build a successful mathematical simulation of the process. As well, Jensen and Lisman argue that data from psychological experiments and rat EEGs support accounts of short term memory capacity (1998) and position reconstruction in rats (2000), respectively, framed in phase-coding terms.

Phase synchrony is also exploited in a number of influential models of cognitive processes. Shastri, in his SHRUTI model of inference and reasoning, uses phase synchrony to bind neurons corresponding to role-filler words; e.g., John, with other neurons corresponding to specific roles in propositions; e.g., X in "X sees Y" (Shastri & Ajjanagadde, 1993; Shastri, 1999). Similarly, phase-coded bindings of roles to role-fillers underlie Hummel and Holyoak's IMM and LISA models of analogy formation (Hummel, Burns & Holyoak, 1994; Hummel & Holyoak, 1997).

Finally, there is a third synchrony phenomenon that we have termed 'spike train synchrony.' This synchrony is present when strong correlations exist between the individual firing times of different neurons or groups of neurons. There is no need that these firing times be quasiperiodic, as in phase synchrony. Nor is it generally possible to explain the aggregate synchronized activity as oscillatory; in fact, the mean overall activity of the neurons involved can remain close to constant except over very short time intervals.

DeCharms and Merzenich (1996) observed the spike train synchrony of neurons in the brains of anesthetized marmoset monkeys responding to a pure tone stimulus. For the duration of each tone, the firing patterns of selected neural regions became correlated, even though their mean firing rates remained unchanged. In addition, this correlation disappeared when the tone ended and was absent before it began. Tightly correlated spike trains have also been reported for neighbouring cells in two early vision regions: the retinal ganglia (Meister, 1996) and the lateral geniculate nucleus (Alonso, Usrey & Reid, 1996). More recently, Steinmetz et al. (2000) have found that certain somatosensory neurons in monkeys increase the correlation of their spike trains when performing visual and tactile tasks requiring increased attention.

All three modes of synchrony outlined here have been empirically observed under conditions which suggest a role for them in specific information processing tasks. Furthermore, previous research, as cited above, has resulted in working models demonstrating how the first two types, oscillatory and phase synchrony, may be generated. The design of computational simulations targeting the generation of synchrony at the level of individual spike trains, however, has received little attention.

Simulation-based research of this type should help to elucidate the structural and functional aspects of the brain necessary for spike train synchrony as it has been observed. Moreover, even in the absence of an artificial system mirroring the exact architecture of the brain, spike train synchrony models should provide insights and help researchers test hypotheses concerning information processing tasks whose realizations in the brain presumably require this form of synchrony. Advances generated by analogous models depending on other synchronies—Shastri's model of logical reasoning, for example—corroborate this claim.

In this paper we present an artificial neural network designed to exhibit spike train synchrony. The neurons in the network are all functionally identical, refractory neurons, and the connections between them are all of the same, standard type.

The controlled tracking network displays a simple behaviour: the *clone neuron* copies or 'tracks' the spike train of the *primary neuron*. The copying process is selective, meaning that it stops and starts in response to signaling from two *actuator neurons*. In this way, the *clone neuron* can be made to fall in and out of synchrony with the *primary neuron*.

The design of the network was completed in two stages. In the first, a basic network was built in which the clone neuron was made to copy the activity of the primary neuron at all times. Pausing or halting the copying is not allowed in this network. These operations were implemented in the second stage of network design, in which the control component of the system was integrated. Finally, in the last section, we highlight the contributions of the model to understanding the generation of biological spike train synchrony and its role in information processing. This includes a discussion of the merits of our model's representational capabilities over those used in other influential modeling approaches.

Mathematical Fundamentals

This section describes the dynamics of the neurons we use to build our spike-tracking networks. In brief, our neurons function like the Spike Response Model neurons developed in Gerstner (1999), with only slight modifications. We review the defining equations of our neurons here, drawing heavily from Gerstner's formulations.

