Biomed. Biochim. Acta **49** (1990) 8/9, 697—707

Complex dynamics and noise in simple neural networks with delayed mixed feedback

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Summary

This paper briefly reviews the role of mixed feedback, neural delays, and neural noise in the genesis of complex oscillations in neurological feedback systems. The results are concretely discussed within the context of recurrent inhibition in the mammalian hippocampus, and a hybrid version of the pupil light reflex with externally imposed electronic feedback.

Introduction

Fluctuating levels of neural activity are commonly observed in a variety of neural systems. For example, rhythmic patterns of neural discharge occur in association with the neural control of respiration, swimming and locomotion, whereas much more complex patterns are seen in the electroencephalogram ([1—5] and references therein).

These experimental observations have, over several decades, spawned a variety of neural oscillator models (for reviews see [4, 6]). These are generally embraced by either pacemaker like models in which there exists spontaneous (autonomous) activity in a given neuronal population, mutually inhibitory models (half center model) in which two populations of neurons are inhibitory to one another and each have tonic excitatory inputs, or the sequential disinhibition (recurring cyclic inhibition) models which are elaborations of the mutually inhibitory models.

Interestingly these network models have almost exclusively ignored a fundamental physiological fact. Namely, neurons communicating with one another do so with delays between the generation and receipt of a signal due to the finite conduction time of action potentials along the axons. A few investigators have taken note of this by including a pure delay in their models [7, 8] without analysis, while others have examined the consequences of incorporating delays either by a careful mathematical analysis of the dynamical properties of the network equations [9—11], by numerical simulation [12—15], by electronic analog simulation [16], or by combination of several of these techniques [11]. In [17] the pure delay was approximated by an "inertial" effect in the underlying network equations.

Here we review the dynamic behaviour of simple recurrent inhibitory neural networks with delay. Recurrent inhibition is ubiquitous in the central nervous system and arises when a tonically active neuron excites a second resting neuron which then, in turn, inhibits the first (Fig. 1). Examples include the inhibition of a spinal motor neuron via an inter-

neuron (Renshaw cell) [18, 19] and the inhibition of a hippocampal neuron via basket cells [7]. In the next section we show that the feedback in a recurrent inhibitory network can be of a "mixed" type, i.e. it can resemble negative feedback over some ranges of the state variable and positive feedback over others [20]. In contrast to neural networks with negative feedback, those with mixed feedback can exhibit exceedingly complex dynamics [10, 20, 21]. We illustrate this point in the third section by discussing the dynamics observed in experiments which involve the pupil light reflex in which the internal feedback normally present is circumvented and externally imposed feedback of mixed type is substituted.

Recurrent inhibitory networks

The dynamics of the neural network shown in Fig. 1 have been investigated previously in the context of the recurrent inhibitory interconnections in the hippocampus [10, 11].

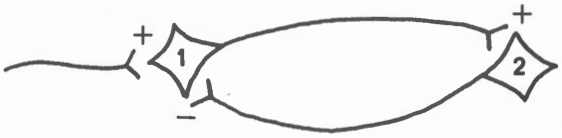

Fig. 1. A simple schematic representation of an excitatory-inhibitory neural network

Briefly, if cell 1 (e.g. CA1 pyramidal neuron) has an excitation level $E(t)$ and a level of inhibition from cell 2 (e.g. basket cell) given by $I(t)$, both measured relative to the resting potential, then it can be shown that the dynamics of the inhibitory potential are determined by the solution of the delay differential equation

$$\frac{dI}{dt} = -\alpha I(t) + \beta F_\tau \frac{K}{K + F_\tau^n}, \tag{1}$$

where $F_\tau \equiv F(t - \tau)$, τ is the time required to transmit information from the soma of cell 1 back to the inhibitory synaptic connection of cell 2 onto the soma of cell 1, and

$$F(t) = \begin{cases} 0 & E - I \leq \theta \\ F_0[E(t) - I(t) - \theta] & E - I > \theta \end{cases} \tag{2}$$

is the instantaneous firing rate in cell 1. For this problem to be well posed, we must specify an initial function $F(\tilde{t})$ for $-\tau \leqq \tilde{t} \leqq 0$.

In equation 1, α is the reciprocal of the cell 1 membrane time constant which determines the rate of decay of the inhibitory potential $I(t)$, K is a constant related to the equilibrium constant of the inhibitory transmitter-receptor complex, n is the number of inhibitory transmitter molecules required to activate one receptor, and β is proportional among other things, to the number N of inhibitory receptors on each cell from the cell 1 population.

