Recognition of Temporal Event Sequences by a Network of Cortical Neurons

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Abstract

Recognition of ordered sequences of temporal events is central to many perceptual recognition tasks, from speech detection to analysis of biological motion. We describe a simple cortical network capable of recognizing event sequences through a process of encoding followed by detection. The network is composed of regular spiking and fast spiking neurons, with minimal connectivity. Ordered sequences of inputs occurring over tens-to-hundreds of milliseconds, are time-compressed by the network into tightly clustered spike outputs occurring over a few milliseconds. We investigate the ability of the network to accurately encode the input pattern, in the presence or absence of noise. We show that information about relative input timings are preserved in the output interspike intervals.

Keywords: Visual cortex, Biological motion detection, Spike-based computation

Introduction

Many instances of perceptual recognition involve the detection of an ordered sequence of events occurring on a time scale of tens-to-hundreds of milliseconds. Individual phonemes in spoken speech, or vocalizations in bird song, must occur in the correct order, and within certain time constraints to produce a recognizable word (or song fragment). We are particularly interested in the visual events that characterize biological motions, such as walking. Each walking cycle (~1 Hz) consists of an ordered sequence of events—starts and stops of motions of various limbs in various directions [1]. Part of what defines "walking" and distinguishes it from similar motions (running, skipping, etc.) is this spatiotemporal pattern of events.

The time scale over which these perceptual events occur poses a challenge for any cortical recognition mechanism. Information must be accumulated over tens or hundreds of milliseconds—enough time to observe several "events" (under some experimental conditions, psychophysical experiments show recognition of point-light walkers in 100-200 ms [2]). But cell time constants are on the order of 10 ms. Any mechanism must also be, to some degree, time-scale invariant [3] (to allow recognition of faster or slower walking), and stride-length invariant.

We describe simulations of a small network of biophysically-realistic cortical neurons that is capable of detecting the occurrence of the correct order of events.

Model Description

The network is composed of four regular spiking (RS) cortical neurons and four fast spiking (FS) interneurons. The cell models were taken from Destexhe, as posted on the SenseLab database [4]. Both cells are single compartment models expressing I_{Na} and $I_{K(DR)}$ currents, and additionally in the RS cell, $I_{K(M)}$. The RS cell has the membrane area 29,000 μm^2, g_L=0.1 mS/cm^2, E_L=-70 mV, g_{Na}=50 mS/cm^2, g_K=5 mS/cm^2, g_M=0.07 mS/cm^2. For the IN cell membrane area is 14,000 μm^2, g_L=0.15 mS/cm^2, E_L=-70 mV, g_{Na}=50 mS/cm^2, g_K=10 mS/cm^2. AMPA and GABA synapses were taken from [5], where g_{AMPA}=0.03 μS with τ_{AMPA}=5.26 ms and g_{GABA}=0.05 μS with τ_{GABA}=5.55 ms. For

background noise in RS and IN cells we use Poisson generated 50 Hz AMPA and GABA bombardment with conductance adjusted to produce a desired standard deviation of resting membrane potential. All simulations were carried out in Neuron [6] with time step of 25 μs.

As shown in Figure 1, each RS cell excites an associated FS cell, which in turn inhibits all of the RS cells. There are no connections between RS cells or between FS cells. In other simulations, not described here, we investigated the role of these additional intracortical connection classes.

Each RS cell receives an input, delivered as an injection current. Each input represents the output of cortical centers responding to a particular "event", and is simulated by a linearly increasing current ramp. In all simulations described here, the ramp increased from 2.1-2.5 nA over 0.6 second. Although this is depolarizing current, it could reflect the net difference between total excitatory and inhibitory inputs to the RS cell (see discussion for possible mechanisms). Each RS cell receives a separate input injection, which begins at a certain time and continues for 0.6 second. These conditions result in a difference between inputs ranging from 0.013 to 0.12 nA. We investigated the response of the network to input streams in which the cells were activated in various orders, in which the time delay between onset times was varied, and to which noise was added.

