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DYNAMIC PROPERTIES OF A BIOLOGICALLY MOTIVATED NEURAL NETWORK MODEL

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We develop a neural network model based on prominent basic features of biological neural networks. The description keeps a simple but coherent link between the subneuronal, neuronal and network levels. In addition, the variables of the model are endowed with realistic numerical values together with their physical units. This permits to reach quantitative significance for the results. To describe the operation of the neuron, a transfer function is used that is believed to convey more biological significance compared to the usual sigmoid transfer function. It is shown that the dynamic properties of the network, which can vary from stability to chaos, are significantly influenced by the choice of the neuron transfer function. Constraints on the synaptic efficacies, as imposed by Dale's rule, are also shown to modify the dynamic properties by increasing the stability of the network. A simple neural architecture is presented that leads to a controllable time evolution of the network activities.

1. Introduction

A central question that arises in the modelling of complex systems like neural networks is the level of description that is suitable to adopt. On the one hand, oversimplified models may be thoroughly computable but will probably yield only trivial or unsignificant results. On the other hand, overly detailed models may be very uneasy to develop farther and more importantly, they may obscure fundamental general properties. The level of description to adopt, in between these two bounds, is in fact very difficult to assess *a priori*; it is in no way unique and largely depends upon one's aims and scope.

In this paper, we consider a neural network model which is based on prominent features of biological neural networks. In the description, we seek to keep a coherent link between the subneuronal, neuronal and network levels. In addition, we pay special attention to attributing to the variables of the models, realistic numerical values together with their physical units. This contrasts with what is frequently done in neural network models where variables are often considered as mere mathematical variables rather than as true physical quantities. The model we develop is simple enough to be studied in various conditions and complex enough to be capable of nontrivial behaviors. What can be interpreted as quantitative prediction can be extracted from the model.

2. The Neural Network Model

In the model we are developing, a neuron i is described with an input signal $I_i(t)$ representing its membrane current and an output signal $f_i(t)$ representing a short-term average of its firing frequency. A given number of these neurons are connected through a set of synapses to form a network. w_{ij} shall denote the synaptic efficacy from neuron j to neuron i. The time evolution of the membrane current $I_i(t)$ is governed by the dynamics of the ion channels of the postsynaptic membrane which themselves are activated by the neurotransmitters existing in the synaptic regions. These neurotransmitters in turn are released by presynaptic neurons in function of their activities, modelled here by the $f_i(t)$'s. A detailed account of these processes is beyond the scope of this paper. But more importantly, we want to focus the analysis not at the neuron or subneuron level but at the network level. So we try to include in the model only prominent features of the single neuron and then investigate the resulting properties at the network level. Accordingly, we chose to model the dynamic of

the current $I_i(t)$ with Eq. (1) which summarizes and simplifies the complex processes governing $I_i(t)$.

$$\frac{dI_i(t)}{dt} = -\frac{I_i(t)}{\tau_I} + \sum_j w_{ij} f_j(t). \qquad (1)$$

Equation (1) expresses that the variation of $I_i(t)$ at time t is formed by a relaxation term $-I_i(t)/\tau_I$ which in its simplest form here is chosen as linear (this term summarizes, for a part, the desactivation of the channels and reuptake processes of neurotransmitters) and by a driving term proportional to the $f_j(t)$'s. In Eq. (1), τ_I is a time constant which determines the typical time scale over which the current $I_i(t)$ can begin to vary significantly.

The input current $I_i(t)$ charges the membrane of neuron i which behaves as a leaky integrator of capacitance C and resistance R. When the electric potential of the membrane reaches the threshold V_s above its resting potential, a spike (an action potential) is emitted by the neuron and the membrane potential is reset to its resting value.