The total membrane potential u_i of each spiking neuron i is given by:

$$u_i(t) = \sum_{t_i \in F_i} \eta(t - t_i) + \sum_{j \in \Gamma_i} \sum_{t_j \in F_j} w_{i,j}\varepsilon_{i,j}(t - t_j). \quad (1)$$

The last term of this equation quantifies the contributions to the membrane potential of neuron i due to excitations and inhibitions from the set Γ_i of neurons with efferent connections to i. Each such neuron j will contribute to i's membrane potential due to post-synaptic potentials ε seen coming from j across the synapse connecting to i. The function $\varepsilon_{i,j}(s)$ equals the excitatory post-synaptic potential seen at post-synaptic neuron i at a time of s seconds after the firing of a pre-synaptic neuron j. The set F_i represents the set of all individual spike times t_i of the neuron i; likewise, F_j and t_j for neuron j. Finally, the constant $w_{i,j}$ represents the strength of the connection from neuron i to neuron j. In summary, the last term of Eq. (1) sums the contributions to membrane potential due to incoming post-synaptic potentials, after scaling these contributions by the appropriate connection weights.

The function $\varepsilon_{i,j}(s)$, introduced above is defined by:

$$\varepsilon_{i,j}(s) = \left[\exp\left(-\frac{s - \Delta_{i,j}}{\tau_m}\right) - \exp\left(-\frac{s - \Delta_{i,j}}{\tau_s}\right)\right] H(s - \Delta_{i,j}), \quad (2)$$

where τ_m and τ_s are time constants determining the shape of the post-synaptic pulse, $\Delta_{i,j}$ is the propagation time of the electric potential signal between the beginning and end of a connection from i to j (also called the axonal delay or 'length') , and H is the Heaviside unit step function:

$$H(t) = \begin{cases} 0 & \text{for } t < 0 \\ 1 & \text{otherwise} \end{cases} \quad (3)$$

The left term on the right hand side of equation (1) accounts for the response of neuron i to its own previous spikes. This term quantifies the *refractoriness* of neurons; i.e., the decreased capacity of a neuron to spike soon after it has just spiked. Refractoriness is modeled as a short-term, decaying, inhibitory signal:

$$\eta(s) = -\eta_0 \exp\left(-\frac{s}{\tau_r}\right) H(s) - K\left[H(s)\right] H(\delta^{abs} - s) \quad (4)$$

The variable s is the time since a previous spike, while the parameters η_o and τ_r scale the amplitude and the decay rate, respectively. $H(s)$ remains the Heaviside unit step function as discussed above. Finally, the constant K represents an arbitrarily large number and the parameter δ^{abs} designates the duration of absolute refractoriness. This represents the length of time after a spike during which a second spike is physically impossible.

A neuron spikes each time that its membrane potential u_i exceeds a threshold value ϑ, provided that u_i is increasing from a subthreshold value on the last discrete time step. Each spike results in a new spike time t_i being added to the set F_i of all spike times for neuron i.

Tracking Network

In this section, we describe a network where one neuron's spike train, the clone neuron, closely duplicates the spike train of another neuron, the primary neuron, at all times. We call this network the *tracking network*. Fig. 1 illustrates the architecture of our tracking network, in which auxiliary neurons serve to enable a clone neuron to synchronize its firing with the primary neuron. The complication of the network, primarily due to the many feedback connections, is necessary in a tracking system composed of refractory neurons. Refractoriness causes a transient desensitization of a neuron to incoming inputs shortly after that neuron's firing. Consequently, a pulse in an input signal will not elicit the same response from a refractory neuron that has just fired as it will from one that has not yet fired. This means that simply connecting the primary neuron to the clone neuron will not produce reliable tracking because the clone neuron will be biased to respond differently to different post-synaptic pulses sent from the primary neuron. The clone neuron will be slower to spike in response to an incoming superthreshold pulse coming a short time after the last one, in comparison to one coming a long time after the last one.

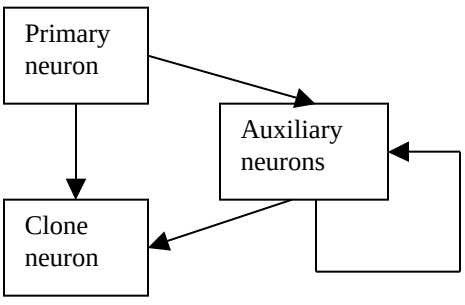

Figure 1. Spike tracking system.