In equation 2, θ is the threshold potential for the generation of an action potential, and F_0 is a constant related to the rate of change of the cell 1 firing frequency with respect to potential changes above threshold. To mimic the spike generating mechanism, we assume that an action potential is generated at a time t' after the last action potential where t' is given by the solution of

$$E(t + t_a + t') - I(t + t_a + t') = \frac{\theta}{1 - e^{-(t' - t_a)/\tau_r}}, \tag{3}$$

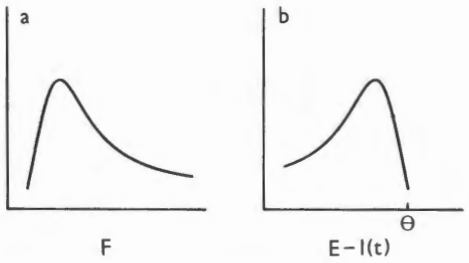

Fig. 2. Illustrating the nature of the mixed feedback function appearing in equation 1 as (a) a function of the firing frequency F in the inhibitory cell, and (b) as a function of the driving potential $E - I(t)$

where t is the time of the last action potential, t_a is the absolute refractory time, and τ_r is the decay constant of the threshold within the relative refractory period.

The feedback in equation 1 is given by the nonlinear term on the right hand side and is illustrated graphically in Fig. 2a. This term gives the rate of change of $I(t)$ due to the combined effect of the excitation of cell 2 by cell 1 and the resultant release of inhibitory transmitter

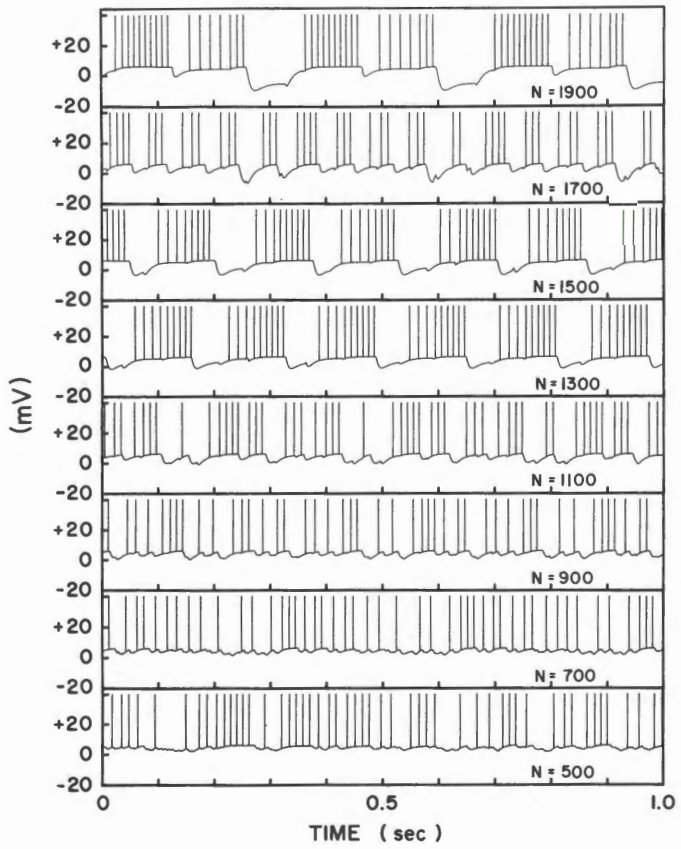

Fig. 3. Model predicted sequences of activity for the parameter values in the text derived for the hippocampal recurrent inhibitory situation.
The voltage values in mV correspond to deviations from the resting potential (0 mV), and the vertical lines superimposed on the variation in the potential $E - I(t)$ correspond to the times of generation of action potentials. The N values in the lower right hand corner of each frame correspond to the assumed number of inhibitory GABA receptors on the some of the CA1 pyramidal cell after MACKEY and AN DER HEIDEN [11]

by cell 2. The amount of inhibitory transmitter released at the cell 2 to cell 1 synapse at time t is proportional to F_τ, and $[K/K + F_\tau^n]$ is the fraction of cell 1 inhibitory receptor sites *available* to be activated by that transmitter. It is this nonlinear term, in combination with equation 2, that confers the "mixed feedback" character to this system. This is because at constant levels of excitatory drive E, increasing the inhibitory potential I from zero leads initially to an increase in this nonlinear term until a maximum value is attained; subsequent further increases in I lead to a progressive decrease in the nonlinearity until it becomes zero when $I = E - \theta$. This gives rise to the "humped" function shown in Fig. 2 b.