An important property is that the duration of a single spike is much lower, at least an order of magnitude lower, than the time scale τ_I over which the current $I_i(t)$ can begin to vary significantly. It is thus possible to perform a short-term average of the spiking rate of the neuron over a time interval of order τ_I over which the driving current I_i can be considered as constant. We are thus in presence of a simple capacitive leaky integrator driven by a constant current for which we easily compute the time needed for the potential to reach the threshold V_s. We thus deduce that the short-term average firing rate $f_i(t)$ of neuron i can be expressed as

$$f_i(t) = 0 \qquad \text{if} \quad I_i(t) \le I_s$$
$$f_i(t) = \frac{1/T_r}{1 - (\tau_m/T_r)\ln[1 - I_s/I_i(t)]} \qquad \text{if} \quad I_i(t) > I_s, \qquad (2)$$

where $T_r = 1/f_{\max}$ is the absolute refractory period of the neuron, $\tau_m = RC$ the time constant of the membrane and $I_s = V_s/R$.

In this description, we want to pay special attention to attributing realistic numerical value, at least in order of magnitude, to every parameter of the model. We take[1] for the characteristic evolution time of the currents $\tau_I = 10$ ms; for the refractory period which is also of the order of the duration of a spike, $T_r = 1$ ms; for the membrane time constant $\tau_m = 10$ ms. With a membrane resistance $R = 100$ MΩ and a threshold V_s of order 10 mV above the resting potential, it follows $I_s = 0.1$ nA. Based on these values, the variations of the firing rate $f_i(t)$ as a function of the membrane current $I_i(t)$ as expressed by Eq. (2) is depicted in Fig. 1.

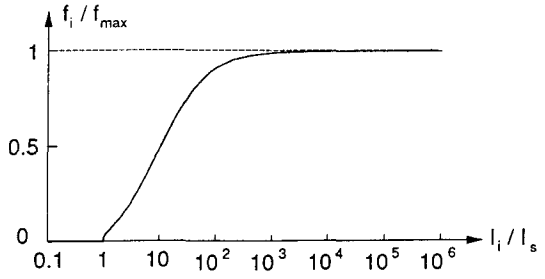

Fig. 1. Input-output transfer function of the neuron described by Eq. (2) with realistic numerical values for the parameters of the model and giving the firing frequency f_i as a function of the membrane current I_i. Note the logarithmic scale for input I_i.

With little surprise, the curve of Fig. 1 exhibits the global shape of a sigmoid. However, this curve here is not postulated at the neuron level, as it is done most of the time in neural network models, but rather it is derived from the description of mechanisms of the subneuronal level. As a result, this input-output curve of the neuron is defined and described with increased accuracy. The curve of Fig. 1 shows a monotone increase and a saturation for large values of the input I_i which together constitute the features that are almost invariably found in any neuron transfer function used in modeling. In addition, the curve of Fig. 1 incorporates an actual threshold below which the output activity is zero and above which the activity is strictly positive and bounded. This threshold, operating in this precise fashion, grounded on physiological mechanisms, is not always present as such in other neuronal transfer functions and as we shall see, it can have an important impact on the dynamic properties of the network. Another very important element conveyed by the curve of Fig. 1 is that the quantities which are involved, namely I_i and f_i, are not considered as mere mathematical variables expressed in arbitrary units, but instead, as actual physical quantities coming with their physical units and realistic orders of magnitude: I_i is a current which can be evaluated in nA, and f_i a frequency in Hz. These quantitative scales for the definition of the neuron transfer function put in light what can be described as a logarithmic influence of the input I_i rather than a linear one. This implies that for the determination of the value of the output f_i, the precise value of the input I_i does not play a

critical role but rather its order of magnitude. This neuron property that appears here can be expected to allow robust behaviors at the network level.

An important element for further use of the neuron transfer function of Fig. 1 in a network is to determine the zone of the curve which is actually covered by the neuron when operating in a typical environment. If this operating zone is typically confined to the region of the threshold of I_i far from saturation, the collective behavior at the network level can be expected to be quite different compared to that based on an operating zone located mainly in the saturation region of the curve. The determination of this operating zone requires to evaluate a typical order of magnitude for a synaptic efficacy w_{ij} that plays a role in the driving term of the currents in Eq. (1). For this evaluation, we assume that on a monosynaptic pathway, a typical presynaptic signal $f_j = 0.1 f_{\max}$ is just sufficient to activate the postsynaptic neuron i, providing that the synapse w_{ij} is at its maximum of efficacy. In these conditions, one can estimate a typical order of magnitude for the maximum value of a synaptic efficacy at 0.1 nA.

