Complex Patterns of Oscillations in a Neural Network Model with Activity-Dependent Outgrowth

A. van Ooyen and J. van Pelt
Netherlands Institute for Brain Research,
Meibergdreef 33, 1105 AZ Amsterdam, The Netherlands

1 Introduction

Many processes that play a role in shaping the structure of the nervous system are modulated by electrical activity. For example, electrical activity can affect neurite outgrowth: high levels of activity, resulting in high intracellular calcium concentrations, cause neurites to retract, whereas low levels of activity, and consequently low calcium concentrations, allow further outgrowth [1]. As a result of this and other activity-dependent processes, a reciprocal influence exits between the formation of connectivity ("slow dynamics") and activity ("fast dynamics"). We have made a start at unravelling the implications of activity-dependent neurite outgrowth [2, 3], and have been able to show that several interesting properties arise as the result of interactions among outgrowth, excitation and inhibition: (i) a transient overproduction ('overshoot') during development with respect to connectivity; (ii) the neuritic fields of inhibitory cells tend to become smaller than those of excitatory cells; (iii) the spatial distribution of inhibitory cells becomes important in determining the level of inhibition; (iv) pruning of connections can no longer take place if the network has grown without activity for longer than a certain time ('critical period'). The results show many similarities with findings in cultures of dissociated cells.

Previously, we studied networks in which ϵ, the level of activity for which the neurites of a cell neither grow out nor retract [see eqn(3)], is the same for all cells. Here, we show that excitatory networks in which ϵ is distributed over a range of values can display complex patterns of oscillations in electrical activity and outgrowth. Oscillations in neurite outgrowth have indeed been observed in tissue cultures of hippocampal cells (S. B. Kater, personal communication).

2 The Model

We use a distributed, excitatory network, with neuron dynamics governed by

$$\frac{dX_i}{dT} = -X_i + (1 - X_i) \sum_j^N W_{ij} F(X_j) \tag{1}$$

where X_i is the membrane potential, N is the total number of cells, W_{ij} represents the connection strength ($W > 0$), and $F()$ is the firing rate:

$$F(u) = \frac{1}{1 + e^{(\theta - u)/\alpha}} \tag{2}$$

M. Marinaro et al. (eds.), *ICANN '94*
© Springer-Verlag London Limited 1994

where α determines the steepness and θ represents the firing threshold. Growing cells are modelled as expanding circular neuritic fields, and neurons become connected when their fields overlap. The outgrowth of each cell depends upon its own level of electrical activity:

$$\frac{dR_i}{dT} = \rho\left[1 - \frac{2}{1 + e^{(\epsilon_i - F(X_i))/\beta}}\right] \tag{3}$$

where R_i is the radius of the field, ρ is the rate of outgrowth, ϵ_i is the firing-rate at which $\frac{dR_i}{dT} = 0$, and β determines the non-linearity. Eqn(3) is just a description of Kater's hypothesis [1] that the depolarization level of the neuron influences its outgrowth. In the simulations we took $\theta = 0.5, \alpha = \beta = 0.5$, and ρ very small so that connectivity is quasi-stationary relative to membrane potential dynamics ($\rho = 0.0001$ in full model, and 0.005 in eqn(5)).