As an illustration of the dynamics predicted by this model we use parameter estimations for a recurrent inhibitory circuit within the hippocampus [11] in which cell 1 is to be indentified with the CA1 pyramidal cells and cell 2 with the basket cells. In this system, the excitatory drive is supplied by the mossy fibres, and the inhibitory transmitter is γ-aminobutyric acid (GABA). The parameters for this system are: $\tau = 100$ ms, $\alpha^{-1} = 10$ ms, $K = 1$ Hz^3, $\theta = 4$ mV, $F_0 = 2.25$ Hz/mV, $n = 3$, and $\beta = 2.4$ TmV, where T is the average number of GABA receptors per pyramidal cell.

The range of dynamic behaviours that this model can display are shown in Fig. 3. There, assuming that the excitatory drive E from the mossy fibres is constant with $E = 1.6$, we show the effect on cell 1 (the CA1 pyramidal cell) of decreasing the number of GABA receptors at the inhibitory synapse from $N = 1900$ (top panel) in steps of 200 to a value of $N = 500$ (bottom panel). The vertical lines indicate the occurrence times of action po-

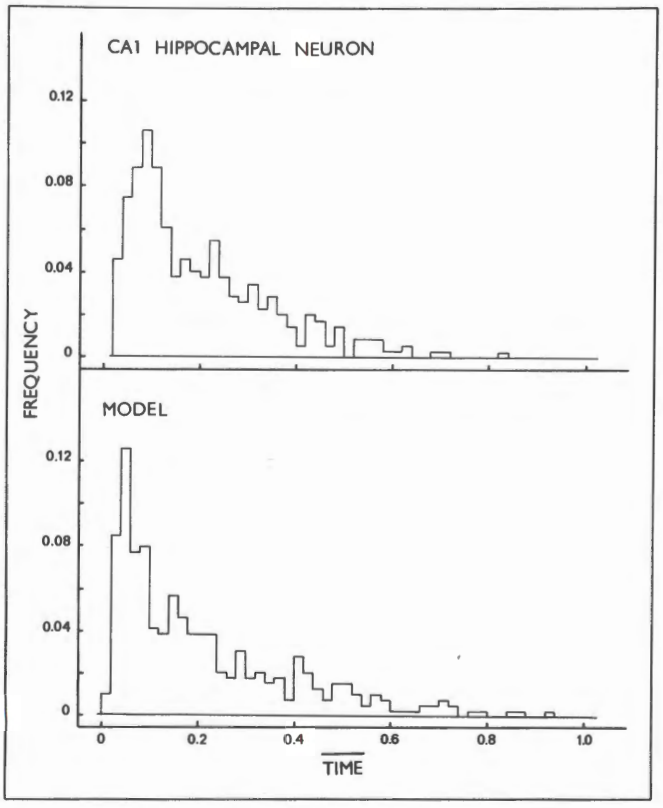

Fig. 4. Histograms of the distribution of interspike intervals for a rat CA1 pyramidal cell (top, data courtesy of Prof. K. KRNJEVIC) and for the model of recurrent inhibition (bottom, calculations courtesy of Mlle. N. MORISSETTE)

tentials with $t_a = 1$ ms and $\tau_r = 10$ ms. As is clear, successive decreases in the number of GABA receptors per cell lead to a progressive loss of the bursting behaviour seen at higher levels of N, until at $N = 500$ the CA1 cell is predicted to be firing almost continuously but in an apparently random fashion.

In Fig. 4 (top panel) we have plotted the experimentally observed distribution of interspike intervals recorded from a rat CA1 pyramidal cell. This is to be compared with Fig. 4 (bottom) where we have plotted the histogram of the model generated interspike intervals for $N = 500$ corresponding to the time series in the bottom panel of Fig. 3. In spite of the fact that there is a close qualitative correspondence between the histograms, there is a quantative discrepancy since the time axis of the data is normalized to 500 ms, while that of the model is normalized to 50 ms.

In spite of the interesting behaviours produced by this simple model for excitatory-inhibitory networks, there are at least two facets that are physiologically unrealistic and which are important to incorporate. The first is related to the assumption that there is a single delay when, in reality, there is a distribution of delays because of the distribution of fibre diameters within the neuronal populations. This distribution will typically be zero up to some minimum delay corresponding to conduction through the shortest and largest portion of the fibre tracts, rise to a maximum, and then decay back to zero corresponding to conduction through the longest and slowest pathways. The second has to do with the well known fact that neuron activation is highly dependent on fibre size. Thus, smaller fibres with longer conduction delays are activated at lower activity levels than are the larger and faster fibres. The consequence of incorporating this, in conjunction with the distribution of delays, is to turn the description of the model into an integro-differential equation with state dependent delays.