An interesting consequence can be deduced from the quantitative estimations that we have performed for the parameters of the model. In a typical biological neural network, the connectivity of a neuron may vary typically between 1 and 10^4. A neuron i in these conditions can be submitted at most to a maximum activation which would be delivered by approximately 10^4 presynaptic neurons firing at a rate around $f_{\max}$. With the values of the parameters and Eq. (1), this would drive the input current I_i of this neuron in the region where I_i/I_s is around 10^4 to 10^5. This bound fixes the limit of the operating zone of the transfer function f_i versus I_i. Such an upper bound, directly related to the maximum connectivity of the neuron, shows that it is with a typical maximum connectivity of 10^4 that the neuron in operation in a network performs what can be seen as an "optimal" use of its nonlinearity. In other words, in view of the neuron transfer function of Fig. 1, if one tries to predict what should be the maximum connectivity of this neuron in a typical network in order to perform optimal use of the nonlinearity of its transfer function, a reasonable answer would be a connectivity of 10^4. A maximum connectivity significantly lower, of order 10^2 or below, would not allow the neuron to benefit from the nonlinearity introduced by the saturation of its response. Conversely, a maximum connectivity significantly higher, of order 10^6 or above, would lead to a neuron behaving for most input levels as a two-state neuron and little use would be made of the continuous curvilinear part of the response.

This point demonstrates that neural network models should not necessarily be confined to qualitative description. Even in simplified models, if the parameters are treated as physical quantities endowed with realistic numerical values and their units, quantitative predictions or at least quantitative consistency can be obtained. In our view, such quantitative aspects have to be progressively introduced with more and more emphasis in neural network models in order to fully appreciate, beyond qualitative aspects, the understanding that they can bring concerning biological neural systems.

3. Dynamic Properties of the Network

With the time evolution specified by Eqs. (1) and (2) very complex dynamics are potentially accessible for the activities $f_i(t)$ that depend critically upon the values of the w_{ij}'s.

To illustrate this, we have simulated a network of 100 neurons, fully connected through synaptic efficacies w_{ij} randomly drawn with uniform probability out of the interval [−0.1 nA, 0.1 nA]. This represents a rather small network, having an equal amount of excitatory and inhibitory synapses, which are totally unconstrained except for the order of magnitude of their efficacies that is set on physical grounds. Figure 2 represents two typical evolutions of an output activity $f_i(t)$ for two different sets of w_{ij}. In Fig. 2(a), after a short transient, the output frequency $f_i(t)$ rapidly converges to a stable state of activity which represents a small fraction of the maximum firing frequency $f_{\max}$. Such low levels of firing activity are frequently observed in biological neural networks.[2] They come here as a direct consequence of the neuron transfer function properly quantified as depicted in Fig. 1. In contrast, with neural network models based on a standard sigmoid transfer function, high firing frequencies close to $f_{\max}$ are often artificially favored.

Besides this kind of stable evolution of Fig. 2(a), the other typical kind of evolution which has been observed is that depicted in Fig. 2(b). Here we have a time evolution which never stabilizes, although it remains in the range of low firing rates, and which is in fact chaotic. For evolutions such as that of Fig. 2(b), classical characteristics of deterministic chaos were

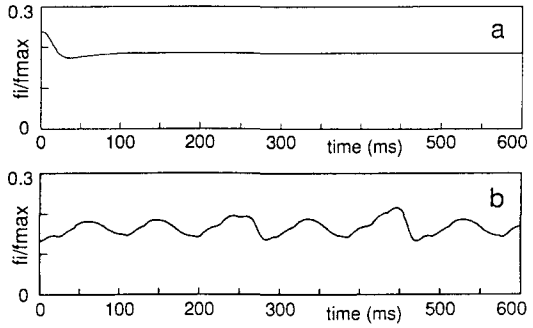

Fig. 2. Two typical time evolutions generated by Eqs. (1) and (2) for a network of 100 neurons. (a) Stable evolution. (b) Unstable chaotic evolution.

