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From stochastic resonance to brain waves

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Abstract

Biological neurons are good examples of a threshold device – this is why neural systems are in the focus when looking for realization of Stochastic Resonance (SR) and spatio-temporal stochastic resonance (STSR) phenomena. In this Letter a simple integrate-and fire model is used to demonstrate the possibility of STSR in a chain of neurons. The theoretical and computational models so far suggest that SR and STSR could occur in neural systems. However, how significant is the role played by these phenomena and what implications might they have on neurobiology is still a question. Because the direct biological proof of SR and STSR seems to be a tough issue one might look at indirect ways to decide whether the internal noise plays any constructive role in the nervous system. A loop of neurons is shown to have interesting features (frequency selection) which might supply a clue for answering the previous question. © 2000 Published by Elsevier Science B.V. All rights reserved.

1. Introduction

Many scientists have been concerned lately with stochastic resonance (SR), a paradoxical phenomenon in which an optimal noise intensity maximizes the information transfer through a nonlinear device [1–21]. One of the simplest devices showing this phenomenon is the level-crossing detector (LCD) which ‘fires’ a pulse at its output whenever the signal at its input crosses a threshold [14–16]. Such a device is also an acceptable model for a biological neuron since they both have a threshold, a crucial feature in signal detection.

A generalization of the notion of SR has been introduced recently in [17], called spatio-temporal stochastic resonance (STSR) in which perturbation propagation in a subexcitable medium is optimized by noise [17,22–25].

It has been shown by computer simulation that STSR is a basic feature of both neural and glial systems. These systems have been investigated by two different ways of modelling, both leading to the same conclusion: that there is an optimal amount of noise which optimizes the perturbation propagation. The reason for this phenomenon lies in the common feature of the two models, that is, they both represent a network of interconnected threshold elements [14,17,26].

The big question is whether this is valid in reality, or does Nature take advantage of STSR (or SR)? Is it

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possible that organisms are using STSR and SR, having tuned their noise (and all of their other parameters) through adaptation and/or millions of years of evolution to the optimal value? It has been shown experimentally that animals like the crayfish can take advantage of the external noise present in the environment when detecting weak signals [11]. SR effects due to internal noise, however, are very difficult to observe in neural cells [27] because by trying to tune the internal noise one changes the cell parameters drastically. It has been speculated that animals benefit from their internal noise also [12]. Quite recent experiments demonstrated the behavioural significance of external noise, which can help the paddlefish in capturing its prey [28].

Nevertheless, there seems to be no *direct* way of proving the existence of STSR (or SR) in natural tissues by using internal noise. It is disputable whether or not internal noise can play any constructive role in biology in addition to external noise [18]. In order to answer this question it is needed to turn to *indirect* ways of investigation. Hence the next question could be: even if it is not possible to ‘reproduce’ SR in biological systems by tuning their internal noise, can we simulate well-known biological (or medical) phenomena by using noisy subexcitable systems?

Since the end of the last century it has been known that periodically fluctuating potentials can be measured outside the skull of an animal, by a method called electroencephalography (EEG). The frequency and amplitude of these fluctuations vary with the alertness of the subject, giving rise to beta, alpha, theta and delta waves. The deeper the sleep, the higher the amplitude and slower the frequency. There have been many attempts to explain this collective oscillation of the neurons in the brain. One of the recent theories supposes a thalamocortical loop of neurons. When external excitation ceases, the loop enters a self-oscillating state, and its neurons begin unrestrained rhythmic firing. The theory leads to ‘EEG waves’ similar to the measured ones [29–31]. These models suppose that even in sleep, impulses circulate around closed loops in the brain.

This is why it is interesting to study loops formed out of a series of neurons as introduced later (Section 2.1). Is it possible that specific eigenfrequencies are selected by our brain structures simply because of

their topology? This is the question we are trying to address in this Letter. First we prove that STSR arises as a consequence of the threshold properties of the neurons, then we go on to modelling brain waves.

2. Results

2.1. The model

A chain of LCDs was constructed in the following way: $\bigcirc \rightarrow \bigcirc \rightarrow \dots \rightarrow \bigcirc$. Each element of the chain had the following parameters: threshold (V_T), spike length (T_I), the net capacitance of the synaptic input area in each neuron ($C = 1$), recovery period length (T_R), Excitatory Post-Synaptic Potential (q) per unit time (ΔT) arising in the next neuron after an action potential arrives to the synapse, and memory (or time constant, τ) representing the time for which the neuron ‘remembers’ the incoming charge quanta q [32]. All the elements of the chain operate in an all-or-none fashion: their output within Δt can only be either a constant value q , or nothing.

The most important parameter describing every neuron’s ($LCD_n, n \geq 0$) dynamics is its excitability $e_n(t)$ – an integer number:

- if $e_n < -T_R$ the neuron is *excitable*;
- if $-T_R < e_n < 0$ the neuron is in a *recovery period* and its output is equal to zero no matter what is the depolarization on the input;
- if $0 < e_n$ the neuron is *excited* and it is emitting constant amounts of charge into the synaptic gap.

The first element of the system (labelled LCD_0) is different from all the rest: it is a subthreshold oscillator, meaning that it generates its own signal even without excitation from other neurons. Constant amounts of charge q are emitted into LCD_1 for a time $T_I \Delta T$ whenever an additive Gaussian white noise $\xi_0(t)$ helps a sinusoidal oscillation cross the threshold of LCD_0 . For the next T_R units of time, LCD_0 is incapable to emit any charge. This LCD does not have memory, and the equations describing its dynamics are:

$$\begin{aligned}
 e_0(t) = & e_0(t - \Delta T) - 1 + (T_I + 1) \\
 & \times H[-e_0(t - \Delta T) - T_R] \\
 & \times H[V_0(t - \Delta T)]
 \end{aligned} \tag{1}$$

$$V_0(t) = A_0 \sin(2\pi ft + \phi) + N_0 \xi_0(t - \Delta T) - V_T \quad (2)$$

$$Q_0(t) = qH[e_0(t)] \quad (3)$$

In Eqs. (1)–(3) the parameter $V_0(t)$ represents the voltage fluctuations of the cell, A_0 , f and ϕ are the usual parameters describing a harmonic function and $Q_0(t)$ is the charge entering LCD_1 . The value of f was always chosen to be much lower than any of the other frequencies specific to the LCDs (e.g. $1/\tau$, $1/T_1$, $1/T_R$). The function H is the well-known Heaviside step-function:

$$H(x) = 0, \text{ if } x \leq 0, \text{ and } 1, \text{ if } x > 0 \quad (4)$$

The existence of such subthreshold oscillators in cortical areas has been proven recently [33,34]. The noise $N_0 \xi_0(t)$ is completely independent of the noise applied on $LCD_n, n > 0$.

