Neural Networks

Overview:
- Anatomy of Neuronal Networks
- Formal Neural Networks
- Are they realistic?
- Oscillations and Phase locking
- Mapping problem: Kohonen Networks

Nice books to start reading:

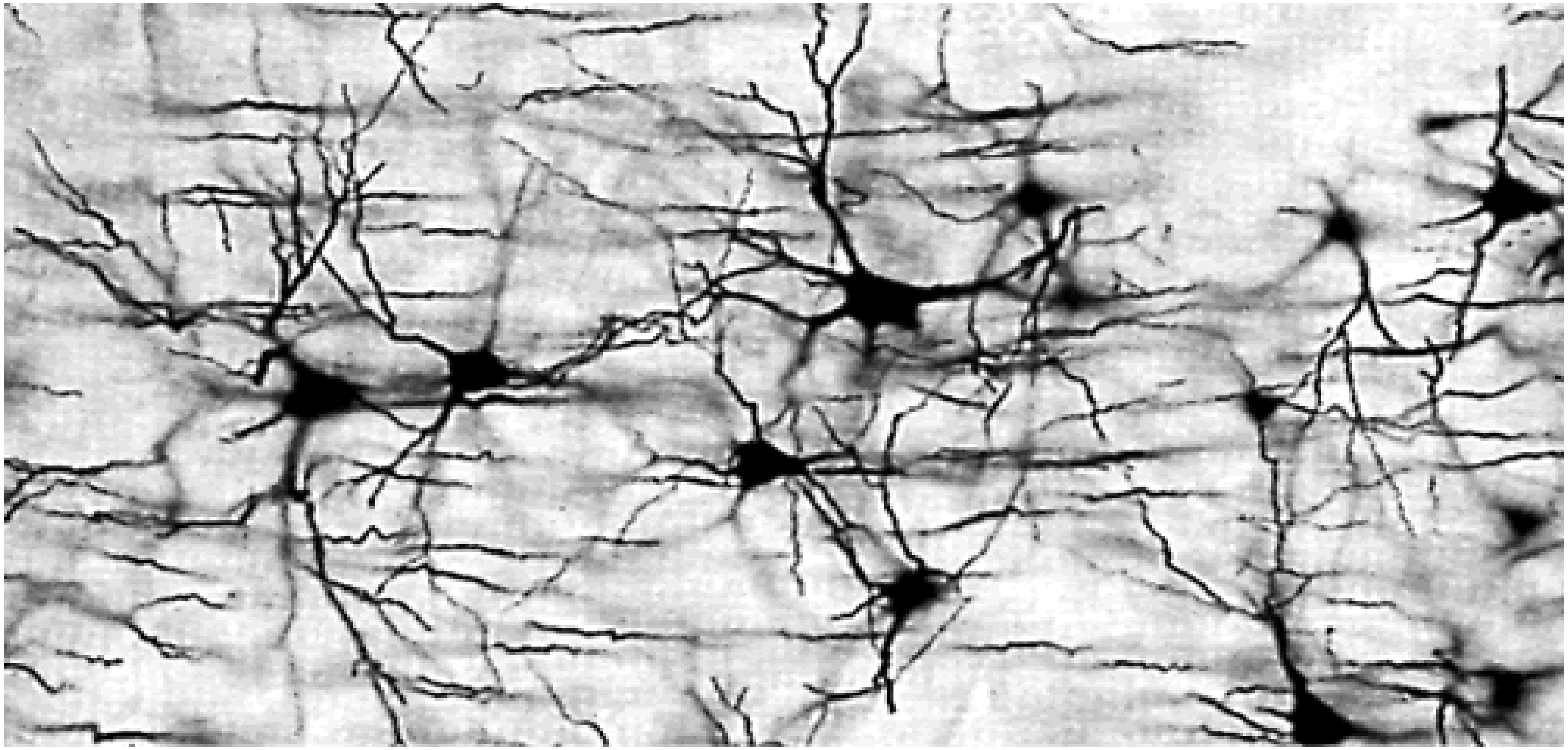
e.g. Manfred Spitzer: Geist im Netz

Brick-like text-books:

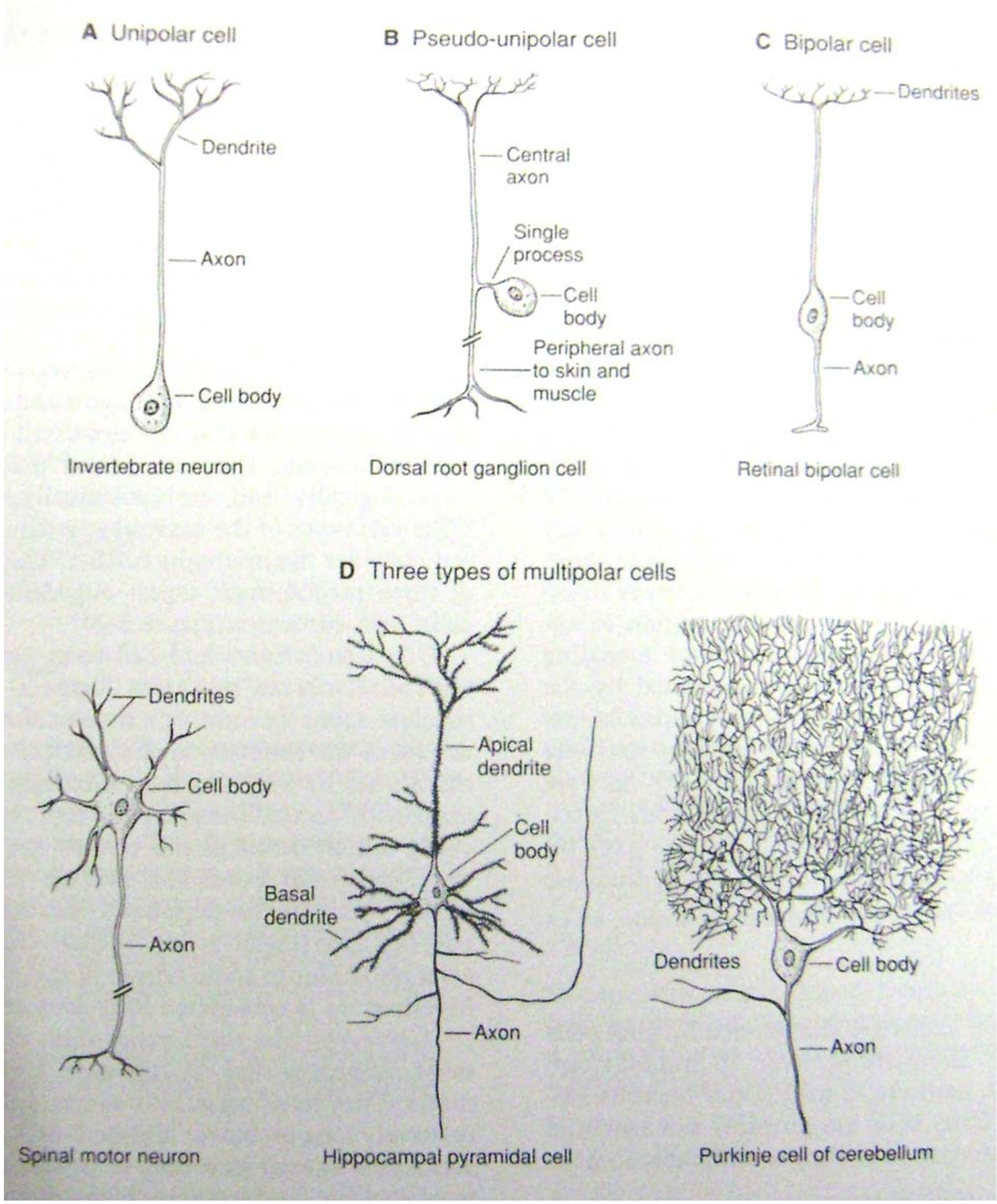
From Neuron to Brain by John G. Nicholls, John G. Nicholls, Bruce G. Wallace, Paul A. Fuchs, A. Robert Martin