easily observed, such as sensitive dependence on initial conditions and strange attractors. In Fig. 3, we have plotted the attractor reached by the system in a phase plane spanned by a pair of arbitrary outputs $\{f_i(t), f_j(t)\}$. The highly structured and thinly folded figure can be qualified of fractal attractor and it reveals a low-dimensional chaotic regime. In the neural systems we are investigating here, we see that two output variables $f_i(t)$ and $f_j(t)$ evaluated at the same time t can play the role of phase space variables that are able to indicate the presence of low-dimensional chaos. This is not classical since usually a phase plane for the system would rather be constructed with quantities as $f_i(t)$, $f_i(t+\Delta t)$. The low dimensional attractor of Fig. 3 can be interpreted as revealing a coherence in the time evolution of the variables $f_i(t)$ and $f_j(t)$. If these two variables were completely independent, one could expect the attractor to densely fill a region of the phase plane. Conversely, if these two variables were connected by a fixed functional dependence, one would expect a regular one-dimensional curve as attractor. The fractal attractor of Fig. 3 describes an intermediate situation characterizing complex connection that can be found between the many output variables belonging to a same neural network.

In view of the possibility for the networks of stable and unstable chaotic regimes, we have investigated the probability of occurrence of one or the other of these regimes. We have considered fully connected networks of N neurons, with synaptic efficacies belonging to certain classes defined by a specified rule to assign them a value. With networks of given size N and given rule to assign values to the synapses, we form an ensemble of networks. In the ensemble, a given network is characterized by a set of synapses having values selected randomly out of the class defined by the assignment rule. A probability of stability can then be evaluated as a statistical frequency of stability over the ensemble of networks. For each network of the ensemble, initial conditions are selected randomly and the dynamics described by Eqs. (1) and (2) is let to evolve for a sufficient time after which the outputs $f_i(t)$ are tested to check if they have reached a stable state (as in Fig. 2(a)) or not (as in Fig. 2(b)). The probability of stability follows as the ratio of the number of evolutions having led to a stable state to the total number of gener-

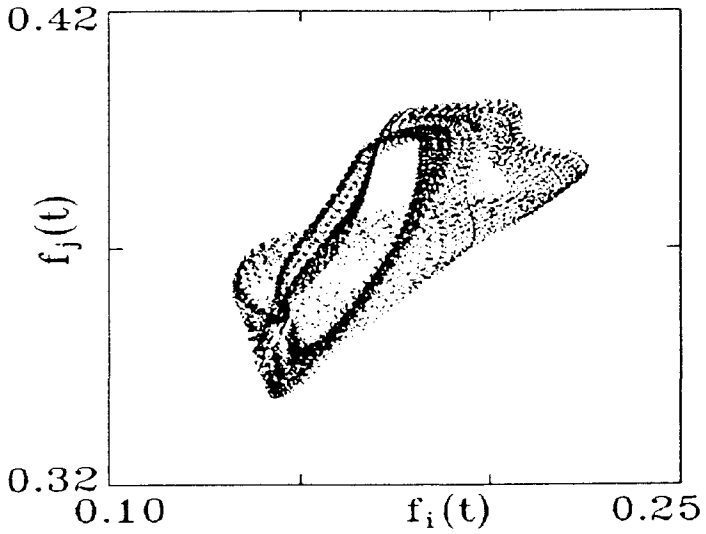

Fig. 3. Phase space attractor for a chaotic evolution in a 100-neuron network. The fractal structure is characteristic of low-dimensional chaos.

ated evolutions. The probability of stability is then studied as a function of the size N of the network.

Figure 4 represents the variation of the probability of stability of a network of N neurons (i.e. the probability that all the neurons of the network have a stable evolution) in which the synaptic efficacies w_{ij} are randomly drawn with uniform probability out of the interval [−0.1 nA, 0.1 nA].