The rest of the LCDs are passive (they can just transmit a signal, not generate one), and are numbered $LCD_1, LCD_2, LCD_3, \dots$. During the simula-

tion, their dynamics is described by the following equations:

$$e_n(t) = e_n(t - \Delta T) - 1 + (T_1 + 1) \times H[-e_n(t - \Delta T) - T_R] \times H[V_n(t - \Delta T) - V_T] \quad (5)$$

$$V_n(t) = \sum_{\Theta=t-\tau\Delta T}^{t-\Delta T} \frac{Q_{n-1}(\Theta) + N\xi_n(\Theta)}{C} \times H[-e_n(t)] \quad (6)$$

$$Q_n(t) = qH[e_n(t)] \quad (7)$$

The variables in Eq. (5)–(7) have the following meaning: $V_n(t)$ is the depolarization at the input of the n th neuron at time t , $Q_n(t)$ is the charge emitted by the n th neuron within the unit time ΔT . The coupling between the elements of the LCD chain is unidirectional.

In a few words, the Eqs. (5)–(7) mean the following: during the simulation, the excitability $e_n, n \geq 0$

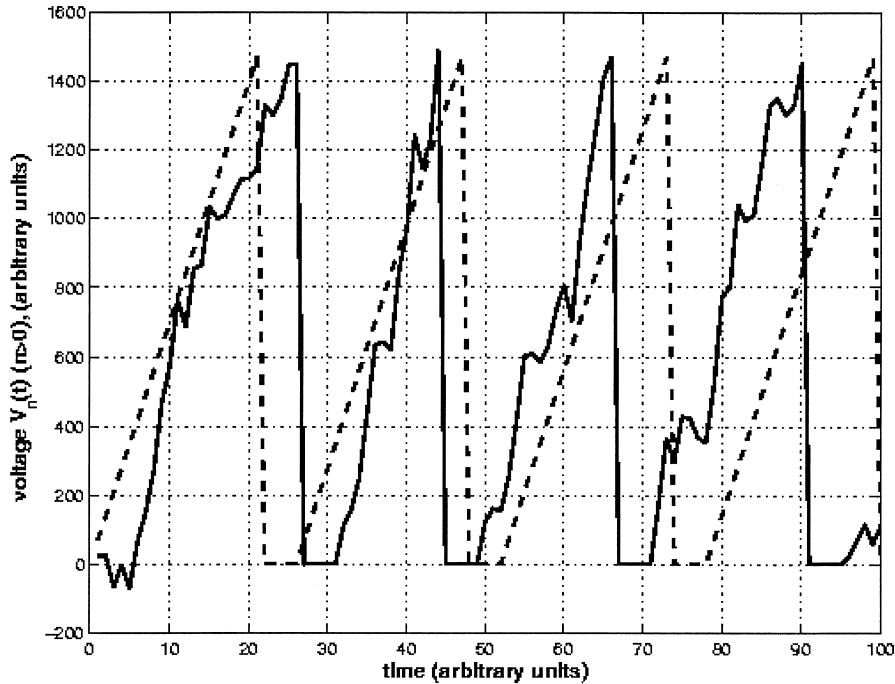


Fig. 1. The voltage $V_n(t)$ of $LCD_n (n > 0)$ when the incoming current is constant. The traces that are: without noise (continuous line) and with noise added to $Q_{n-1}(t)$ (dashed line).

is decreased by 1 at every time step, and it is reset to $e_n = T_1$ whenever the neuron is excitable ($e_n < -T_R$) and the voltage is above the threshold ($V_n(t) > V_T$). The voltage $V_n(t)$ is the sum of the $Q_{n-1}(t) + N\xi_n(t)$ values which arrived within the previous τ time steps, and is reset to zero while LCD_n is excited ($e_n > 0$). The neuron does not emit any charge until it is excited.

The Gaussian noise term $N\xi_n(t)$ added to the input of the n th neuron at time t , has the property

$$\langle \xi_n(t) \xi_m(t') \rangle = \delta(t-t') \delta_{mn} \quad (8)$$

The intensity of such type of noise (i.e. $N\xi_n(t)$) is defined by its standard deviation, equal to N (ξ_n itself is defined to have a standard deviation $\sigma = 1$).

Eqs. (5)–(7) are similar in nature to the integrate-and-fire model of neurobiology [35] (the sum in Eq. 6 is in fact an integration over the interval $[t - \tau\Delta t, t]$). This model is, however, even more simplified (from a computational point of view): the usual definition of the time constant $\tau\Delta t = R/C$, corresponding to an exponential decay of any depolariza-

tion becomes a time within which the neuron completely remembers and any depolarization (and completely forgets it if it arrived before $t - \tau\Delta t$). Hence the exponential decay is replaced by a step function:

$$\begin{aligned} Q(t) &= Q(0)e^{-t/(\tau\Delta t)} \rightarrow Q(t) \\ &= Q(0)H(\tau\Delta t - t), \quad t > 0 \end{aligned} \quad (9)$$

A similar two-dimensional model has been used for the definition of STSR in 1995 [17]. The present model has the following new features:

- the spikes fired by the neurons are no longer delta-functions. They are rather square pulses of amplitude q and of duration T_1 .
- the neurons have a memory $T_1\Delta t$ during which all the depolarization is remembered completely. $Q_n(t - \Theta)$ is forgotten completely if $\Theta > \tau\Delta t$.