3 Results

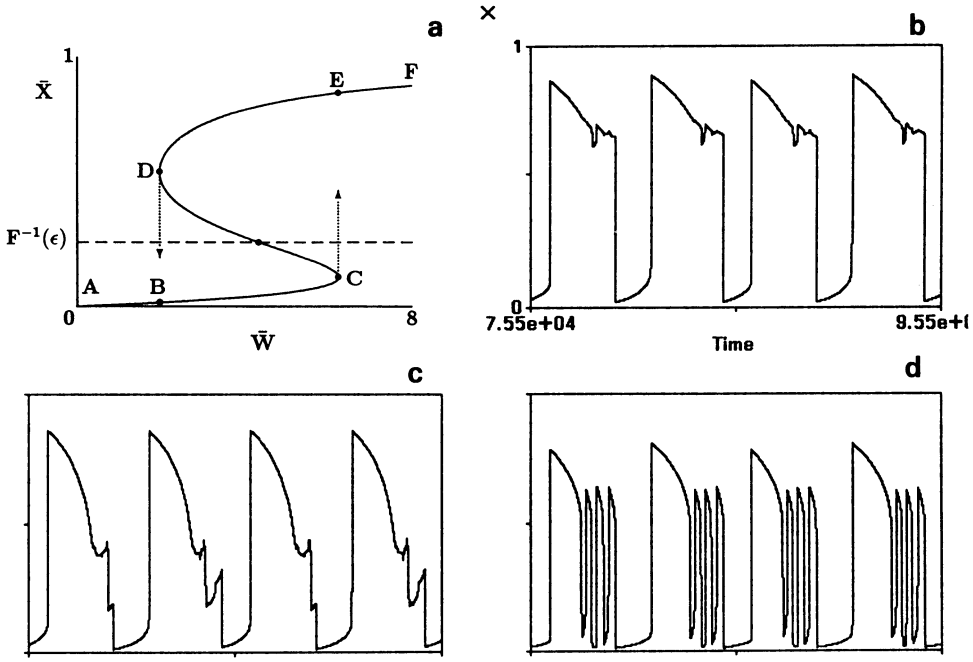

Fig.1 a Equilibrium manifold of $\bar{X}$ ($\frac{d\bar{X}}{dT} = 0$). **b,c,d** The membrane potential, X, of three different cells in a network in which ϵ is uniformly distributed between 0.3 and 0.8.

To understand the ocurrence of complex oscillations, first consider a network in which all the cells have the same ϵ. If the variations in X_i are small (relative to $\bar{X}$, the average membrane potential of the network), we find that for a given connectivity $\mathbf{W}$ the equilibrium points are solutions of:

$$0 \simeq -\bar{X} + (1 - \bar{X})\bar{W}F(\bar{X}) \tag{4}$$

This gives us the equilibrium manifold of $\bar{X}$ ($\frac{d\bar{X}}{dT} = 0$) as depending on the average connectivity $\bar{W}$ (Fig. 1a). The equilibria are stable on the branches ABC and DEF, and unstable on CD. The size of a neuritic field remains constant if $X_i = F^{-1}(\epsilon)$, where F^{-1} is the inverse of F; thus, since all cells have indentical ϵ, $\bar{W}$ remains constant if $\bar{X} = F^{-1}(\epsilon)$. An intersection point of this line on ABC (quiescent state) or DEF (activated state) of the manifold results in a stable point of the whole system, whereas an intersection point on DC results in oscillations following the path $ABCEDBCEDBC....$ These oscillations, in connectivity and activity, are similar for all cells. On the other hand, a network in which the cells have different ϵ values exhibits a complex pattern of oscillations: cells can differ in frequency, phase and amplitude, while other cells show no oscillations at all (Fig. 1b, c ,d).