To restore the efficacy of the external input to a refractory neuron, which in our system corresponds to the primary neuron's outgoing post-synaptic pulses, we construct an auxiliary signal that provides a second input to the clone neuron. This signal is implemented using the output pulses of *auxiliary neurons* connected efferently to the clone neuron. These auxiliary neurons are coerced to fire in such a way that the potential pulses they output individually to the clone neuron add up to a negative approximation of the η refractory term of Eq. (1).

Mathematically, each connection from an auxiliary neuron to the clone neuron results in an additional term in the expansion of the double summation of Eq. (1). Therefore, for the set A of auxiliary neurons to successfully mitigate refraction, the mathematical constraint to be satisfied is specified by Eq. (5) in the appendix. The variables $w_{c,a}$ and $\Delta_{c,a}$ represent the auxiliary to clone neuron connection strengths and time delays, respectively, while t_a represents the firing times of the auxiliary neurons. These three sets of variables remain to be specified, in addition to the total number of auxiliary neurons to be used. To determine acceptable values for these unknowns we first perform the following simplifying analysis. The analysis solves the approximation problem for only a limiting case, but we go on to show that straightforward modifications lead to a more general solution.

For now we assume a single, isolated refractory incident corresponding to a single, isolated firing of the clone neuron at time $t_c = 0$. Second, we require that pulses from the auxiliary neurons arrive instantaneously at the membrane of the clone neuron; i.e., we set the connection time delays $\Delta_{c,a}$ to 0. Finally, we tentatively prohibit each auxiliary neuron from firing more than once. These conditions allowed us to reformulate the constraint of Eq. (5) using Eq. (6) in the appendix. In graphical terms, this simplified constraint commits us to build up an approximation to the refractory function η by summing time-shifted and scaled postsynaptic pulses ε. The pulses are vertically scaled according to the connection strengths $w_{c,a}$. Their time shifting is specified by the times of firing t_a of the auxiliary neurons.

For convenience we chose to limit the maximum relative error of our particular approximation to less than 5%. Better approximations are possible, but as error decreases, the number of required auxiliary neurons increases, making the network larger. For our error tolerance of 5% we were able to construct a feasible solution set of values t_a and $w_{c,a}$ using 23 auxiliary neurons.

The set of solution values t_a indicate the time each auxiliary neuron must spike, *relative to the clone neuron*, in order to mitigate refractoriness. To force the neurons to spike at these times, we apply the same external input signal that the clone neuron experiences

to each of the auxiliary neurons, with a time delay of length t_a, the firing offset time for that neuron. In physical terms this means connecting the primary neuron to each auxiliary neuron. The strengths $w_{a,p}$ of these primary to auxiliary connections should be identical to the strength $w_{c,p}$ of the single primary to clone neuron connection. The lengths $\Delta_{a,p}$ of the primary to auxiliary connections, however, must equal the primary to clone neuron connection length *plus* the firing time offset t_a calculated for the particular auxiliary neuron *a* in question.

Returning to the assumptions we made earlier, the system thus connected is only strictly guaranteed to mitigate the clone neuron's refractoriness due to its very first spike. This is because the clone neuron was assumed to fire only once in isolation. To compensate for the refractoriness following *all* spikes, the auxiliary neurons need to spike—and with a precise time lag of t_a—not just the first time that the clone neuron spikes, but *every* time it spikes.

Though all the auxiliary neurons share the same external input as the clone neuron, they will fail to spike as required (i.e., every time the clone neuron spikes) because the clone neuron now has an additional input signal compensating for its internal refractoriness. It is no longer desensitized due to the refractoriness following from its first spike, but the auxiliary neurons still are.

We remedy this problem by feeding an additional input into each of the auxiliary neurons. Each of these additional input signals should be identical to the refractoriness compensation signal for the clone neuron, except that, as before, the signals should arrive with a time lag or delay of t_a specific to the auxiliary neuron *a* in question.

In terms of implementation, this translates into an additional bundle of connections to each auxiliary neuron. Each bundle consists of a set of connections leading from *each* auxiliary neuron to *one* of those auxiliary neurons. Therefore, if there are N auxiliary neurons, N feedback bundles are required, each containing N unique connections. This makes for N^2 auxiliary neuron feedback connections in total.