Principles of Neural Science by Eric R. Kandel, James H. Schwartz, Thomas M. Jessell

Models of Neural Networks I-III, Domany, van Hemmen, Schulten, Springer 1991,1995



Neuroanatomy



From: Principles of Neural Science
Kandel, Schwartz, Jessel, 1991

The brain mostly consists NOT of neurons, there are about **10-50 times more glia** (greek: “glue”) cells in the central nervous tissue of vertebrates.

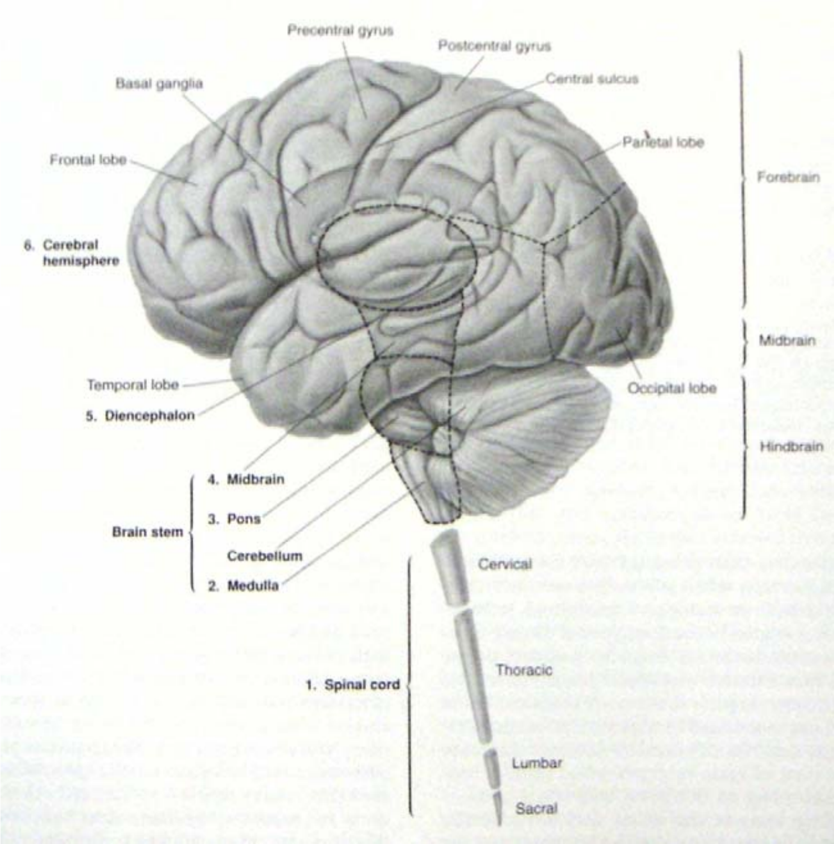
The function of **glia** is not understood in full detail, but their active role in signal transduction in the brain is probably small.

Electrical and **chemical synapses** allow for **excitatory** or **inhibitory** stimulation. They most often sit at the dendritic tree, but some also at the surface of a neuron.

In many neuron types, these inputs are can trigger **an action potential** in the **axon** which makes connections with other dendrites.

However, only recently, it was found, that action potentials also travel **back into the dendritic tree,** a crucial prerequisite for learning.

Neuroanatomy



The brain consists of about **10^{11}** neurons, divided into approx. **10,000 cell types** with highly diverse functions.

The cortex, the outer “skin” of the brain, appears to be very **similar** all over the brain, only more detailed analysis also shows here specialization in different regions of the cortex.

Most of the **brain volume are “wires”** in the white matter of the brain.