Results

Simulations reveal that the network transforms these ordered, continuous input streams into clusters of tightly ordered spikes. Figure 2 shows the output of the 4 RS and 4 FS cells in response to the input sequence 1-2-3-4. In response, each RS cell fires periodically at roughly 80 Hz (this rate depends on current injection amplitude), however, the spikes of all 4 RS cells are tightly clustered. Inspection of the spike order shows that the order of RS cell firing exactly corresponds to the order of the inputs (1-2-3-4). (See the inset in Figure 2). Note that without the tight phase-locking, it would be difficult to distinguish spikes in one cluster from those in the next.

Closer inspection of the relative interspike intervals between RS cell spikes shows a rough, quantitative preservation of the relative onset times of the inputs. Figure 3 shows results of 10 simulations in which the onset times of each input were varied over a range of 80 ms. Input 1 commenced at a randomly chosen time between 410-490 ms (after start of the simulation), input 2 commenced between 510-590 ms, input 3 between 610-690, and input 4 between 710-790 ms (Figure 4A). Thus, the order of the inputs was constant (1-2-3-4) but the onset times varied significantly. Figure 3 shows histograms that compare the relative intervals of the inputs to the relative interspike intervals. The abscissa plots the reliability ratio calculated as shown in Table 1. A value of 100 would indicate perfect preservation of the time of event information. Values for these simulations ranged between 80-110%, indicating a substantial amount of quantitative information is preserved.

One would expect these results to be affected by the presence of noise. To test this assumption, we added a background noise to RS and FS cells with parameters indicated in methods. The distribution of reliability ratio is asymmetric as is shown at Figure 4C. Therefore to compare the results for different noise levels, we calculate the percentage of the reliability ratios that have values in an interval (50, 150)%. We choose this range, because we think that it represents a reasonable window of accuracy of the circuit. 50% stands for relative spike timing of RS cells two times shorter than input intervals and

respectively 150% stands for two times longer. We tested three noise levels that resulted in a standard deviation of the resting membrane potential of RS and FS cells σ=0.5, 0.7 and1.0 mV. Results presented in Table 2 indicate that the circuit is able to maintain reliable information about structure of the inputs in the presence of noise with standard deviation up to 0.5 mV.

Next we repeat the simulation without a background noise, but with input distorted by adding values generated from normal distribution with standard deviation 0.01 nA every 25 ms. This results in 68.25% and 69.64% of the inputs, 1-2-3 and 2-3-4 respectively, having a reliability ratio between (50,150)%.

Since the 4 RS cells receive significantly different levels of current injection, one might expect them to fire at different rates. The reason they fire synchronously (but with a critical few millisecond delay) at the same rate is due to inhibition from the FS interneurons. Elimination of inhibition (as might occur with bicuculline), releases each RS cell to fire at a significantly different rate, in a non-correlated manner. We analyze the Phase Response Curve (PRC) [7] for one RS cell (Figure 5). Since all RS cells receive identical, simultaneous IPSPs, the effect of inhibition is common to all RS cells. When RS cells fire at different times, they receive the common inhibition at different phases of their ISI, therefore the effect upon ISI is different. The earlier a cell fires the less effect inhibition has upon its ISI. This allows the network to find a stable state, where the effect of inhibition upon each RS cell balances out differences in inputs (Figure 5, arrows 1,2,3,4). Instantaneous frequencies of all four RS cells are equal at each time point, but during the course of the simulation changes. At the beginning it drops from about 95 Hz to 75 Hz because of the adaptation and after 250 ms it starts to rise up to 95 Hz due to increasing input ramp.