Clamped pupil light reflex

Direct verification that delayed "mixed" feedback mechanisms can produce complex dynamics in vivo has been hindered by the lack of suitable experimental paradigms. One exception are experiments that have involved the 'clamped' pupil light reflex [14, 22, 23]. The pupil light reflex is a delayed negative feedback neural control mechanism which regulates the retinal light flux (equal to the light intensity multiplied by the pupil area) by changing the pupil area [21, 24]. The time delay is $\sim$ 300 ms [25]. In the clamped pupil light reflex (Fig. 5) this feedback loop is first "opened" by focusing a small light beam onto the center of the pupil in order to circumvent the shading effects of the iris on the retina [26]. The feedback loop is then reclosed with a clamping box which relates measured changes in pupil area to changes in retinal illumination [5, 22, 23, 26, 27].

The advantage of studying the clamped pupil light reflex is that by appropriate design of the clamping box, the reflex can be made unstable and the types of dynamical behaviours explored in a precisely controlled manner. The design of the clamping box for mixed feedback is shown in the inset to Fig. 5. Precisely speaking this type of feedback is referred to as piecewise constant mixed feedback since the retinal illumination can have only one of two values, i.e. on or off. It clearly is an approximation of the smooth type of mixed feedback shown in Fig. 2. When pupil area exceeds a lower treshold, θ_1, the light is turned on and the pupil will constrict (negative feedback). However, whenever pupil area exceeds a higher area threshold, $\theta_2 > \theta_1$, the light is turned off and the pupil can continue to dilate (positive feedback). Studies of pupil area oscillations that occur when the reflex is clamped

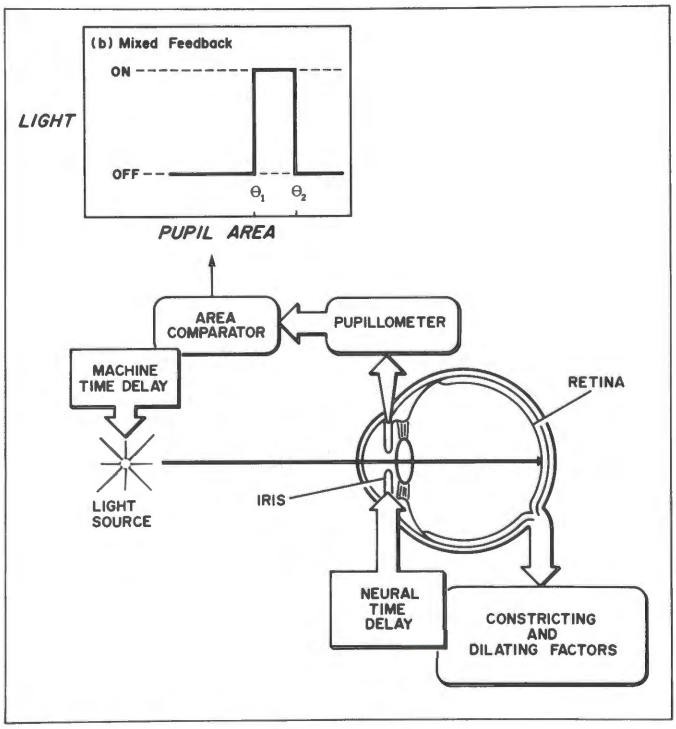

Fig. 5. Schematic diagram of the clamped pupil light reflex. The area comparator or "clamping box" converts pupil area variations into light intensity variations according to a specified feedback function. In our study, the area comparator was used to synthesize piecewise constant mixed feedback, shown in the inset

with piecewise constant negative feedback, i.e. when $\theta_2 \gg \theta_1$, have proven useful as a diagnostic test for the presence of optic nerve demyelination [25] and for quantifying the properties of the efferent arc of the reflex [23].

Figure 6 reviews the results of an experiment in which the pupil light reflex is clamped with piecewise constant mixed feedback [5, 22]. The experimental results have been compared to those predicted by the model

$$\frac{dA}{dt} + \alpha A = \begin{cases} A_{\text{off}}, & \text{if} \quad A_\tau < \theta_1 \\ A_{\text{on}}, & \text{if} \quad \theta_1 < A_\tau < \theta_2 \\ A_{\text{off}}, & \text{if} \quad A_\tau > \theta_2 \end{cases} \tag{4}$$

where τ is the total time delay and A_{on}, A_{off}, are constants which depend on physiological parameters and the intensity of the light beam and the background illumination [21, 22]. The left hand side of equation 4 follows from the experimental observation that the time courses for constriction and dilation can each be approximated by a single exponential [22]. The rate constants for pupillary movements differ for constriction (α_c) and dilation (α_d). The values of the constants τ, α_c, α_d, A_{on}, A_{off} can be readily determined from experimental measurements [23]. For equation 4 it has been possible to prove for simple initial conditions the existence of stable equilibria, of stable and unstable limit cycles, and Li and Yorke type chaos as well as mixing and exact motions as θ_1 and θ_2 are varied [20, 28].