The voltage $V_n(t)$ of LCD_n ($n > 0$) when $Q_{n-1}(t) = \text{const.}$ can be seen on Fig. 1. Two cases are plotted: with no noise and with Gaussian noise $\xi_n(t)$ added to $Q_{n-1}(t)$ at every moment t . One can get a

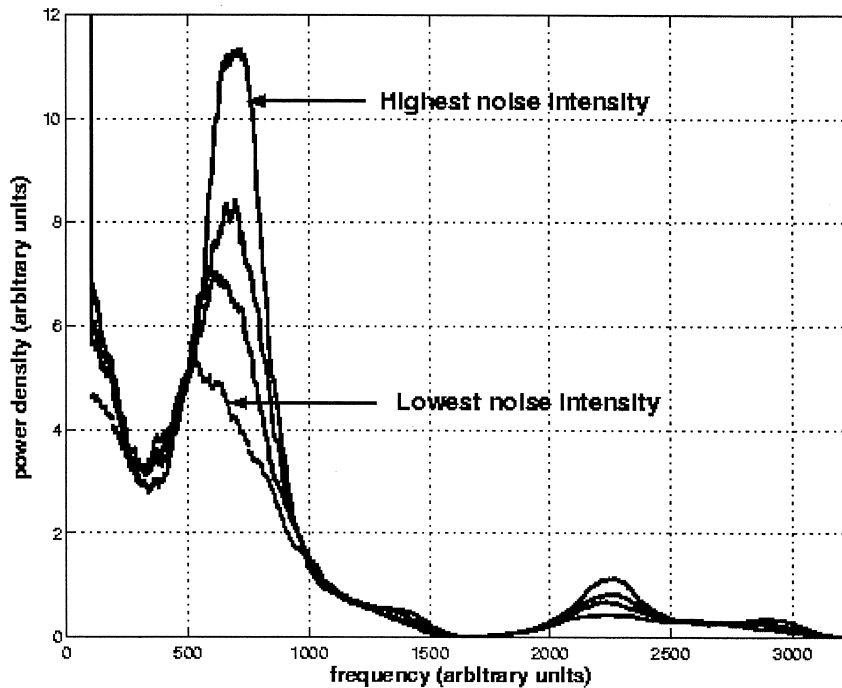


Fig. 2. The spectral density of LCD_n ($n > 0$) if only noise is present on the input. One can see an increase of the peak height and a shift to higher frequencies as the noise intensity increases.

feeling that even if for some reason without noise there were no chance for $V_n(t)$ to reach the threshold, the presence of noise could facilitate threshold crossing. The incoming charge per time step was $Q_{n-1}(t) = 60$ and held constant, other parameters were: $\tau = 30$, $T_I = 5$, $T_R = 5$, $\Delta t = 0.25$, $V_T = 1500.0$. The fact that noise facilitates signal propagation is also illustrated by Fig. 2, where the incoming current is $Q_{n-1}(t) = 0$.

If one would look at the power spectrum of $LCD_n(n > 0)$ with no input ($Q_{n-1}(t) = 0$), without noise there would be no output. If a Gaussian white noise ($N \neq 0$) is also present, the power spectrum on the output for sufficiently high noise intensity N would look like in Fig. 2. The LCD's input contains in this case sequences of random walk, (because of integration) and the probability of threshold crossing becomes nonzero.

One can notice the presence of a peak in the spectrum without any periodic signal on the input. The height of the peak increases with the noise intensity N . In addition, the peak shifts to the right

as we increase the noise intensity. The shift appears just for the lower range of noise intensities. These features of the output of $LCD_n(n > 0)$ can be explained in the following way:

- The presence of the peak: there is an inherent periodicity in the behaviour of $LCD_n(n > 0)$. This periodicity is a consequence of the fact that the time between two consecutive spikes cannot be lower than $(T_I + T_R)\Delta t$. If the location of the peak is f_0 , it means that the most frequent interspike interval is $1/f_0$.
- The increase of the peak height: for higher noise intensity more threshold crossings happen. This increases the output power of $LCD_n(n > 0)$.
- The shift to the right for low noise intensities: the most frequent interspike interval decreases as more and more threshold crossings occur.

2.2. Spatio-temporal stochastic resonance

We set up this system in such a way that noise is necessary for propagation at any 'synapse'. This can

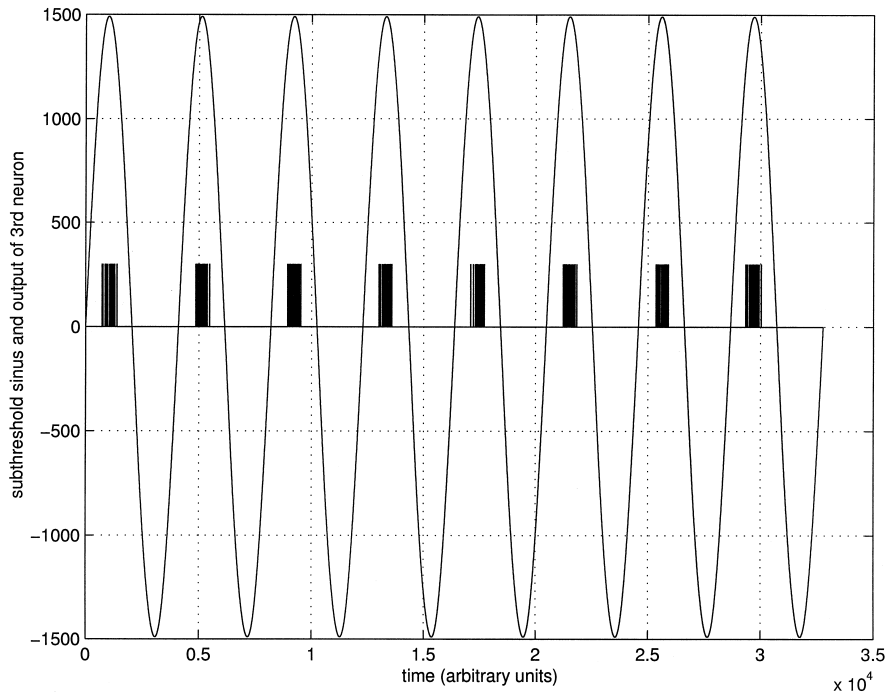


Fig. 3. The subthreshold oscillation and the EPSP's emitted by the 3rd neuron. One can notice that the bursts of spikes are in good synchron with the peaks of the sinus oscillation.