Discussion

We have shown that a profoundly simple network architecture (2 cell types, one class of cell-cell connection) can translate an ordered sequence of inputs into an ordered sequence of cell firings, preserving (to some degree) the quantitative ratio of input timings. The advantage of such an analog-to-digital type conversion is the time compression. Events which transpired over tens or hundreds of milliseconds are now represented in a set of inter-spike intervals occurring over a few milliseconds. This renders the problem of recognizing the sequence much more tractable for a cortical neuron (or network).

If this network generates an *encoding* of the event sequence, recognition still requires *detection* of the correct encoded sequence. We would propose that the detection process could involve coupling a specific set of transmission delays to a coincidence detection mechanism. For example, to detect the spike sequence 1-2-3-4, each spike would be delayed by 2 ms less than the previous spike, and then a single cell would be capable of detecting the temporal coincidence of the incoming spikes. One mechanism for generating such time delays could be axonal delays, however other mechanisms, including passive or active dendritic delays are conceivable. If the inputs to the coincidence detector simultaneously activate feedforward inhibition of the detector, then any set of (non-coincident) inputs that doesn't quickly activate the detector will evoke inhibition that quells the process. Thus, only the proper event sequence will be detected.

The model proposed here shares some features developed by previous investigators. Hopfield and Brody [3] identified the problem of recognizing event sequences (in speech patterns, etc.) and their model similarly initiates a decay (or

accumulation) process with each event. Our mechanism differs in that it compresses the event timings into narrowly clustered spike times. Lisman and Idiart [8], in their CA3 model of short-term memory, developed the idea of time compressing spike representations. However, their mechanism requires an underlying voltage oscillation, and focuses more on stable repetition of the spike pattern rather than the input encoding process. A related model was suggested by Mehta et al. [9]

The major assumption of this model is existence of a steadily increasing current injection, initiated by each event. Such a process might reflect a mechanism at either the cellular or network level. For example, a controlled increase in the fraction of cells activated in a local population, or a net shift in the excitation versus inhibition due to differential facilitation and/or depression of synaptic inputs. At the cellular level, possible mechanisms might include voltage-dependent (e.g., NMDA) or Ca^{++}-dependent synaptic inputs, or current due to persistent Na^{+} channels or metabotropic receptors.

An advantage of the proposed model is that by separating the recognition process into encoding and detection stages, a single circuit can be used to encode all input patterns, and separate cells/circuits are only required for the detection phase. Given the brief (~2 ms) spike intervals, which as axonal conduction delays would correspond to a distance of ~200-600 μm of transmission along an unmyelinated cortical fiber) both processes of encoding and recognition could easily occur within the spatial domain of a cortical hypercolumn.

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Figure 1. Each RS cell excites its associated FS cell, which in turn inhibits all of the RS cells. Inputs are numbered 1-4 for reference.

Figure 2. Spike output of the 4 RS cells (bottom) and 4 FS cells (top) in response to the input sequence 1-2-3-4.

Figure 3. Histograms of the reliability ratio (upper for input/output 1-2-3, bottom 2-3-4). 100% would indicate perfect quantitative match between the inter-input intervals and relative spike timing of RS cells. Simulation was run without noise.

Figure 4. Results with injected background noise resulting in 0.5 mV standard deviation of membrane resting potential (A) Injected current as an input (B) Relative spike timing of RS cells (C) reliability ratio histogram in the presence of the background noise

Figure 5. Phase Response Curve for RS #4. X-axis represents the phase of the inhibition. Arrows with numbers represent the timing of inhibition with respect to RS cell firing in the steady state.

Table 1. Definition of the Input ratios, Output ratios and reliability ratios. Where Δt^{x}_{ij} is the time interval between i and j signals, x is equal inp for inputs or out for RS spikes and $i,j \in \{1,2,3,4\}$.

Table 2. Percentage of the reliability ratios within (50,150)% range for different background noise.