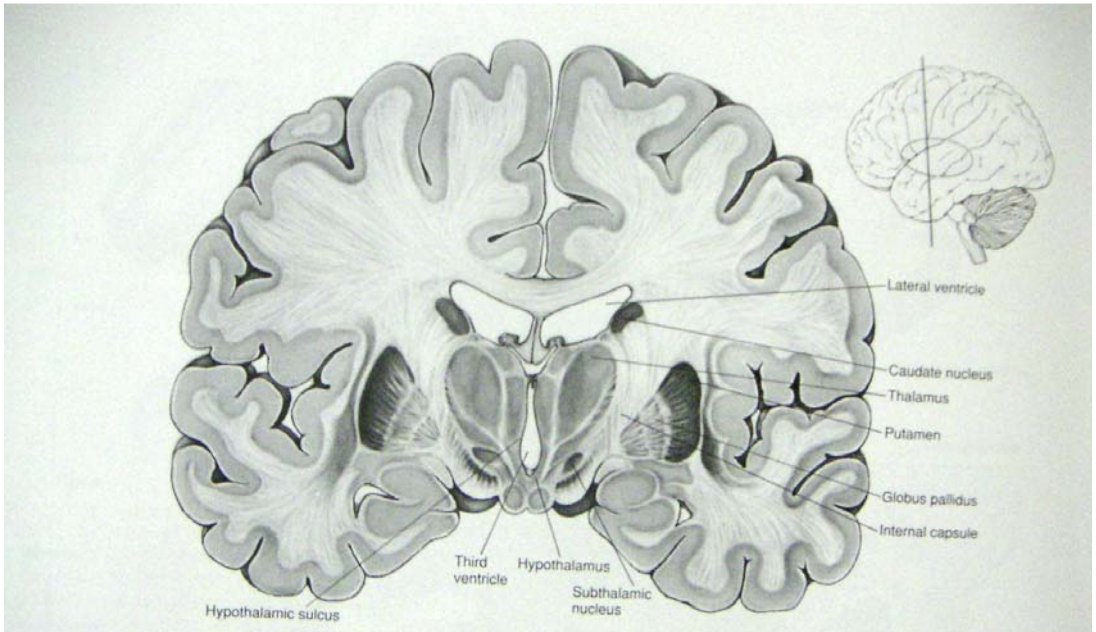
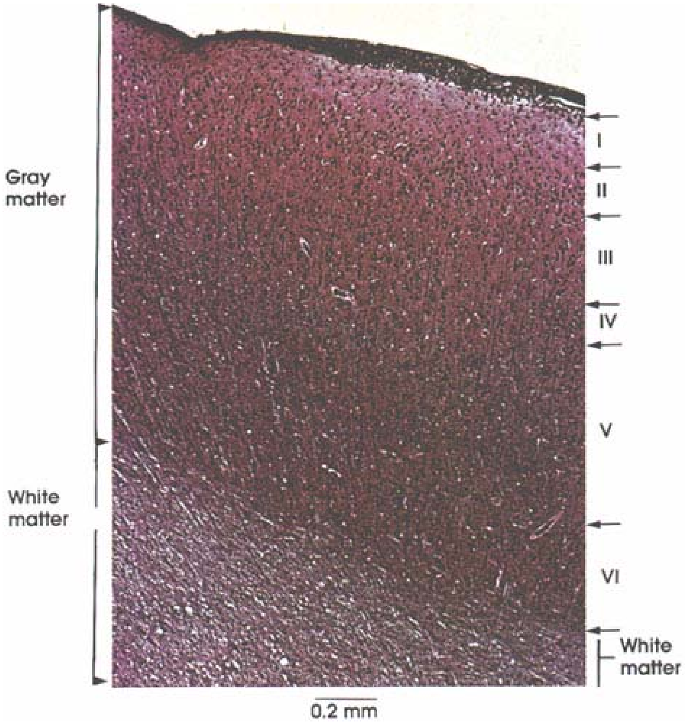


FIGURE 20–8
The thalamus is clearly visible in this coronal section through the diencephalon (the **inset** shows the plane of the section). The thalamus lies dorsal to the hypothalamus and forms the walls of the third ventricle. (Adapted from Nieuwenhuys, Voogd, and van Huijzen, 1981.)

From: Principles of Neural Science
Kandel, Schwartz, Jessel, 1991

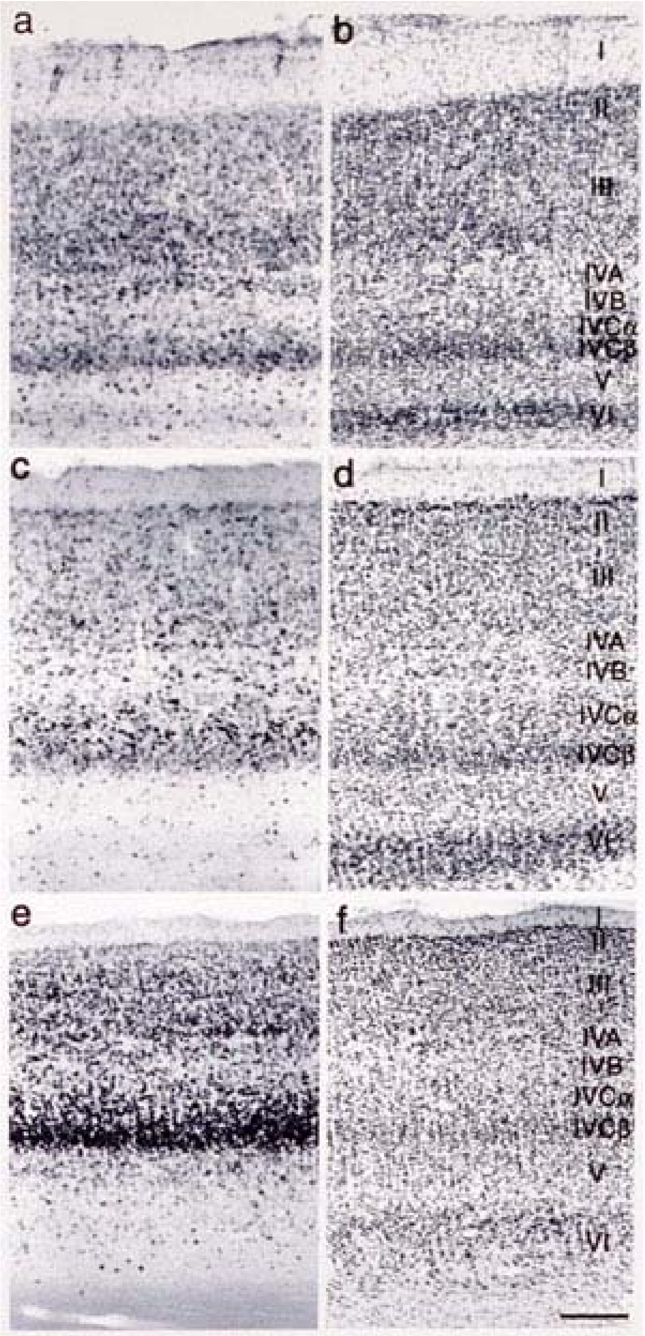


Cortex Layers

The Cortex is organized into layers which are numbered from I to VI.