be achieved by adjusting q . Above a *critical value* of q (given by Eq. 10), the perturbation propagates with no loss along the chain.

$$\frac{V_T}{qC\Delta t} = T_I \frac{\tau}{T_I + T_R} + \left\{ T_R \frac{\tau}{T_I + T_R} \right\} \quad (10)$$

Here $\{x\}$ means the fractional part of x , so $x = n[x] + \{x\}$, $n \in \{\dots, -2, -1, 0, 1, 2, \dots\}$. For fairly small q , the perturbation is not propagated farther than LCD_1 . Close to the critical value for q , there can be a few LCDs which are ‘excited’ by LCD_0 (the oscillator), depending on the value of the time constant (τ). Therefore q describes the excitability at a given synapse. By adding gaussian white noise ($N\xi_n(t), n > 0$) to each of the synapses, the effect of the oscillator can be extended along the chain. This noise can arise biologically from signals sent by other neurons to the same synapse or from the neuron itself. It is an internal noise in the sense that it arises inside the organism during information processing. The noise intensity N_0 is different from N ; while N_0 is held constant, N is going to be varied. N_0 is chosen such a way that the SNR of the output $Q_0(t)$ is maximized. One could always replace LCD_0

with any device emitting square pulse bursts of constant amplitude. The reason for choosing LCD_0 to be a subthreshold oscillator is entirely biological.

Is the chain of neurons capable of transmitting the information supplied by this oscillator if q is close to, but under the critical value? It all depends on the parameters of the neurons. Defining the information transmission quality as the signal-to-noise ratio at the basic frequency of the oscillator:

$$SNR_n = \frac{S_n - N_n}{N_n}, \quad (11)$$

where S_n and N_n are LCD_n ’s signal power and noise power at the basic frequency, respectively – one can track down the information propagation along the chain.

First of all, the oscillating cell is going to emit bursts of spikes, and these bursts follow more or less the peaks of the subthreshold sinusoidal oscillation (see Fig. 3). The neurons farther down the chain will also tend to fire when the previous neuron fired. Although the noise we add to q at the synapses helps the oscillator (LCD_0) extend its effect over to farther and farther LCD’s, it also makes the SNR decrease along the chain.

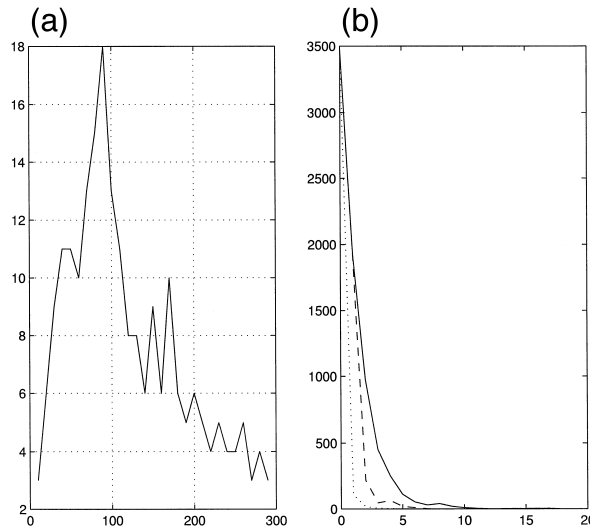


Fig. 4. a. Propagation length as function of noise intensity b. SNR versus distance for three different noise intensities: dashed line = low noise; continuous line = optimal noise; dotted line = high noise. The parameters used were $V_T = 1500$, $T_I = 5$, $T_R = 5$, $\tau = 20$, $q = 280$ (the critical value is $q = 300$).

The value of the SNR is represented in Fig. 4 as a function of the distance from LCD_0 . The same signal $Q_0(t)$ was applied repeatedly at the entrance of LCD_1 . Only the noise terms $\xi_n(t)$ ($n > 0$) were varied. One can notice a decay along the chain, and can define the ‘propagation length’ in the following way: the number of the LCD where SNR becomes less than 1.5 for the first time. This definition of the propagation length is similar to the one used in [36]. Also on Fig. 4 can be seen the dependence of the propagation length upon noise.

Although the SNR decays with distance along the chain of neurons, according to Fig. 4 there is an optimal noise intensity, for which the signal propagates the farthest. Therefore in this simple model we have STSR. The shape of the STSR curve depends on q , that is, for higher q the peak increases in amplitude and its location moves toward zero noise intensity. For supercritical q , there is no peak, just a simple decay.

One of the subtleties that must be noted is that signal transmission strongly depends on the filling

factor of the signal at the entrance, that is at low filling factor there is good transmission. The filling factor can be defined as the mean duration of a burst over the period of oscillation. According to [37] the filling factor is what determines the degree to which a stochastic resonator can improve the SNR. This point seems to be of crucial importance here, since as the filling factor approaches 0.5, the propagation ceases totally. Based on the same paper, the conditions for optimal wave propagation can be determined. At the entrance there must be a perfectly periodic square impulse train with low filling factor; the memory of the neurons has to be very small and q close to the threshold. Indeed, in such a setup one can obtain propagation as far as hundreds of neurons away from the subthreshold oscillator.