We should emphasize that we have been ignoring the absolute refractory component of the refractoriness signal corresponding to the second term in Eq. (2). Physiologically the absolute refractoriness of neurons could never be compensated for in biological networks in any event, because it arises from a fundamental electrochemical constraint on the availability of certain molecules (Paul, 1975). Furthermore, because all our neurons are identical, a clone neuron would never be expected to track a spike which would land itself in the absolute refractory period following a previous spike: the primary neuron would not be capable of producing such a spike train.

We tested the capacity of the clone neuron to copy the spike train of the primary neuron, as connected in the tracking network described above. The network was simulated using the SpikeSim program we designed in Java. As input to the tracking system, spike trains were evoked from the primary neuron that corresponded to an exponential distribution of spikes with a mean of 30 time steps between spikes.

The results indicate that missing spikes and bursts of spikes at inappropriate times ('ringing') seem to be the only symptoms of inaccurate tracking. In biological systems such aberrations could be explained by molecular and thermal noise.

A very strong cross correlation over a set of ten trials was observed between the spike trains of the primary and clone neuron. We found that the spike train of the clone neuron slightly lags the spike train of the primary neuron. A non-zero time lag is inevitable because it takes time for the post-synaptic pulses ε to peak. Nevertheless, it should not be of too much concern because the signals can be time-shifted so they coincide as seen from a third neuron's perspective. Setting appropriate connection lengths for the primary to third neuron connection and the clone to third neuron connection will implement the necessary shifting.

Discussion

We have presented a new computational model for synchrony generation, a model that implements controlled spike train tracking of one neuron by another. In this closing discussion, we investigate a possible variation of the model promising greater physiological plausibility. We then move on to compare the merits of this model to others in the field; namely, Shastri's and Hummel's. Finally, we argue that our basic model and its variations will likely prove helpful in the effort to develop larger scale simulations of cognitive processes.

To build the controlled spike tracking system, we constructed connections between the auxiliary neurons and the clone neuron of length (or time delay) zero. The connections between the primary neuron and the clone neuron, however, are variable in length and all non zero, in order to implement time lag delays. As a result of this configuration all the auxiliary neurons and clone neurons end up generating identical but time-shifted spike trains: these neurons fire in a wave-like or 'follow-the-leader' type manner.

Theoretic considerations suggest that an operationally identical network can be implemented in a way that is more physiologically realistic. Instead of implementing the time delays through lengthening the connections from the primary to auxiliary neurons, we can set these constant and stagger the lengths of two sets of connections: those in the feedback bundles, and those between the auxiliary neurons and the clone neuron. This removes the need for problematic zero-length connections across which electrical pulses would presumably need to instantaneously travel, while preserving the critical time-delay dynamics of the

refractoriness compensation signal. In this revised setup, the auxiliary neurons and the clone neuron would all fire identical spike trains, just as before, but with no time lag asynchrony.

We also reason that if auxiliary neurons exist in the brain to mitigate for refraction, they likely play roles in other brain circuits as well. If this is so, their mutually synchronous firing would help to maintain signal or information synchrony in all of these circuits. Such synchrony would not evolve from the original controlled tracking network, but would characterize the auxiliary neurons in the modified variation, as described above. In short, there are several reasons for believing that the proposed network variation should be superior to the original controlled tracking network.

The artificial neural networks whose results were presented above were simulated with the assumption of no noise. In later trials we investigated the effect of introducing noisy dynamics to the Spike Response Neuron model defining the neurons in our network. More specifically, the firing thresholds were made noisy by adding to the membrane threshold function a Gaussian random variable with mean of zero and standard deviation proportional to the degree of desired noisiness. This method of introducing noise is one of the standard ones discussed in Gerstner (1999).

When simulated, noisy neurons resulted in severely poor tracking performance, even when the noisiness was kept low. The largest errors in tracking were bursts of extraneous spikes occurring after a single premature firing. This initial firing was in turn due to a transient lowering of the membrane threshold of a single neuron when that neuron's membrane potential was close to the average threshold.

We expect that poor tracking under noise conditions can be mostly eliminated by a redesign of the feedback connections between auxiliary neurons, one which would not modify their basic role in the network. Our ideas are still only in an early stage of development, however. For now we have to concede that our system's spike tracking performance is poor under noisy conditions.