Different types of cells are found in the layers.

The layer structure differs for different parts of the brain.



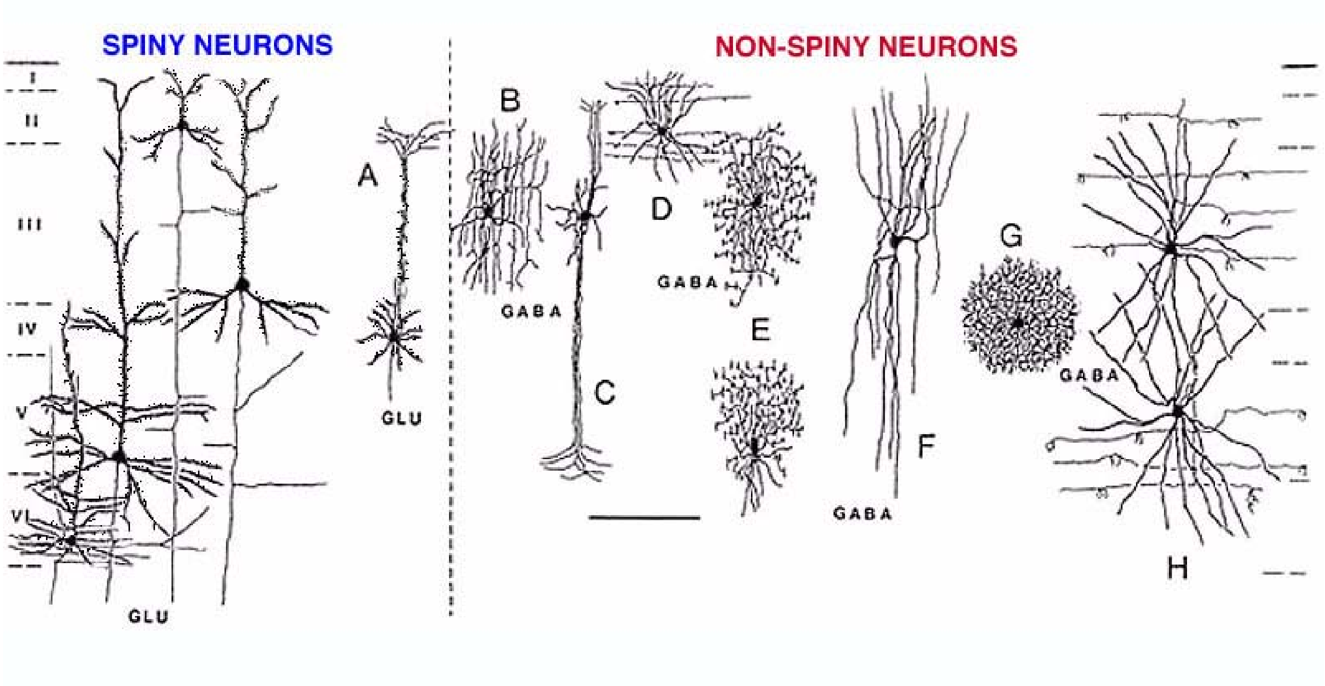
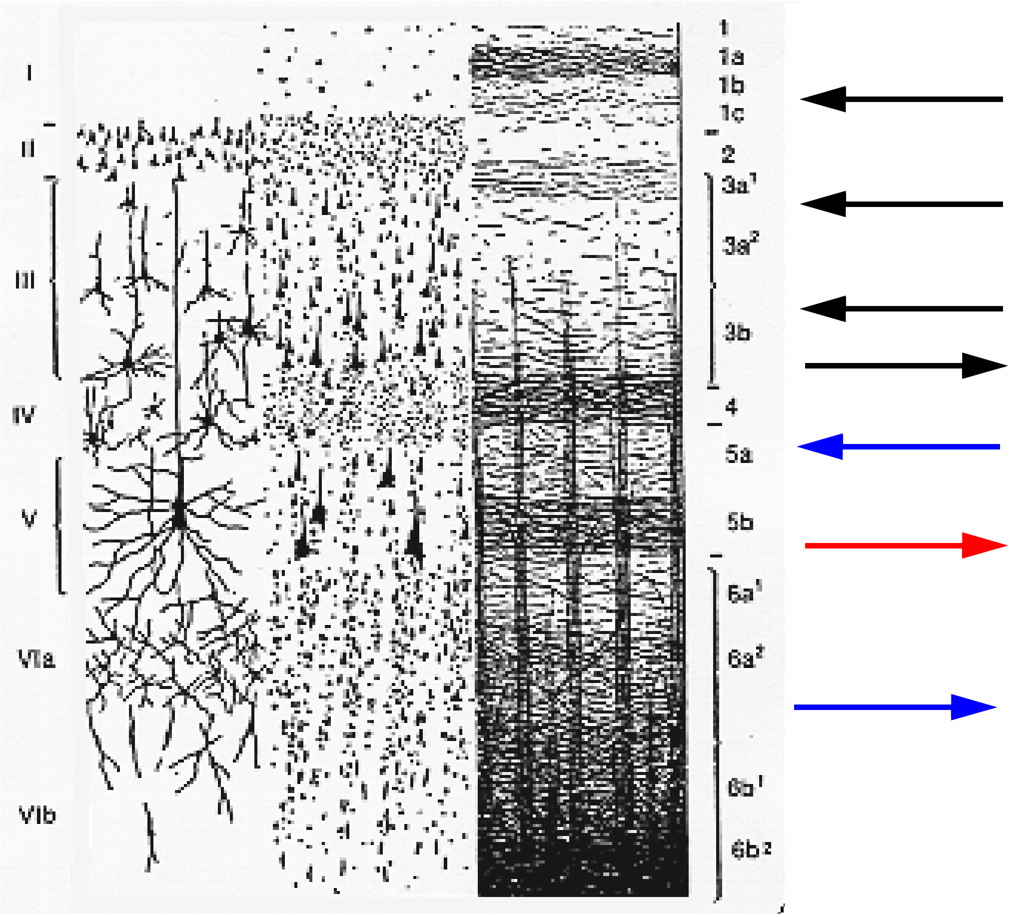