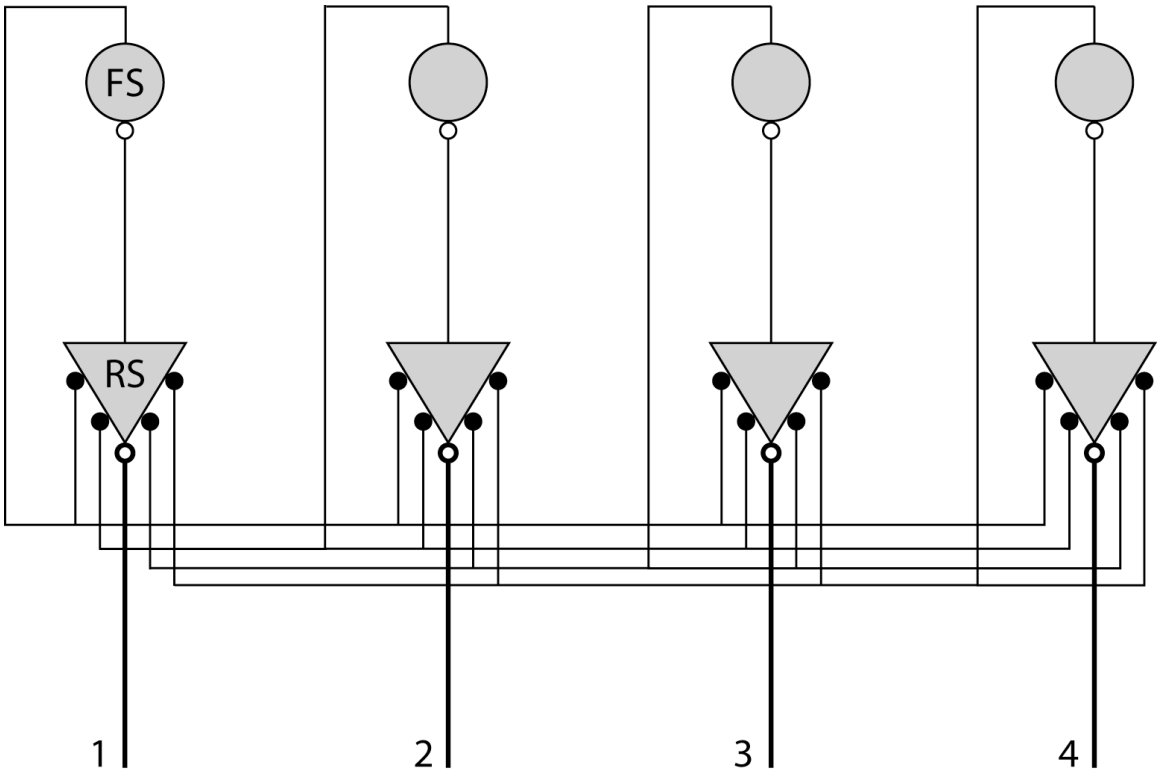

Figure 1.