The curve of Fig. 4 shows that the probability of stability for the network is close to 1 for small networks and then regularly decreases as the size of the network is increased. This enhancement of unstability is a general behavior which can be found in many systems when more and more units with random interactions are acting collectively. If the curve of Fig. 4 is extrapolated for large networks of biological size, unstability is then a very probable feature. For most of the unstable evolutions that we observed, unstability is due to the onset of a chaotic regime displaying sensitive dependence on initial conditions. In such a regime, the macroscopic states of activity of the neurons are very unlikely to be the substrate for any "cognitive" or "informative" process because these states will depend critically on uncontrollable microscopic fluctuations. Thus, in general, very specific mechanisms have to come in play for the precise tuning of the synaptic efficacies if for instance stability is required to implement cognitive processes such as memory.

It is interesting to compare the stability curve of Fig. 4 to the same type of curve obtained with a network in which the neurons are endowed with a classic sigmoid input-output transfer function[3] as described by Eq. (3).

$$f_i(I_i) = \frac{f_{\max}}{1 + \exp[-\beta(I_i - I_s)]}. \tag{3}$$

With this approach, the transfer function of Eq. (3) is usually postulated for the neuron. A difficulty is then to attribute meaningful numerical values to the parameters β and I_s. One understands that a value of β of 10^3 or of 10^{-3} may indeed change the resulting properties of the neuron network. With no clear justification, the slope β and the threshold I_s are usually chosen close to 1 with no mention of physical units. With $\beta = 1$, and $I_s = 1$ when I_i is expressed in nA, we have used Eq. (3) in place of Eq. (2) in the simulation of the neural network dynamics. The resulting stability curve that corresponds to that of Fig. 4 is given in Fig. 5.

The curves of Figs. 4 and 5 clearly show that the two neural networks based on two different transfer functions do not have the same dynamical properties. The neural network associated with the curve of Fig. 4 is intrinsically more stable. This may be attributed to the fact that its neuron transfer function incorporates an actual threshold below which the output activity is strictly zero and that the

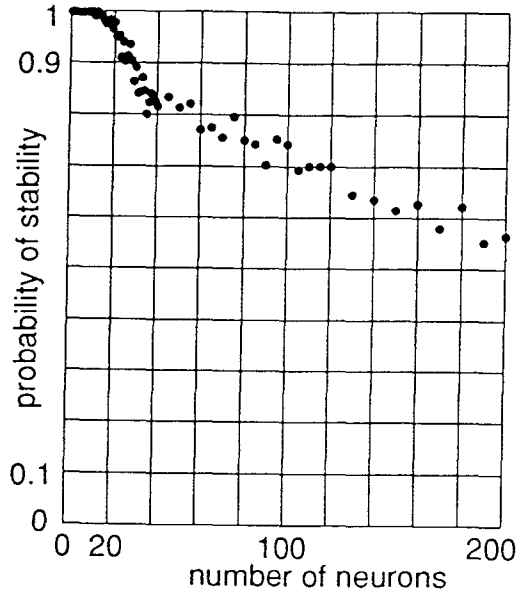

Fig. 4. Probability of stability for a network with a neuron transfer function according to Eq. (2) and with completely random synaptic efficacies.

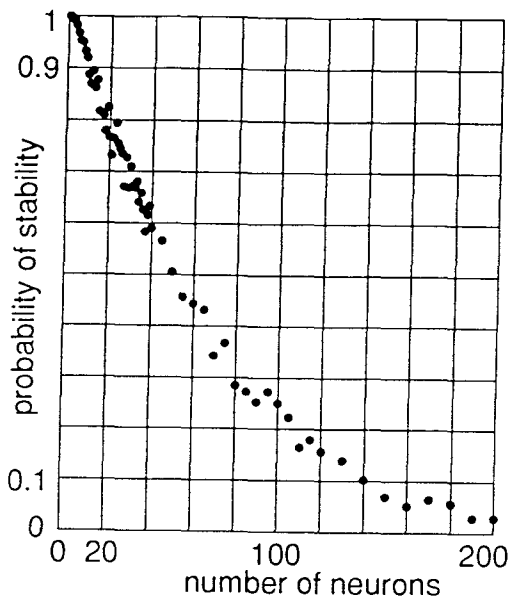

Fig. 5. Probability of stability for a network with a sigmoid neuron transfer function according to Eq. (3) and with completely random synaptic efficacies.