The results in Figure 6 show that as the area thresholds are varied a variety of qualitatively different oscillations in pupil area are obtained. Experimentally obtained oscillations include simple limit cycles (Fig. 6c), more complex limit cycles (Fig. 6d and e) and aperiodic

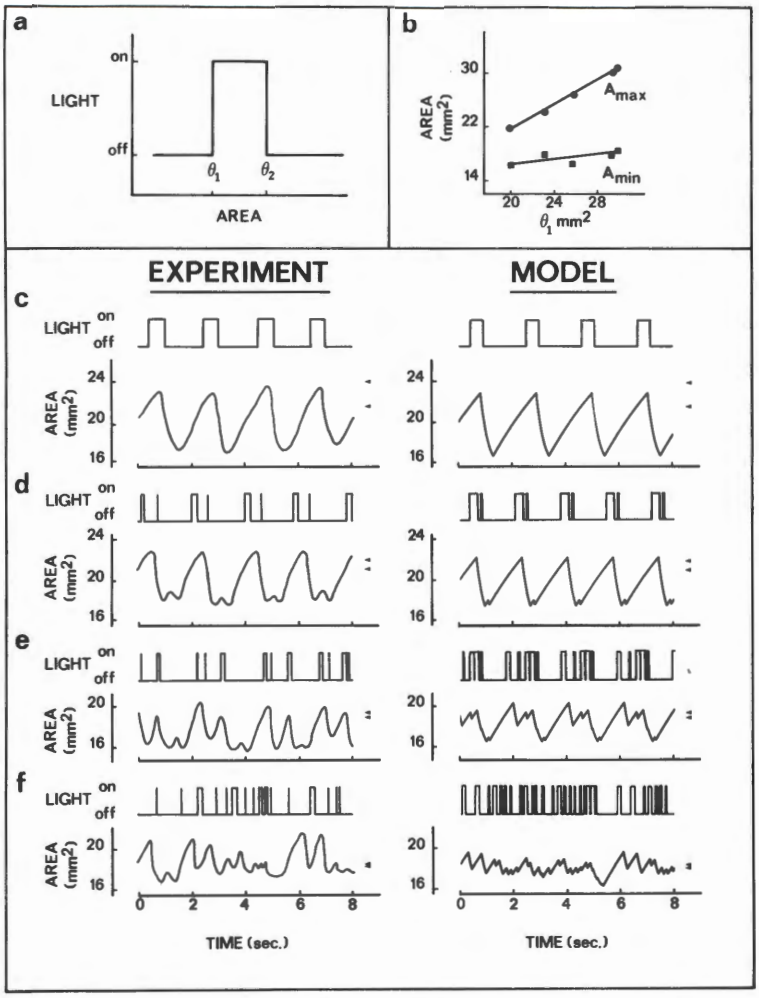

Fig. 6. Comparison of the changes in pupil area that occur as a function of time with imposed mixed feedback to those predicted by Eq. 4.
The piecewise constant mixed feedback is shown in (a). The parameters $\alpha_c = 3.88\ s^{-1}$, $\alpha_d = 0.265\ s^{-1}$, $A_{on} = 15.5\ mm^2$, $A_{off} = 34.2\ mm^2$ were obtained from a preliminary experiment with piecewise constant negative feedback which produces simple oscillations of the type shown in (c). The parameters are calculated from the slope and intercept of plots of the maxima and the minima of these oscillations as a function of the area threshold (data shown in b) [23]. The neural time delay is measured from the latency of the pupil response to a single pulse of light. The total delay in the experiment comprises this neural delay and the electronic processing delay (100 ms) and was 411 ms. The upper (θ_2) and lower (θ_1) area thresholds have been indicated by the " " at the right hand sides of the figure and were respectively: (c) 21.5 mm², 24.5 mm²; (d) 21 mm², 22 mm²; (e) 18.9 mm², 19.5 mm²; (f) 17.95 mm², 18.5 mm². After MILTON et al. [5]

oscillations (Fig. 6f). Qualitatively similar solutions are produced by equation 4 for the parameter values measured experimentally. The best agreement occurs for the simpler oscillations, e.g. Fig. 6c and d. However, although equation 4 correctly predicts that complex oscillations should be observed for certain choices of θ_1, θ_2 (Fig. 6f), the predicted oscillations are periodic and qualitatively clearly different from those seen experimentally.