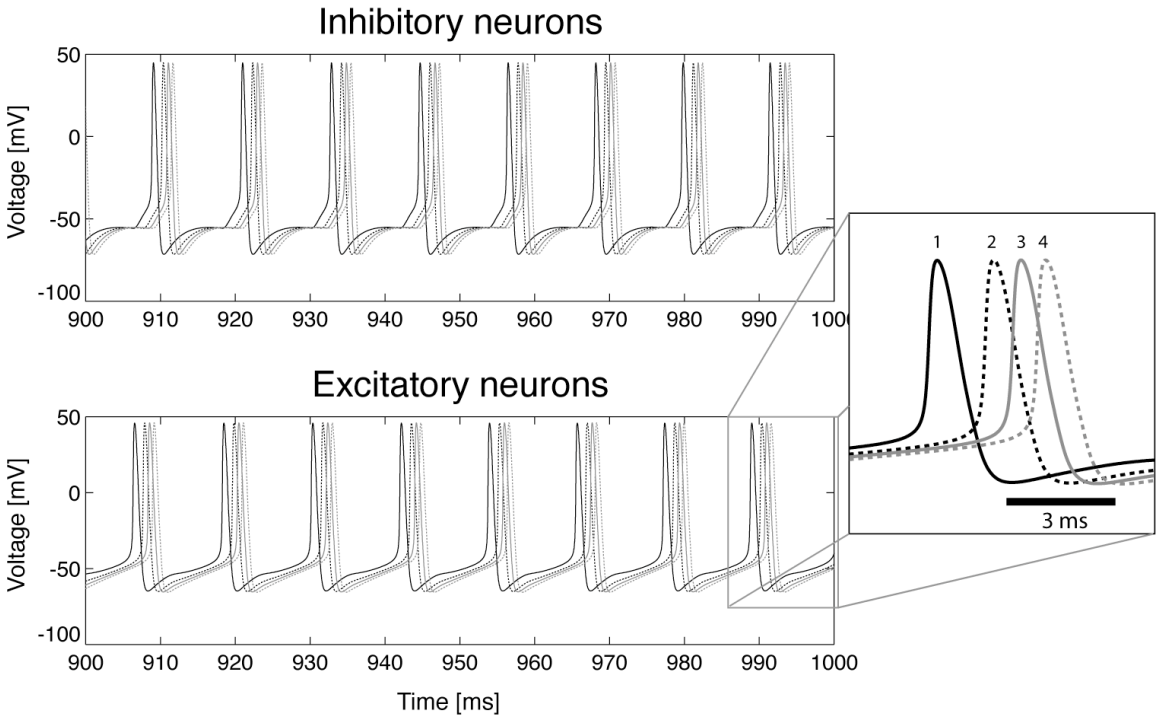
Inhibitory neurons
Excitatory neurons
Voltage [mV]
50
0
-50
-100
900
910
920
930
940
950
960
970
980
990
1000
Time [ms]
1
2
3
4
3 ms

Figure 2.

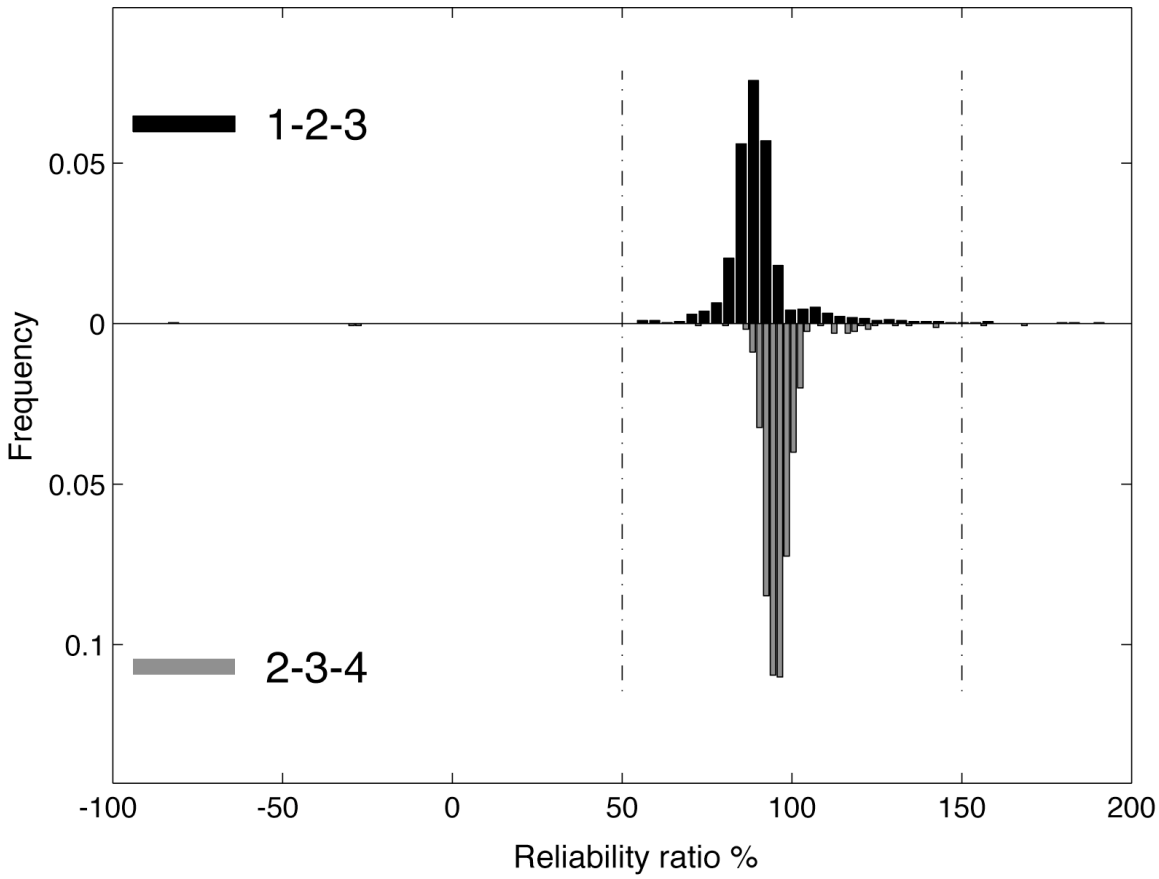
1-2-3
2-3-4
0.05
0
0.05
0.1
Frequency
-100
-50
0
50
100
150
200
Reliability ratio %