sigmoid does not have. In addition, the increase of the output f_i with input I_i is much slower for Eq. (2) than for the sigmoid of Eq. (3) even with low values of β. As a consequence of these differences, the modelling of the dynamic properties of the neural network may differ significantly depending on the choice of the transfer function for the neuron. A transfer function as that of Eq. (2), deduced from subneuronal properties and expressed with "physical" numerical values, will *a priori* bear more significance.

For the network based on the transfer function of Eq. (2), we have also evaluated the probability of stability when the synaptic efficacies are constrained with the biological specification expressed by Dale's rule. Dale's rule, although probably not of thorough validity in biological neural networks, is a principle based on experimental observations which specifies that generally the synapses emitted by a given neuron are all of the same type (either excitatory or inhibitory).[4]

Figure 6 represents the variation of the probability of stability of a network of N neurons in which the synaptic efficacies are randomly drawn with uniform probability out of the interval [−0.1 nA, 0.1 nA] and with a random but fixed sign for all the synapses w_{ij} emitted by a same neuron j.

The curve of Fig. 6 when compared to that of Fig. 4 shows that the stability of the network is largely increased on average when Dale's rule operates to organize the synapses. If neural networks are to perform memory tasks based on the learning and retrieval of stable states of the dynamics of their activities, Dale's rule may thus be a useful condition to preorganize the synapses in the direction of that goal.

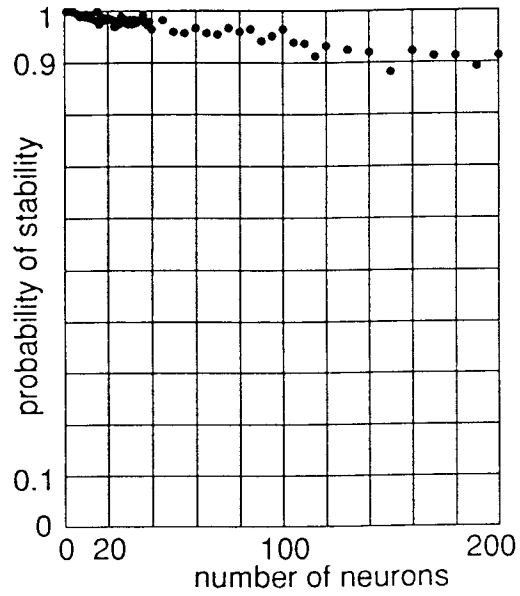

Fig. 6. Probability of stability for a network with a neuron transfer function according to Eq. (2) and with random synaptic efficacies constrained by Dale's rule.

4. Realization of a Specific Dynamic Evolution

We saw that in a fully connected network, the output activities $f_i(t)$ can have complex time evolutions that critically depend on the set of synaptic efficacies w_{ij}. An important question is to examine how it is possible to determine a set of w_{ij} in order to obtain a specific dynamic evolution for the network. This problem is usually answered with the development of learning algorithms. For instance Hopfield[5] gives a procedure to define the synaptic weights of a neural network,

(i) which leads to dynamics which are always stable and
(ii) whose stable states can be imposed.

This scheme however relies on symmetric synaptic weights which are difficult to biologically justify. Beyond the imposition of static behaviors to a neural network, the emphasis is turning now to the imposition of dynamic behaviors. Williams and Zipser[6] have proposed an algorithm to learn, not simply the stable states of a stable dynamics but a complete time evolution of a network. For instance, the synaptic weights can be adjusted to obtain a network which produces a periodic oscillation at a given frequency. However, this type of approach has an important limitation: no control is provided to adapt the characteristics of the evolution that has been learnt. For instance, if the period of an oscillation is to be modified, a complete learning phase has to be started anew.