It has been suggested that this discrepancy between model and experiment may reflect the influence of multiplicative noise [5, 22]. In other words, the region in parameter space over which complex dynamics would be expected to be observed is relatively small compared to the magnitude of the fluctuations in certain of the parameters. Thus complex dynamics, such as those shown in Fig. 6f, may reflect a combination of different types of solutions in adjacent regions of parameter space and transients resulting from the perturbations introduced by the noisy parameter fluctuations. Further, the observability of a given type of behaviour will depend not only on the area of parameter space over which it occurs, but also on the correlation time of the noise, i.e. on how fast the value of the noisy parameter(s) changes.

The pupil area oscillations shown in Fig. 6c have the same overall morphology as the corresponding solutions of equation 4, but possess less detail. This likely reflects the inability of the slowest elements of the reflex arc to undergo rapid, sudden changes in direction. One approach for including the elasto-mechanical properties of the iris and its musculature is to extend equation 4 to a second order delay-differential equation of the form

$$\frac{dA^2}{dt^2} + \beta \frac{dA}{dt} + \alpha A = G(A_\tau) \tag{5}$$

where the term $\frac{dA^2}{dt^2}$ represents a mechanical inertial force, $\beta \frac{dA}{dt}$ is a frictional, or damping, force, αA is a restoring force, and $G(A_\tau)$ is a delayed restoring force (equal to the right hand side of equation 4 for piecewise constant mixed feedback). Preliminary computer simulations indicate that the solutions of equation 5 are in better agreement with the oscillations shown in Fig. 6e and that the regions of parameter space over which more complex dynamics are observed are extremely narrow [29].

Equation 5 commonly arises in the description of the delayed feedback control of the movement of mechanical or neuromuscular systems [30]. Potential applications include the sensory feedback control of upper and lower limb motor prosthesis [31] and the remote control of robotic arms in space [32]. However, very little is known about the properties of the solutions of equation 5. The existence of periodic oscillations has been proved for the special case when $\beta = a + b$ and $\alpha = ab$, where a, b are positive constants [33]. When $\beta = 0$, the solutions of equation 5 can be constructed geometrically and reveal a remarkable richness of different types of dynamical behaviours including a variety of unconventional bifurcation schemes [34].

Discussion

The nervous system is capable of generating exceedingly complex dynamics. Recently emphasized examples include finger tapping of parkinsonian and normal subjects [35], neural spike trains recorded from sea slugs [36] and the pre- and post-central gyri of monkeys [37], and the electroencephalogram recorded from humans under a variety of conditions [38—46] and from the olfactory bulb of rabbits [47]. Several authors have suggested that these complex noise-like time series may be of deterministic origin and, in particular, chaotic in nature. This claim has been supported by the demonstration that the experimentally collected time series can be described by measures typically associated with the description of chaotic dynamical systems, e.g. fractal dimensions and positive Liapunov exponents.

Surprisingly, little attention has been directed towards the identification of neural mechanisms which would be capable of generating complex dynamics. Indeed it appears to have been implicitly assumed that complex dynamics are a consequence of the obvious anatomical complexity of the nervous system. Here we have pointed out that very simple neural networks are capable of generating complex dynamics. In other words, complexity of a time series does not necessarily imply an underlying complexity in neural mechanisms.

We have shown that simple neural networks which incorporate mixed feedback, e.g. recurrent inhibition, are capable of producing very complex dynamics. The fact that recurrent inhibitory loops are associated with virtually every neuron in the central nervous system [48] emphasizes their importance. The observation that the statistics of CA1 neural spike trains can be reproduced by a simple deterministic model for hippocampal recurrent inhibition indicates that complex neural time series can arise from relatively simple neural networks. The functional consequences of recurrent inhibition are typically discussed in the context of larger neural networks in which the inhibitory interneuron is capable of inhibiting a large number of neurons simultaneously. KANDEL and SCHWARTZ [49] have emphasized, for example, the possible role played by recurrent inhibition in shortening the output of a group of tonically active neurons and for highlighting the activity of those neurons which are relatively more activated. The tendency for recurrent inhibitory networks to produce complex dynamics does not appear to have been previously emphasized. In this sense recurrent inhibitory dynamics are quite distinct from the simple dynamics produced with negative feedback as in, for example, the pupil light reflex.