Figure 12. Basic cell types in the monkey cerebral cortex. Left: spiny neurons that include pyramidal cells and stellate cells (A). Spiny neurons utilize the neurotransmitter glutamate (Glu). Right: smooth cells that use the neurotransmitter GABA. B, cell with local axon arcades; C, double bouquet cell; D, H, basket cells; E, chandelier cells; F, bitufted, usually peptide-containing cell; G, neurogliaform cell.

Cortex Layers



Cortex

Cortex

Thalamus

Motor

Thalamus

I. Molecular layer: few scattered neurons, extensions of apical dendrites and horizontally oriented axons.

II. External granular layer: small pyramidal neurons and numerous stellate neurons.

III. External pyramidal layer: predominantly small and medium sized pyramidal neurons and non-pyramidal neurons.

I-III are main target and Layer III the principal source of of intercortical connections.

IV. Internal granular layer: stellate and pyramidal neurons. Main target from thamalus.

V. Internal pyramidal layer: large pyramidal neurons and interneurons. Source of motor-related signals.

VI. Multiform layer contains few large pyramidal neurons and many small spindle-like pyramidal and multiform neurons. Source of thalamus connections.

Neuronal Signals

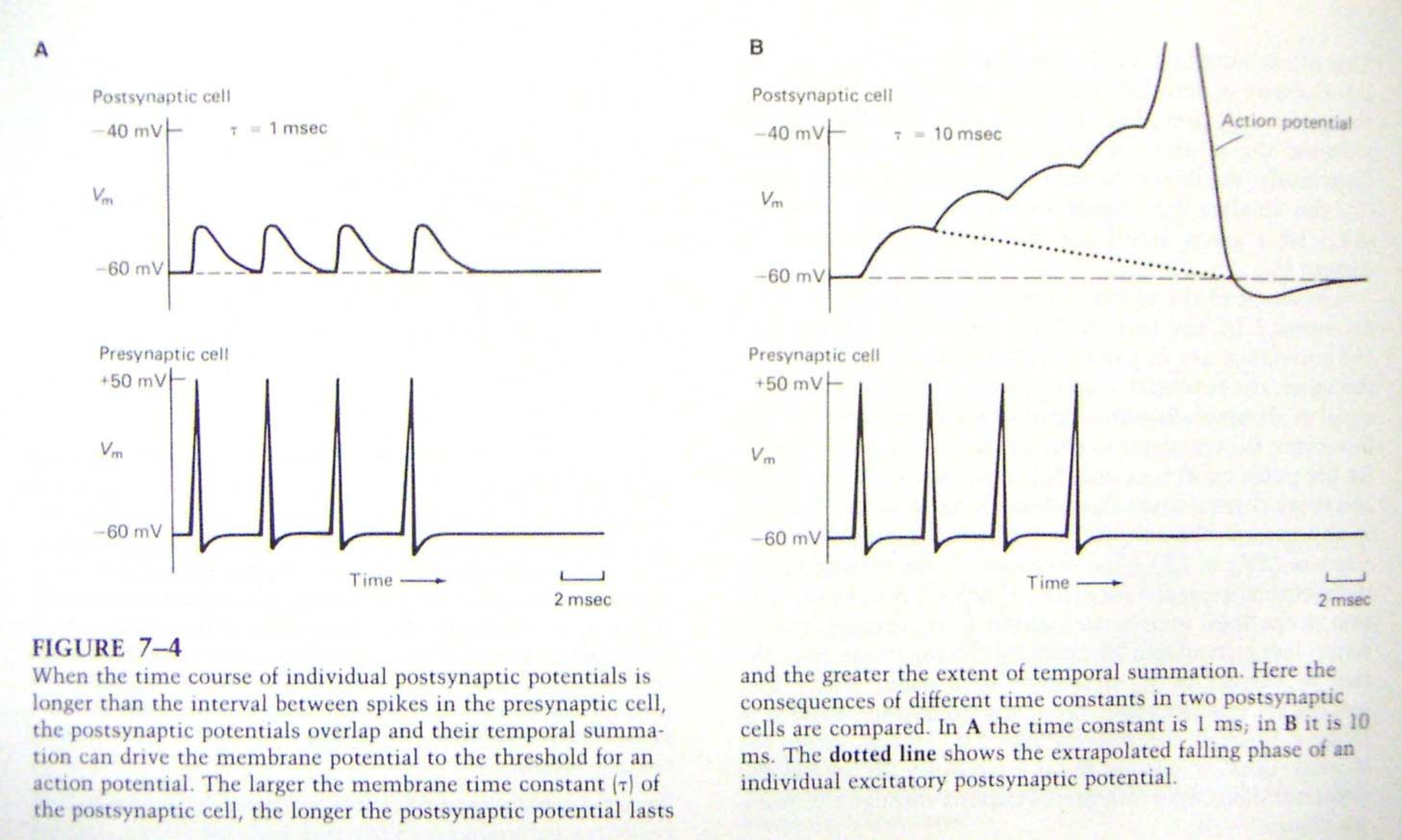


FIGURE 7–4
When the time course of individual postsynaptic potentials is longer than the interval between spikes in the presynaptic cell, the postsynaptic potentials overlap and their temporal summation can drive the membrane potential to the threshold for an action potential. The larger the membrane time constant (τ) of the postsynaptic cell, the longer the postsynaptic potential lasts and the greater the extent of temporal summation. Here the consequences of different time constants in two postsynaptic cells are compared. In **A** the time constant is 1 ms; in **B** it is 10 ms. The **dotted line** shows the extrapolated falling phase of an individual excitatory postsynaptic potential.