These approaches that we just mentioned are based on networks with nonspecific architectures. We want to draw the attention here with an example on the interest of using networks with specific architectures in order to obtain a specific dynamic evolution together with its control.

We consider the neural circuit depicted in Fig. 7 with a dynamics governed by Eqs. (1) and (2). Neuron 1 receives a constant external input $f_0 = 0.2 f_{max}$ through a synaptic efficacy $w_{10} = 0.1$ nA. In addition we take $w_{21} = 0.1$ nA and $w_{12} = 0.1$ nA. Because

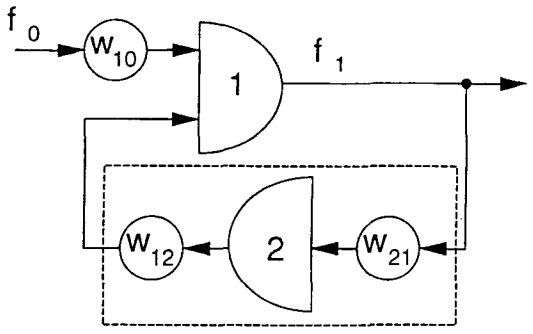

Fig. 7. Neural circuit that behaves as a controllable oscillator. The control of the period of oscillation is obtained through the number of neurons recruited in the inhibitory feedback loop appearing in the dashed box.

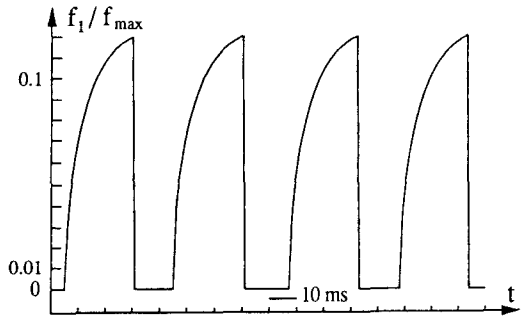

Fig. 8. Typical time evolution of the output activity of the oscillatory circuit of Fig. 7.

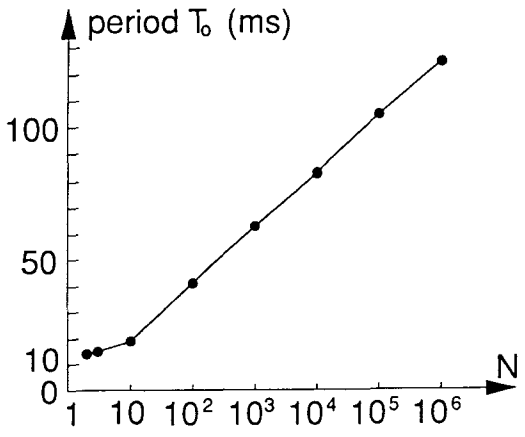

Fig. 9. Period of the oscillation generated by the circuit of Fig. 7 as a function of the number of neurons participating in the recurrent inhibition.

of the recurrent inhibition performed by the feedback loop appearing in a dashed box in Fig. 7, the neural circuit can operate as an oscillator. In the configuration of Fig. 7 with the values given to the parameters, the circuit does not oscillate and its output activity is a constant $f_1 \approx 0.1 f_{\max}$. It is possible to have the circuit oscillate by reinforcing the recurrent inhibition. This reinforcement can be achieved by increasing the number N of the neurons participating in the recurrent inhibition. This is obtained by replicating in a parallel association the feedback structure appearing in the dashed box in Fig. 7. In these conditions, the inhibition signal can vary as the typical connectivity of a neuron (thus as N) in a range of order 1 to 10^4. The oscillation of the circuit starts with $N = 2$. Figure 8 shows a typical time evolution for the output $f_1(t)$ obtained for $N = 100$. In the observed oscillations, as we can note in Fig. 8, the firing frequency f_1 always stays well below the maximum firing rate $f_{\max}$. At most f_1 is of order $0.1 f_{\max}$ which corresponds to low firing rate levels as they are often found is biological neural networks.