Another way to improve signal propagation is by considering a multiple chain, in which each neuron in a given section can receive impulses from all the neurons in the previous section. The behaviour of the multiple chain is similar to that of the simple one with the difference that here the neurons also do a

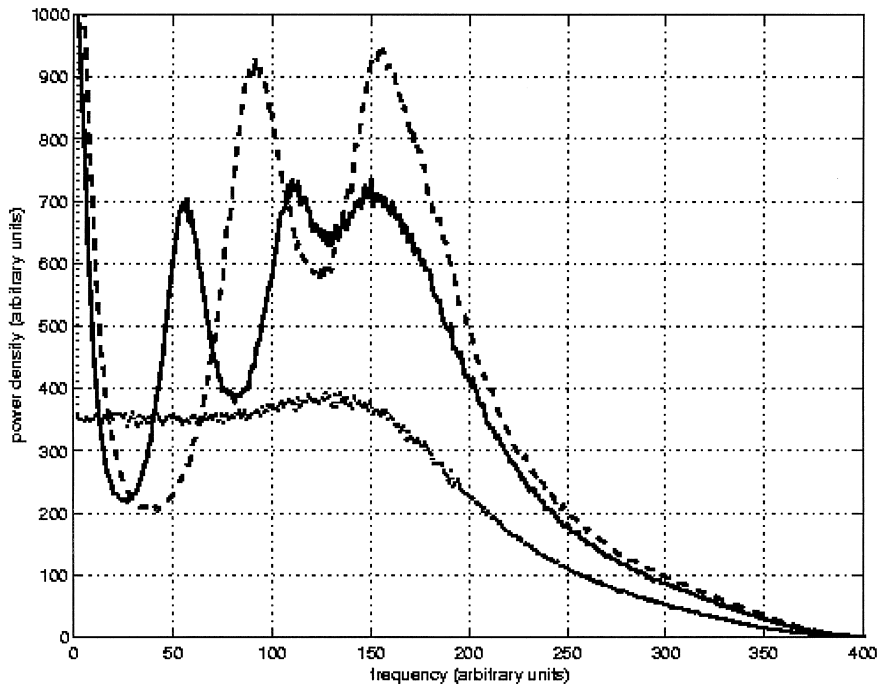


Fig. 5. Averaged power spectra for an LCD inside the loop. Dotted line: no loop; dashed line: loop length = 25; continuous line: loop length = 40. Other parameters are: $V_T = 1500$, $T_I = T_R = 20$, $\tau = 2$, $q = 1450$, $N = 60$, $f_{\text{cutoff}} = 2/(T_I + T_R)$.

spatial summation of the incoming impulses (from the previous chain section).

To summarize, the STSR effect appears because:

- By adjusting the excitability q we set up the system such a way that it is only able to transmit perturbation within a few elements of the chain. This is going to be the case for low noise intensity also;
- For high noise intensity the effect of the noise is overwhelming compared to the effect of LCD_0 ;
- There is an optimal noise intensity for which the effect of LCD_0 can be extended farthest down the LCD chain.

2.3. The effect of a back-connection

A loop has been formed from the previous LCD chain in the following way: the output of a specific LCD from the array was back-connected to the input of LCD_1 . Therefore this LCD's input was equal to the sum of the outputs of LCD_0 and the back-connecting LCD:

$$V_1(t) = \sum_{\Theta=t-\Delta T}^{t-\tau\Delta T} \frac{Q_0(\Theta) + N\xi_1(\Theta)}{C} \times H[-e_1(t)] Q_L(\Theta) + N\xi_1(t) \quad (12)$$

After such a setup, a random (Poisson-) series of spikes was fed into the loop via LCD_0 . The probabil-

ity that M spikes are generated by LCD_0 within a time interval T is described by Poisson's law:

$$p(M, t) = \frac{(rT)^M}{M!} e^{-rT} \quad (13)$$

where r is the average generation rate. The propagation of the Poisson process along the chain was again assured by adding noise to the EPSP's at each of the synapses. The LCD's had high excitability, with $V_T = 1500$, $q = 1450$, $\tau = 2$. The power spectrum of a given LCD inside the loop was plotted after averaging. The results for different lengths of the loop can be seen on Fig. 5 (the loop lengths are defined as the number of LCDs from the LCD with two inputs to the LCD from which the back-connection was made).

The plot just shows the region of lowest frequencies of the power spectrum. The spectrum is a series of bumps with decreasing height. Here one can only see the first bump from the spectrum. It is obvious that the spectrum is smooth if the chain does not have a back-connection. Making a back-connection creates peaks in the power spectrum. Analyzing the position of the peaks that appear, the following is observed:

- the locations of the two peaks corresponding to the loop with length 40 are, respectively: $f_{11} = 57$

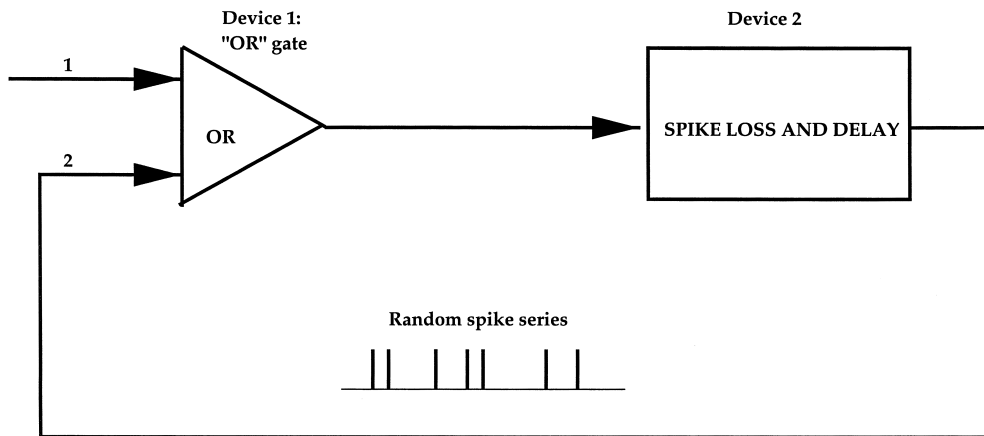


Fig. 6. Electronic circuit for producing frequency selection. A random series of spikes of length 1 time unit were fed into the circuit through input 1. The two devices were incapable of producing output for 1 time unit after each spike. The random spike series at input 1 of the 'OR' gate resulted from the threshold crossing of an uncorrelated gaussian process. The threshold was 1, the standard deviation of the Gaussian process was 0.5, the probability of spike loss was 0.1, and the delay caused by the second device was a variable parameter. The spikes were of unit amplitude. Frequencies proportional to the reciprocal of the delay were selected.