However, the experiments with the pupil light reflex clamped with mixed feedback suggest that complex neural dynamics can also arise because of the influence of noise. Thus, in general, the different components of the observed dynamics must be carefully identified and evaluated before the aperiodic behaviours generated by the nervous system can be confidently assigned a deterministic origin.

The pupil light reflex and a recurrent inhibition are examples of very simple neural networks, which, under appropriate circumstances, can generate complex dynamics. In both instances the anatomy and neurophysiology are well known. Thus it is possible to investigate the origins of the complex dynamics in greater detail than is possible, for example, with the electroencephalogram of the brain. An important aspect of this approach is the development of simple, but realistic mathematical models. However, physiological considerations suggest a number of extensions to existing models for which little is known. Examples include state-dependent delays (recurrent inhibition), second-order delay differential equations (pupil light reflex) and delay equations with distributed delays to account for a distribution of conduction velocities (pupil light reflex, recurrent inhibition). Moreover, since the ultimate aim of any modelling study is to compare prediction to experimental observation, it would seem to be prudent to eventually study the behaviour of these models in the presence of stochastic perturbations (additive and/or multiplicative) [29]. We expect that by the developing of appropriate mathematical models in conjunction with careful experimentation it should be possible to gain better insights into the generation of complex dynamical behaviours by the nervous system.