Figure 9 gives the evolution of the period T_o of the oscillation as a function of the number N of the neurons participating to the recurrent inhibition. We see from Fig. 9 that the period of oscillation can be continuously varied by means of the number N of neurons recruited to form the recurrent feedback loop. Such a recruitment can be performed for instance by the lifting of an inhibitory signal on a neuron connected in the loop. Moreover, the control which is obtained on the period is robust because it depends very little on the exact value of N, but rather on its order of magnitude. It is interesting to note that when N varies inside the typical range of variation for the neuron connectivity (1 to 10^4), the period of oscillation varies in a ratio of approximately 1 to 5. This ratio from 1 to 5 happens to correspond to a typical range of variation for the period of oscillators controlling rhythmic activities of the organism which are easily observable. For instance we can think of respiration or locomotion and the range of variation of their rhythm between their minimum and maximum levels of activity. Again, we are here in the presence of a quantitative consistency which can be extracted from a simple neural network model when attention is paid to attributing numerical significance to the variables.

5. Conclusion

In this paper, we have investigated a neural network model based on a neuron input-output transfer function which is derived from a simple description of mechanisms of the subneuronal level, rather than postulated at the neuron level. As a

consequence, the neuron transfer function is expressed with parameters to which it is possible to attribute realistic numerical values and physical units. Based on this properly quantified neuron transfer function, it was possible to also quantify the typical value of a synaptic efficacy. From these quantitative estimates, we predicted an order of magnitude for the typical maximum connectivity of a neuron which is in agreement with the values known from experiment.

We then showed that the dynamic properties of neural networks based on this more realistic transfer function are in general complex and capable of chaotic evolutions. Moreover, they are significantly different from the properties of networks based on a standard sigmoid transfer function.

In addition, we showed the influence of the synaptic efficacies to constraint the dynamic variability of the network, for instance the use of Dale's rule as a means to enhance its stability. Specific circuit architectures, rather than complex synaptic plasticity algorithms in completely unstructured networks, are also emphasized in order to obtain specified dynamic behaviors together with their control. An example of a simple oscillating circuit is presented out of which, beyond qualitative reproduction of an evolution, quantitative consistency can be extracted.

The neural network model we examined here is, in its form, as simple as other popular models like Hopfield's[5,7] for instance, and it relies on the same basic elements (nonlinear-response units densely connected in a network). Nevertheless, we believe that this model bears more relevance, because without departing from a simple formulation, it seeks to incorporate quantitative description and a coherent (although very schematical) link between subneuronal, neuronal and network levels. This type of model, simple enough to be easily simulated in many conditions, stands as a kind of "minimal" neural network model appropriate, as a first coarse approximation, to investigate dynamic properties of biological neural systems (as synaptic plasticity for instance) with both qualitative and quantitative prospect.

References

1. C. Hammond and D. Trisch, *Neurobiologie Cellulaire* (Doin, Paris, 1990).
2. D. J. Amit and A. Treves, "Associative memory neural networks with low temporal spiking rates," in *Proc. Nat. Acad. Sci. USA* **86**, 7671 (1989).
3. J. Dayhoff, *Neural Network Architectures* (Van Nostrand Reinhold, New York, 1990).
4. J. C. Eccles, *The Understanding of the Brain* (McGraw-Hill, New York, 1977).
5. J. J. Hopfield, "Neural networks and physical systems with emergent collective computational abilities," in *Proc. Nat. Acad. Sci. USA* **79**, 2554–2558 (1982).
6. R. J. Williams and D. Zipser, "A learning algorithm for continually running fully recurrent networks," *Neural Comput.* **1**, 270–280 (1989).
7. J. J. Hopfield, "Neurons with graded response have collective computational properties like those of two-state neurons," in *Proc. Nat. Acad. Sci. USA* **81**, 3088–3092 (1984).