± 3 and $f_{12} = 112 \pm 6$. The ratio of these numbers shows that f_{12} and f_{11} , are harmonics:

$$\frac{f_{11}}{f_{12}} \approx 0.5089 \approx \frac{1}{2} \quad (14)$$

where f_{11} is the fundamental frequency (or first harmonic), and f_{12} is the second harmonic.

- the location of the first peak corresponding to the loop with length 40 and the first peak corresponding to the loop with length 25 are, respectively: $f_{11} = 57 \pm 3$ and $f_{21} = 91 \pm 3$. The ratio of these numbers shows that they are frequencies selected by the loop:

$$\frac{f_{11}}{f_{21}} \approx \frac{57}{91} \approx 0.626 \quad (15)$$

$$\frac{L_2}{L_1} = \frac{25}{40} \approx 0.625 \quad (16)$$

where L_1 and L_2 are the loop lengths, equal to 40 and 25, respectively. This situation reminds one

the fact that in a resonating cavity, the eigenwavelengths are proportional to the dimension of the cavity (e.g. the length of a tube or the perimeter of a Bohr orbit), or the eigenfrequencies are proportional to the inverse of the cavity's dimension:

$$f_n = \frac{v}{\lambda_n} = n \frac{v}{2L}, \quad n = 1, 2, 3, \dots \quad (17)$$

where f_n and λ_n are the frequencies and wavelengths of the n th harmonic, respectively, v is the wave velocity, and L is the dimension (length) of the cavity.

The 'selection of frequencies' does not mean anything analogous to standing wave formation in classical mechanics. The eigenfrequencies, however, are related to the length of the loop in a similar fashion as the standing waves in classical mechanics or electrodynamics. A more suitable analogy would be the bloop produced with a microphone and a

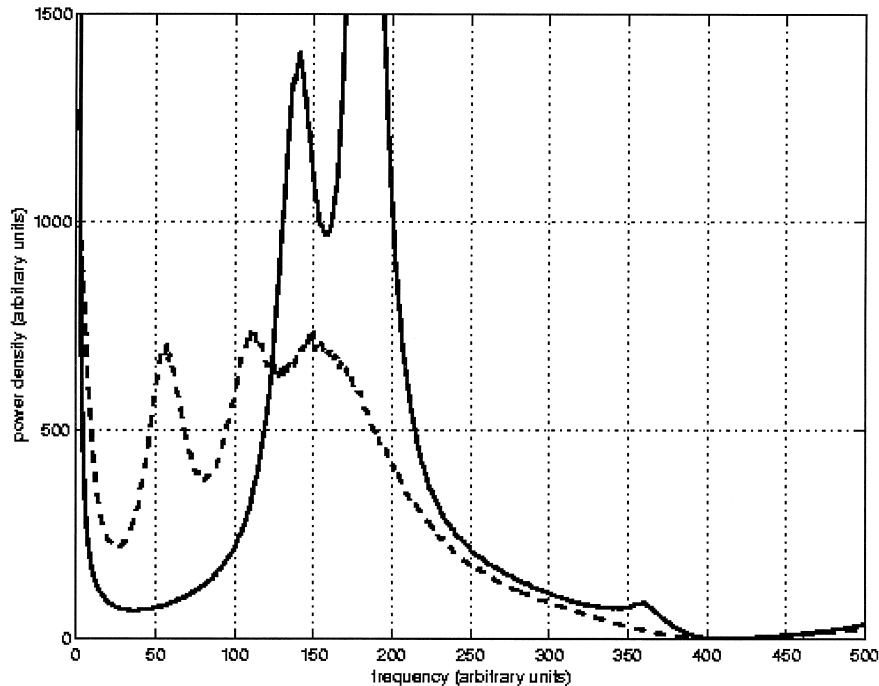


Fig. 7. Power spectra for an LCD inside the loop for two different noise intensities. Dashed line: noise = 60; continuous line: noise = 200. Other parameters were chosen as before.

speaker by positive feedback. The effect can be reproduced with a simple electronic circuit, where LCD_1 is replaced with an ‘OR’ gate, with inputs from LCD_0 and LCD_L . The rest of the chain ($LCD_2 \rightarrow \dots \rightarrow LCD_L$) is replaced by a device which causes a delay of the spike train and loss of spikes with a given probability (see Fig. 6). It is easy to show by computer simulation that this simple circuit selects frequencies similarly to the simulated neuronal loop. The role of the noise besides assuring propagation is to decrease the number of propagating spikes along the loop, thereby avoiding complete reverberation (and saturation of spike density).

The explanation of this phenomenon lies in Pulse Frequency Modulation (PFM), which is the basis of how the LCD transfers a low frequency signal [38]. It is basically an additive and linear process: if spikes originating from two or more different process are ‘added’ by LCD_1 , the resulting output signal will

contain the sum of the individual low frequency signals. The condition for this is that the spike overlap should be small. A random spike train ‘carries’ a wide spectrum of frequencies. The two spike trains entering the ‘OR’ gate (or LCD_1) must have a maximum in their cross-correlation function due to the fact that one is simply the time-shifted noisy copy of the other. This maximum causes the eigenfrequency peaks to appear.

The ‘eigenfrequency peaks’ have another interesting feature which does not apply for the classical (or quantum mechanical) eigenmodes: their location is dependent on the noise intensity present at the synapses. This is represented in Fig. 7. The only explanation for this fact can be that the speed of perturbation propagation increases with noise intensity. Thus the location of the peaks are shifted, according to Eq. 18. The peak location versus noise intensity can be seen in Fig. 8.