Figure 3,

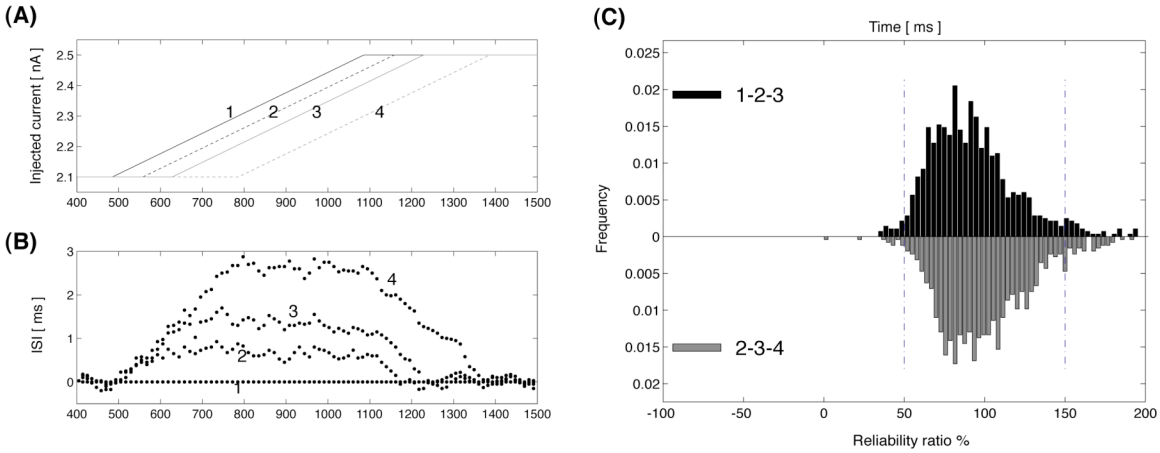
(A)
Injected current [nA]
2.5
2.4
2.3
2.2
2.1
1
2
3
4
400 500 600 700 800 900 1000 1100 1200 1300 1400 1500
(B)
ISI [ms]
3
2
1
0
4
3
2
1
400 500 600 700 800 900 1000 1100 1200 1300 1400 1500
(C)
Time [ms]
Frequency
0.025
0.02
0.015
0.01
0.005
0
0.005
0.01
0.015
0.02
1-2-3
2-3-4
-100 -50 0 50 100 150 200
Reliability ratio %

Figure4.