A typical **synapse** delivers about **10 - 30 pA** into the neuron. In many cases, this means that it increases the membrane voltage at the cell body by about **0.2-1 mV**.

Therefore, **many** synaptic inputs have to happen **synchronously** to trigger an action potential.

From: Principles of Neural Science
Kandel, Schwartz, Jessel, 1991

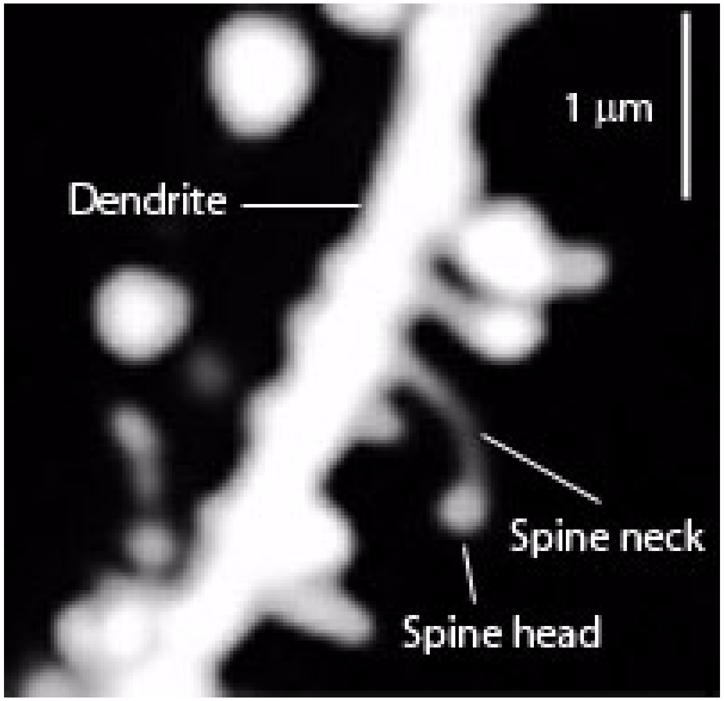
Dendritic Spines: Inputs for Synapses

16 *The Synaptic Organization of the Brain*

Fig. 1.7. The fine structure of a dendritic spine. This electron micrograph shows, at the bottom, a longitudinally cut dendrite from which a spine (s) arises. The spine is approximately 1.5 μm in length and 0.1 μm at its narrowest width. At its head it receives a synapse, which has the round vesicles and asymmetrical density characteristic of Gray's type 1. In the neck and head are small clumps of ribosomes; in the dendrite are longitudinally cut microtubules. [From Feldman, 1984, with permission.]

Excitatory synapses form often at spines which are bulges of dendritic membrane.

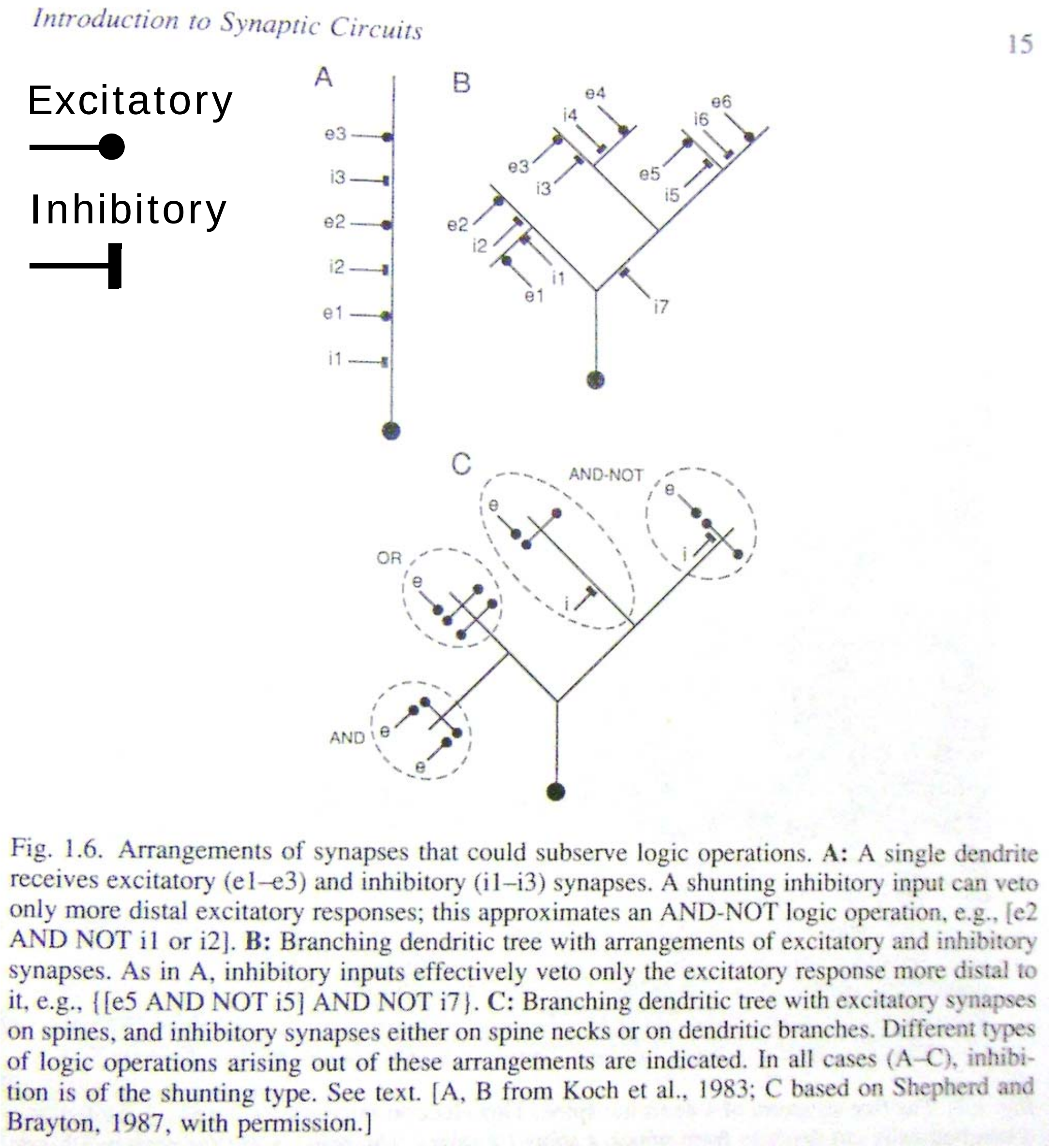
Although much is unknown, they probably act as local diffusion reservoir for Calcium signals and change their shape upon learning.