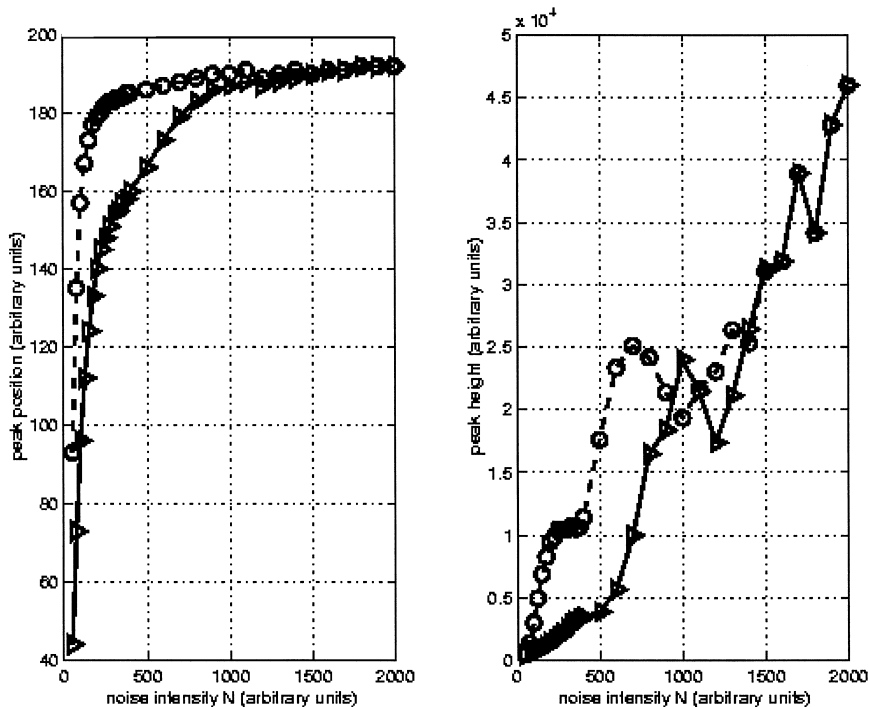


Fig. 8. Left: peak location versus noise intensity. The location of the first (\triangleright) and last (\circ) peaks are represented. Only the first peak is due to the back connection, the last peak appears simply because of the inherent periodicity of the LCDs. Right: height of the first (\triangleright) and last (\circ) peaks versus noise intensity. The parameters were $\tau = 2$, $T_I = 20$, $T_R = 20$, $q = 1450.0$, $V_T = 1500$, $N_0 = 50$.

The frequency selection described here appears for various parameters, requiring the following conditions:

- a choice of parameters (including noise intensity) such that the propagation length discussed in Section 2.1. is greater than the loop length:

$$L_{\text{prop}} > L_{\text{loop}} \quad (18)$$

This requires low τ and high q – in other words, the ‘neurons’ in the chain must have high excitability.

- the loop length has to be such that the arising eigenfrequencies are smaller than half of the cut-off frequency specific for each element of the LCD chain, which is equal to

$$f_1 < f_{\text{cutoff}} = \frac{2}{T_1 + T_R} \quad (19)$$

The question arises, why consider noise-supported propagation? The reason is that considering q exceeding the threshold, the back-connection causes a build-up of the power of the propagating signal

because of the back-connection. After a few reverberative cycles the spike density reaches its maximum.

3. Conclusions and further questions

In conclusion, it has been shown that for highly excitable neural pathways show the following features based on a simple integrate-and-fire LCD model:

- STSR is possible in such systems, meaning that there is a specific internal noise intensity for which a periodic perturbation applied at the entrance is detectable farthest down the chain;
- if a loop is formed out of linearly connected LCDs, and fed with a random (Poisson-)process, specific frequencies are selected by the loop if the parameters are such that the propagation length is greater than the length of the loop.

If highly excitable neural pathways exist in the brain, which make at least a back-connection, one

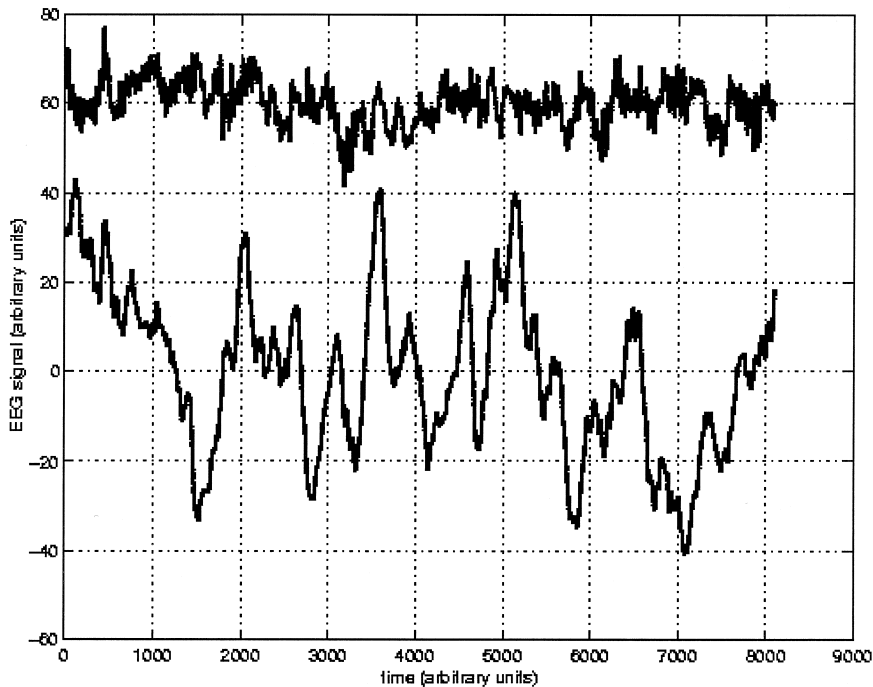


Fig. 9. Simulated EEG recordings for a loop of length 100. The upper recording corresponds to high noise, the lower to medium noise.

can speculate that the phenomenon can be connected to the EEG waves for two reasons. First, the selected eigenfrequencies shift towards the lower range for decreasing noise intensity. Secondly, the amplitude of a simulated EEG recording decreases with noise intensity.

The ‘EEG waves’ were simulated the following way: the individual ‘recordings’ of all LCDs in the chain were averaged, for several Poisson processes at the entrance:

$$\text{EEG}(t) = \left\langle \sum_{n=0}^L Q_n(t) \right\rangle_{\text{Poisson processes}} \quad (20)$$

Averaging over Poisson processes means that we run the simulation with several realizations of the Poisson process at the entrance of the chain, and average the results. Finally a running average was calculated on the resulting (averaged) recordings, to eliminate high-frequency components (this is also done by EEG recorders in practice). The simulated ‘recordings’ can be seen in Fig. 9.