Future research could investigate the implications of the unique information encoding and processing properties our system possesses by nature of its design. For instance, the system allows for more versatile synchronic neural coding than that available in networks used in Shastri's SHRUTI model of logical reasoning or Hummel's LISA or IMM models of analogy formation. In these systems individual neurons are active for a maximum of one portion or 'phase' of each background oscillatory cycle, during which they may fire either alone or more generally in synchrony with a group of other neurons that are also active only during that phase. During other periods of the background oscillatory cycle all these neurons are inactive.

The neurons in the controlled spike-tracking system presented here can be made to synchronize in this way, but they are capable of more sophisticated synchronic dynamics as well, as the simulation results demonstrated. Through the control interface, variable length periods of transient synchrony were elicited from our system on demand. Furthermore, as will be demonstrated below, the clone neuron can be made to regularly switch between synchrony with different groups of neurons. In contrast, the other three models require a neuron to synchronize with only one group of neurons and only then during short, periodic time windows of constant width.

Another significant difference lies in the firing patterns during periods of synchrony. In the SHRUTI, LISA and IMM-based networks, the firing of a neuron within its interval of synchrony is only described in terms of its overall firing rate or 'level of activity.' But in controlled spike tracking systems, significant *subcoding* can take place within time intervals of synchronized activity. By subcoding we mean that additional information can be stored in the relative timing of spikes within each period or instance of neural synchrony. For the other models noted, supporting subcoding within periods of synchrony would effectively require system redesign. Consequently, we claim that our tracking system allows for more detailed elaboration of representations during synchronous neural firing.

Finally, we suggest that sophisticated neural networks for simulating higher level cognitive processes could be designed using the controlled tracking system as a reusable component, a repeated building block. Such networks would broaden our understanding of the processes they model, just as SHRUTI and LISA have provided insights into the reasoning and analogy-forming processes they were built to simulate.

Imagine a network with six inputs. Three of these are generated by three primary neurons, each of which we assume signals a separate and unique stream of representations coded within its spike train. The other three inputs are provided by three *switch neurons*, each corresponding to one of the three primary neurons. The network takes advantage of these inputs, and four spike tracking subsystems embedded within it, to implement a relatively complex behaviour: on receiving a single spike from one of the three switch neurons, the network begins to track the spike train of the corresponding primary neuron. When a different switch neuron spikes, the network switches inputs and starts tracking a different primary neuron. In effect, this network implements the ability to reorganize arbitrary parts of neural signals, corresponding to different temporally coded representations, in sophisticated ways. Such reorganization or 'splicing' mechanisms could prove quite useful for a number of information processing tasks. Compressions of

sequences of elaborate representations into more succinct streams could be one such task.

This network demonstrates the potential increase in information processing sophistication that emerges from constructing larger networks within which simple spike tracking subsystems work in coordination. We hope that the application of this kind of design approach will contribute to the future development of neural network models that carry out information processing tasks similar in complexity to those that the brain performs. Moreover, by shedding light on the internal mechanics of the processes involved, such models might not only demonstrate or mimic *what* the brain does but also help to further clarify just *how* it does it.

Acknowledgments

We thank Brandon Wagar and Sid Fingerote for helpful discussions. This work was funded by NSERC.

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Appendix

Equation (5):

$$\sum_{t_c}\sum_{F_c}\eta(t-t_c) \approx -\sum_{a}\sum_{\Gamma_A}\sum_{t_a}\sum_{F_a} w_{c,a}\varepsilon_{c,a}(t-t_a)$$

$$= -\sum_{a}\sum_{\Gamma_A}\sum_{t_a}\sum_{F_a} w_{c,a}\left[\exp\left(-\frac{t-t_a-\Delta_{c,a}}{\tau_m}\right)-\exp\left(-\frac{t-t_a-\Delta_{c,a}}{\tau_s}\right)\right]H(t-t_a-\Delta_{c,a})$$

Equation (6):

$$\eta(t) \approx -\sum_{a}\sum_{\Gamma_A} w_{c,a}\left[\exp\left(-\frac{t-t_a}{\tau_m}\right)-\exp\left(-\frac{t-t_a}{\tau_s}\right)\right]H(t-t_a)$$