Dendritic Logics

Excitatory ─●

Inhibitory ─┃



Introduction to Synaptic Circuits 15

Fig. 1.6. Arrangements of synapses that could subserve logic operations. **A:** A single dendrite receives excitatory (e1–e3) and inhibitory (i1–i3) synapses. A shunting inhibitory input can veto only more distal excitatory responses; this approximates an AND-NOT logic operation, e.g., [e2 AND NOT i1 or i2]. **B:** Branching dendritic tree with arrangements of excitatory and inhibitory synapses. As in A, inhibitory inputs effectively veto only the excitatory response more distal to it, e.g., {[e5 AND NOT i5] AND NOT i7}. **C:** Branching dendritic tree with excitatory synapses on spines, and inhibitory synapses either on spine necks or on dendritic branches. Different types of logic operations arising out of these arrangements are indicated. In all cases (A–C), inhibition is of the shunting type. See text. [A, B from Koch et al., 1983; C based on Shepherd and Brayton, 1987, with permission.]

From: The Synaptic Organization of the Brain,
Gordon M. Shepherd 1998

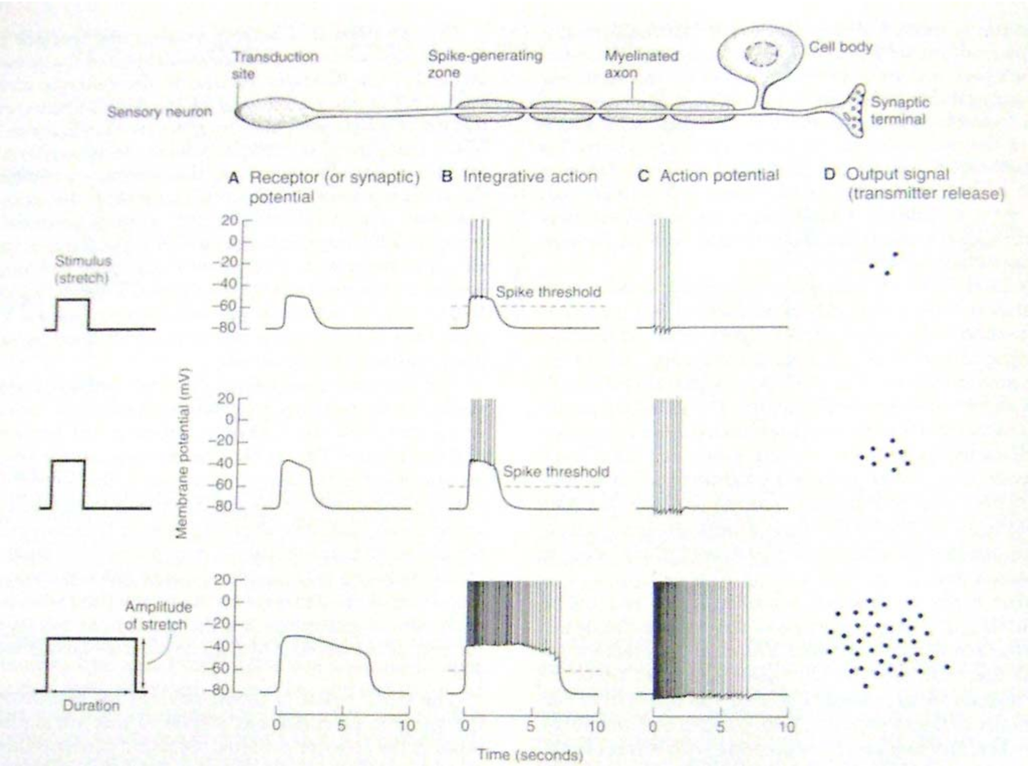
The **interplay** of currents along the dendritic tree can be **intricate** and allows the neuronal network to implement **various logical operations** (left):

A: Inhibitory synapses can **veto** more distal excitatory synapses: output = [e3 and not (i3 or i2 or i1)] or [e2 and not (i2 or i1)] or [e1 and not i1].

B: Branches can **overcome** the inhibitory effects. For example [e5 and not i5] and not i7.

So the assumption that a dendritic tree is a simple **addition** is very simplistic.

Sparse Firing

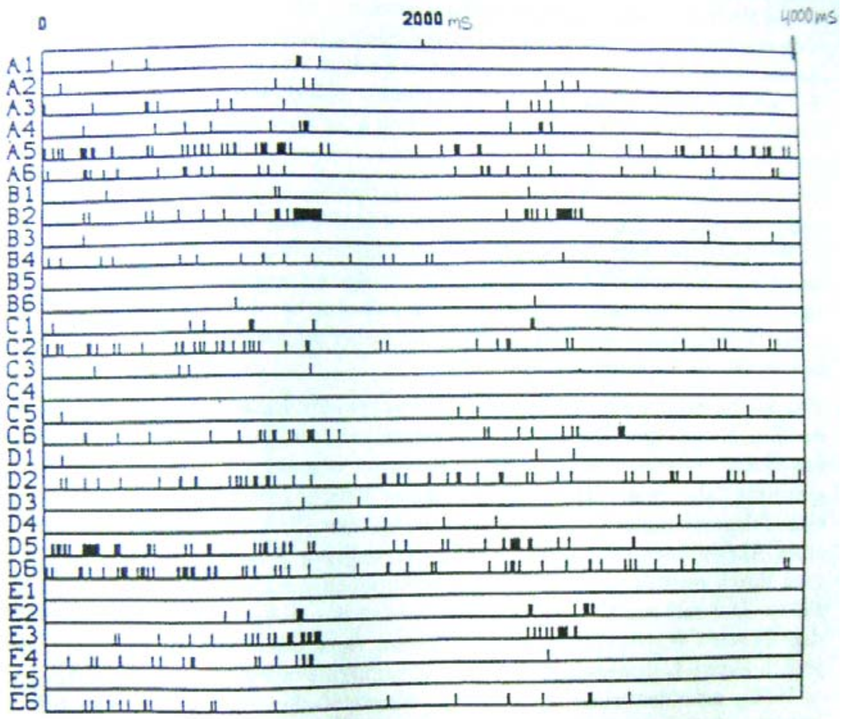


From: Principles of Neural Science
Kandel, Schwartz, Jessel, 1991

Experimentally, one can excite large **trains of action potential** (top).