Supposing that being awake and sleep represent different noise environments, the frequency shift and amplitude increase of the EEG waves are explained by this simple model.

Further investigation is needed to study the biological validity of the introduced phenomena. There is no theory up to this point explaining these interesting effects. Further research goals include study of the response of a loop to periodic driving. The response of the loop is to be dependent on the driving frequency, and new resonance effects are to be expected.

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References

- [1] R. Benzi, A. Sutera, A. Vulpiani, *J. Phys. A* 14 (1981) L435.
- [2] C. Nicolis, G. Nicolis, *Tellus* 33 (1981) 225.
- [3] R. Benzi, G. Parisi, A. Sutera, A. Vulpiani, *SIAM J. Appl. Math.* 43 (1983) 565.
- [4] R. Benzi, K. Wiesenfeld, R. Roy, *Phys. Rev. Lett.* 60 (1988) 2626.
- [5] A. Longtin, A. Bulsara, F. Moss, *Phys. Rev. Lett.* 67 (1991) 656.
- [6] A. Bulsara, E. Jacobs, T. Zhou, F. Moss, L. Kiss, *J. Theor. Biol.* 152 (1991) 531.
- [7] M.L. Spano, M. Wun-Fogle, W.L. Ditto, *Phys. Rev. A* 46 (1992) R5253.
- [8] P. Jung, U. Behn, E. Pantazelou, F. Moss, *Phys. Rev. A* 46 (1992) R1709.
- [9] M.I. Dykman, H. Haken, G. Hu, D.G. Luchinsky, R. Mannella, P.V.E. McClintock, C.N. Ning, N.D. Stein, N.G. Stocks, *Phys. Lett. A* 180 (1993) 332.
- [10] A. Bulsara, G. Schnera, *Phys. Rev. E* 47 (1993) 3734.
- [11] J. Douglass, L. Wilkens, E. Pantazelou, F. Moss, *Nature* 365 (1993) 337.
- [12] K. Wiesenfeld, D. Pierson, E. Pantazelou, F. Moss, *Phys. Rev. Lett.* 72 (1994) 2125.
- [13] F. Moss, in: *An introduction to Some Contemporary Problems in Statistical Physics*, George H. Weiss (Ed.), SIAM, Philadelphia, 1994, pp. 205–248.
- [14] P. Jung, *Phys. Rev. E* 50 (1994) 2513–2522.
- [15] Z. Gingl, L.B. Kiss, F. Moss, *Europhys. Lett.* 29 (1995) 191.
- [16] Z. Gingl, L.B. Kiss, F. Moss, in: A.R. Bulsara, S. Chillemi, L.B. Kiss, P.V.E. McClintock, R. Mannella, F. Marchesoni, K. Nicolis, K. Wiesenfeld (Eds.), *Int. Workshop on Fluctuations in Physics and Biology: Stochastic Resonance, Signal Processing, and Related Phenomena*, *Nuovo Cimento D* 17 (1995) 795.
- [17] P. Jung, G. Mayer-Kress, *Phys. Rev. Lett.* 47 (1995) 2130–2133.
- [18] J.J. Collins, C.C. Chow, T.T. Imhoff, *Nature* 376 (1995) 236.
- [19] S.M. Bezrukov, I. Vodyanoy, *Nature* 378 (1995) 362.
- [20] Z. Neda, *Phys. Rev. E* 51 (1995) 5315.
- [21] B. Shulgin, A. Neiman, V. Anishchenko, *Phys. Rev. Lett.* 75 (1995) 4157.
- [22] M. Löcher, G.A. Johnson, E.R. Hunt, *Phys. Rev. Lett.* 77 (1996) 4698.
- [23] S. Kádár, J. Wang, K. Showalter, *Nature (London)* 391 (1998) 770–772.
- [24] H. Hempel, L. Schimansky-Geier, J. García Ojalvo, *Phys. Rev. Lett.* 82 (1999) 3713.
- [25] P. Jung, A. Cornell-Bell, F. Moss, S. Kádár, K. Showalter, *Chaos* 8 (1998) 567–575.
- [26] G. Balázs, L.B. Kiss, F. Moss, in: D. Abbott, L.B. Kiss (Eds.), *Proc. 2nd International Conference on Unsolved Problems of Noise and Fluctuations*, July 1999, Adelaide, Australia, in press.
- [27] E. Pantazelou, C. Dames, F. Moss, *Int. J. Bif. Chaos* 8 (1995) 101.
- [28] D.F. Russell, L.A. Wilkens, F. Moss, *Nature* 402 (1999) 291–294.
- [29] J.A. Hobson, *Sleep*, New York: Scientific American Library: distributed by W.H. Freeman, 1995.

- [30] A. Destexhe, *Phys. Lett. A* 187 (1994) 309–316.
- [31] D. Contreras, A. Destexhe, T.J. Sejnowski, M. Steriade, *J. Neurosci.* 17 (1997) 1179–1196.
- [32] J.E. Dowling, *Neurons and Networks – An Introduction to Neuroscience*.
- [33] Y. Gutfreund, Y. Yarom, Idan Segev, *J. Physiol.* 483 (1995) 621.
- [34] H.A. Braun, M.T. Huber, M. Dewald, K. Schäfer, K. Voigt, *Int. J. Bif. Chaos* 8 (1998) 881.
- [35] H.C. Tuckwell, *Introduction to Theoretical Neurobiology*, Cambridge University Press, New York, 1988.
- [36] J.F. Lindner, S. Chandramouli, A.R. Bulsara, M. Löcher, W.L. Ditto, *Phys. Rev. Lett.* 81 (1998) 5048–5051.
- [37] K. Loerincz, Z. Gingl, L.B. Kiss, *Phys. Lett. A.* 224 (1996) 63–67.
- [38] L.B. Kiss, in: R. Katz (Ed.), *Chaotic, Fractal, and Nonlinear Signal Processing*, American Institute of Physics Press, August 1996.