Acknowledgement

U. AN DER HEIDEN acknowledges support by Deutsche Forschungsgemeinschaft.

References

[1] MACKEY, M. C., L. GLASS: Science **197**, 287—289 (1977)
[2] MACKEY, M. C., U. AN DER HEIDEN: Funkt. Biol. Med. **1**, 156—184 (1982)
[3] MACKEY, M. C., J. G. MILTON: Ann. N.Y. Acad. Sci. **504**, 16—32 (1987)
[4] GLASS, L., M. C. MACKEY: From Clocks to Chaos: The Rhythms of Life. Princeton University Press, Princeton, New Jersey 1988
[5] MILTON, J. G., A. LONGTIN, A. BEUTER, M. C. MACKEY, L. GLASS: J. Theoret. Biol. **138**, 129—147 (1989)
[6] HOPPENSTEADT, F. C.: An Introduction to the Mathematics of Neurons. Cambridge University Press, Cambridge, Massachusetts 1986
[7] KILMER, W. L., T. MCLARDY: Int. J. Neurosci. **1**, 107—112 (1970)
[8] FELDMAN, J. L., J. D. COWAN: Biol. Cybern. **17**, 29—38 (1975)
[9] PLANT, R. E.: SIAM J. Appl. Math. **40**, 150—162 (1981)
[10] AN DER HEIDEN, U., M. C. MACKEY, H. O. WALTHER: Lect. Appl. Math. **19**, 355—360 (1981)
[11] MACKEY, M. C., U. AN DER HEIDEN: J. Math. Biol. **19**, 211—228 (1984)
[12] KACZMAREK, L. K.: Biol. Cybern. **22**, 229—234 (1976)
[13] KACZMAREK, L. K., A BABLOYANTZ: Biol. Cybern. **26**, 199—208 (1977)
[14] MATES, J. W. B., J. M. HOROWITZ: Comput. Programs Biomed. **6**, 74—84 (1976)
[15] TRAUB, R. D., R. MILLS, R. K. S. WONG: Science **243**, 1319—1325 (1989)
[16] MARCUS, C. M., R. M. WESTERVELT: Phys. Rev. A **39**, 347—359 (1989)
[17] HOROWITZ, J. M., W. J. FREEMAN, P. J. STOLL: Int. J. Neurosci. **5**, 113—123 (1973)
[18] RENSHAW, B.: J. Neurophysiol. **4**, 167—183 (1941)
[19] ECCLES, J. C., P. FATT, K. KOTETSU: J. Physiol. **126**, 524—562 (1954)
[20] AN DER HEIDEN, U., M. C. MACKEY: J. Math. Biol. **16**, 75—101 (1982)
[21] LONGTIN, A., J. G. MILTON: Bull. Math. Biol. **51**, 605 (1989)
[22] LONGTIN, A., J. G. MILTON: Math. Biosci. **90**, 183—199 (1988)
[23] MILTON, J. G., A. LONGTIN: Vision Res. **30**, 515—525 (1990)
[24] STARK, L.: Proc. IRE **47**, 1925—1939 (1959)
[25] MILTON, J. G., A. LONGTIN, T. H. KIRKHAM, G. S. FRANCIS: Am. J. Ophthalmol. **105**, 402—407 (1988)
[26] STARK, L.: J. Opt. Soc. Am. **52**, 925—930 (1962)
[27] REULEN, J. P. H., M. J. MARCUS, M. J. VAN GILST, D. KOOPS, J. E. BOS, G. TIESINGA, F. R. DE VRIES, K. BOSHUIZEN: Med. Biol. Eng. Comput. **26**, 27—32 (1988)
[28] AN DER HEIDEN, U. in: Delay Equations, Approximation and Application. pp. 117—134. G. MEINARDUS, G. NÜRNBERGER (Eds.). Birkhäuser, Basel 1985
[29] LONGTIN, A.: Ph. D. Thesis, McGill University, Montreal, Canada 1989
[30] MAYNARD SMITH, J.: Mathematical Ideas in Biology. pp. 98—104. Cambridge University Press, Cambridge, Massachusetts 1968
[31] PHILLIPS, C. A.: CRC Crit. Rev. Biomed. Eng. **16**, 311—438 (1988)
[32] BEJCZY, A. K., J. K. SALISBURY, JR.: Comp. Mech. Eng., July, 48—60 (1983)
[33] AN DER HEIDEN, U.: J. Math. Anal. Appl. **70**, 599—609 (1979)
[34] AN DER HEIDEN,U., LONGITIN, M. C. MACKEY, J. G. MILTON, R. SCHOLL: J. Dyn. Sys. Diff. Eqns. (1990, in press)
[35] KRAUS, P. H., H. R. BITTNER, P. KLOTZ, H. PRZUNTEK in: Temporal Disorder in Human Oscillatory Systems. pp. 102—109. L. RENSING, U. AN DER HEIDEN, M. C. MACKEY (Eds.). Springer-Verlag, New York 1987
[36] MITSOS, G. J., R. M. BURTON, H. C. CREECH, S. O. SOLINA: Brain Res. Bull. **21**, 529—538 (1988)
[37] RAPP, P. E., I. D. ZIMMERMAN, A. M. ALBANO, G. C. DEGUZMAN, N. N. GREENBAUM: Phys. Lett. **110A**, 335—338 (1985)
[38] BABLOYANTZ, A., C. NICOLIS, M. SALAZAR: Phys. Lett. **111A**, 152—155 (1985)
[39] ALBANO, A. M., N. B. ABRAHAM, G. C. DE GUZMAN, M. F. H. TARROJA, D. K. BANDY, R. S. GIOGGIA, P. E. RAPP, I. D. ZIMMERMAN, N. N. GREENBAUM, T. R. BASHORE in: Dimensions and Entropies in Chaotic Systems. pp. 231—240. G. MAYER-KRESS (Ed.). Springer-Verlag, New York 1987
[40] BABLOYANTZ, A. in: Dimensions and Entropies in Chaotic Systems. pp. 241—245. G. MAYER-KRESS (Ed.). Springer-Verlag, New York 1987
[41] BABLOYANTZ, A., A. DESTREXHE: Proc. Natl. Acad. Sci. USA **83**, 3513—3517 (1986)
[42] MAYER-KRESS, G., S. P. LAYNE: Ann. N. Y. Acad Sci. **504**, 62—87 (1987)

[43] MAYER-KRESS, G., J. HOLZFUSS in: Temporal Disorder in Human Oscillatory Systems. pp. 57—68. L. RENSING, U. AN DER HEIDEN, M. C. MACKEY (Eds.). Springer-Verlag, New York 1987
[44] RAPP, P. E., I. D. ZIMMERMAN, A. M. ALBANO, G. C. DE GUZMAN, N. N. GREENBAUM, T. R. BASHORE in: Nonlinear Oscillations in Biology and Chemistry. pp. 175—205. H. G. OTHMER (Ed.). Springer-Verlag, New York 1986
[45] WATT, R. C., G. MAYER-KRESS: Ann. N.Y. Acad. Sci. **504**, 286—288 (1987)
[46] NAN, X., X. JINGHUA: Bull. Math. Biol. **50**, 559—565 (1988)
[47] SKARDA, C. A., W. J. FREEMAN: Behav. Brain Sci. **10**, 161—195 (1987)
[48] CAJAL, R. S. y: Histologie du Systéme Nerveux de l'Homme et des Vertébrés. Vol. **I**. Malöine, Paris 1909
[49] KANDEL, E. R., J. H. SCHWARTZ: Principles of Neural Science. pp. 295—296. Elsevier/North-Holland, New York 1981