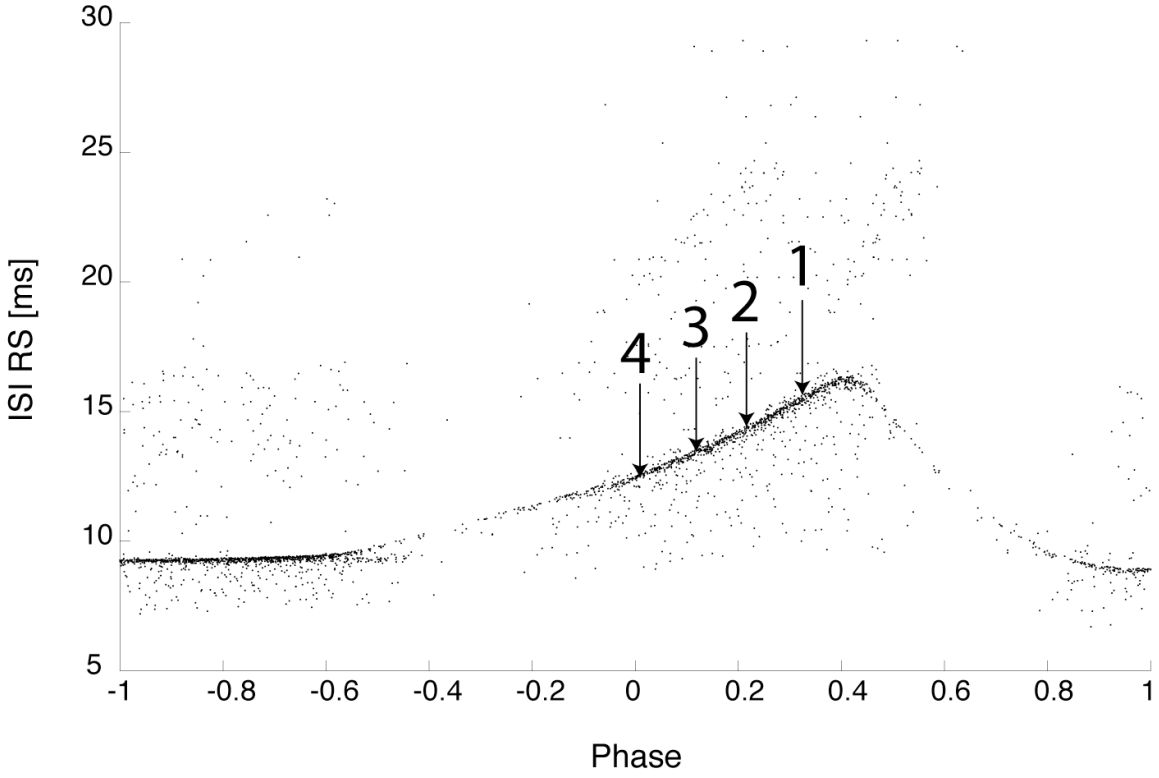

Figure 5.

Input ratio	$r_{123}^{inp} = \frac{\Delta t_{32}^{inp}}{\Delta t_{21}^{inp}}$	$r_{234}^{inp} = \frac{\Delta t_{43}^{inp}}{\Delta t_{32}^{inp}}$
Output ratio	$r_{123}^{out} = \frac{\Delta t_{32}^{out}}{\Delta t_{21}^{out}}$	$r_{234}^{out} = \frac{\Delta t_{43}^{out}}{\Delta t_{32}^{out}}$
Reliability ratio	$r_{123} = \frac{r_{123}^{out}}{r_{123}^{inp}} 100\%$	$r_{234} = \frac{r_{234}^{out}}{r_{234}^{inp}} 100\%$

Table 1.

σ [mV]	1-2-3 [%]	2-3-4 [%]
0.5	92.18	91.95
0.7	68.25	69.64
1.0	35.12	37.44

Table 2.