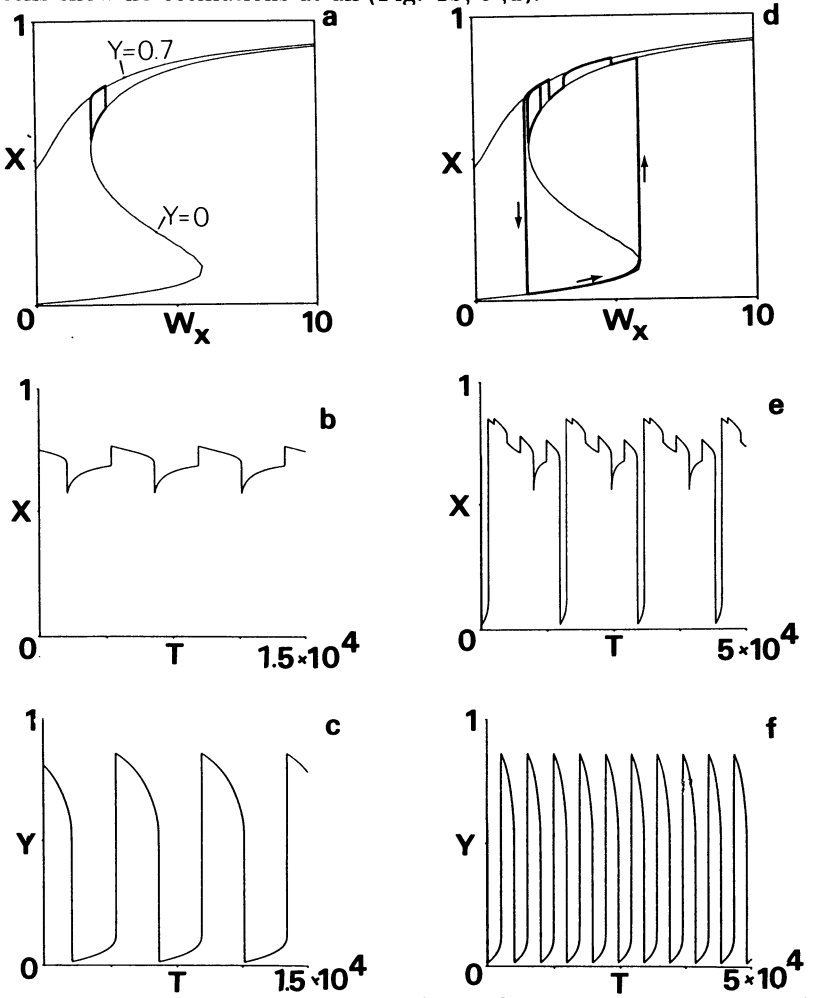

Fig.2 Behaviour of the simplified model [eqn(5)] with $\epsilon_Y = 0.4$ and in **a,b,c** $\epsilon_X = 0.7$ and in **d,e,f** $\epsilon_X = 0.685$. In **a,d** the manifolds of X for $Y = 0$ and $Y = 0.7$ are drawn together with the trajectory of X (thick line) (skipping the initial transients). In **b,c,d,f** X and Y are plotted against time.

Simplified models can be used to explain how these patterns can arise:

$$\begin{aligned} \frac{dX}{dT} &= -X + (1-X)(W_X F(X) + F(Y)) \\ \frac{dY}{dT} &= -Y + (1-Y) W_Y F(Y) \\ \frac{dW_X}{dT} &= \rho(\epsilon_X - X) \\ \frac{dW_Y}{dT} &= \rho(\epsilon_Y - Y) \end{aligned} \tag{5}$$

where X (W_X) and Y (W_Y) are the average membrane potential (connectivity) of two different cell populations which differ in their ϵ value. We assume that the influence of X on Y can be neglected and that the connection strength between X and Y is constant. The connectivity in both populations depends on their activity according to a simplified form of eqn(3). In the examples (Fig. 2) ϵ_Y is such that Y oscillates, and ϵ_X is such that, *without* the effect of Y, X goes to a stable point. Thus, Y oscillates independently of X between activated and quiescent state, and X is "forced" by Y. Depending on ϵ_X, X oscillates also between quiescent and activated state (possibly with a different frequency: Fig. 2e) or remains in the activated state (Fig. 2b). In Fig. 2 the equilibrium manifold of X as depending on W_X is drawn for Y at the quiescent state ($Y = 0$) and the activated state ($Y = 0.7$). As can be seen, X jumps between these two manifolds.

4 Conclusions

We have shown that complex patterns of oscillations in connectivity, and consequently in activity, can arise when cells grow depending on their own level of activity. The emergence of these oscillations (on the timescale of growth) hinges upon the presence of a hysteresis loop between activity and connectivity (Fig. 1a). Simplified models can help to understand the occurrence of oscillations of different forms and frequencies, in terms of trajectories on manifolds. Extensions of the simplified model presented here are readily possible.

5 References

[1] Kater SB, Guthrie PB, Mills LR (1990) Integration by the neuronal growth cone: a continuum from neuroplasticity to neuropathology. Progress in Brain Research 86: 117-128.

[2] Van Ooyen A, Van Pelt J (1993) Activity-dependant outgrowth of neurons and overshoot phenomena in developing neural networks. Journal of Theoretical Biology, in press.

[3] Van Ooyen A, Van Pelt J (1993) Implications of activity-dependent neurite outgrowth for developing neural networks. In: S. Gielen and B. Kappen (eds.), Proceedings of ICANN'93, pp. 177-182.