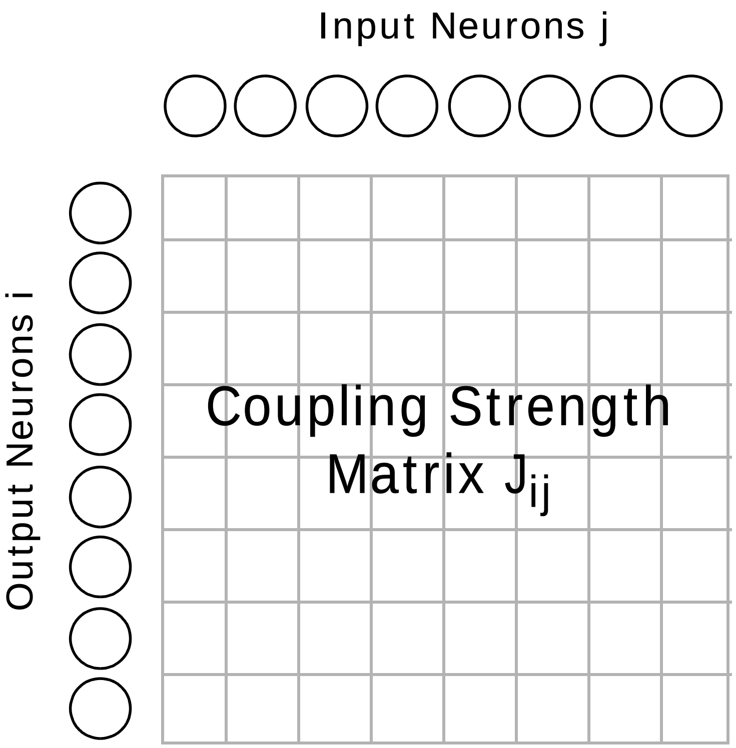
Thus, for long, the **average firing rates** were taken as main parameter of neural networks.

Fast spiking is not the normal mode of operation for most neurons in the brain. Typically, neurons fire **sparsely** where each action potential counts (below).



Figur 2.4: *Spike raster.* Dargestellt ist die Pulsfolge von 30 Neuronen (A1-E6) während der Stimulation mit einem bewegten Balken. Die Neuronen sind vertikal und die Zeit ist horizontal aufgetragen. Jedes Aktionspotential ist durch einen kurzen vertikalen Strich zum Zeitpunkt des Feuerns dargestellt. Die Gesamtdauer der Messung beträgt 4000 *ms* (Krüger und Aiple 1988).

Simple Model: Associative Memory



Input Neurons j

Output Neurons i

Coupling Strength Matrix J_{ij}

$$\overrightarrow{S_{OUT}} = t(\underline{J}\,\overrightarrow{S_{IN}})$$

$$t(h) = \mathrm{sign}[h]$$

Dynamics from OUT=IN

$$S_i(t+\Delta t) = \mathrm{sign}[h_i(t)]$$

McCulloch and Pitts simplified neuronal signalling to **two states**:

- Neurons i=1..N are either in state $S_i=-1$ or $S_i=+1$, i.e they are silent or fire an action potential

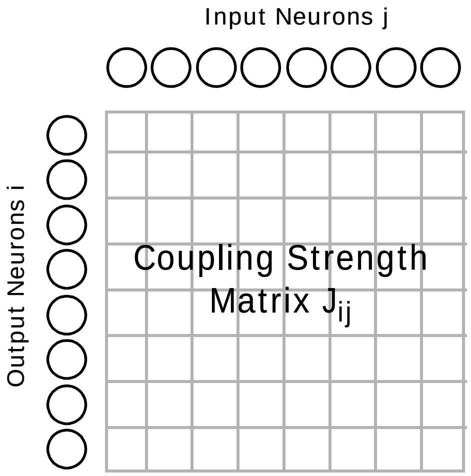
In the simplest model of an associative memory, the neurons are connected to themselves with a **coupling strength matrix J_{ij}**. It contains the “strength” or synaptic weight of the connections between the neurons.

Assume that the dendrites of neuron i only add the signals. The internal signal of the neuron h_i is then the matrix product of incoming neuronal states S_j according to $\mathbf{h_i=J_{ij}S_j}$ (sum over common indexes).

In the simplest form, neuron i fires if h_i is positive: $S_i=\mathrm{sign}[h_i]$.

This update can be performed with time lags, sequentially or in parallel and defines a dynamic of the neuronal net.

Simple Model: Associative Memory



Pattern μ for neuron i:

$$\xi_i^{\mu} = \pm 1$$

Probability for “+1”:

$$\frac{1 \pm a}{2}$$

Learning the Patterns with a Hebbian learning rule leads to:

$$J_{ij} = \frac{2}{N(1-a^2)} \sum_{\mu=0}^{q} \xi_i^{\mu}(\xi_j^{\mu} - a)$$

The dynamics has a number of defined fix points. By setting Jij, activity patterns can be memorized and dynamically retrieved.

You want to memorize the patterns ξ^{μ} into the network. The recipe to do this is reminiscient of an old postulate in neuroscience.

Hebb postulated in 1949: “*When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A’s efficiency, as one of the cells firing B, is increased.*”.

Both proportionalities are still present in the learning rule for J_{ij} on the left.

Simple Model: Associative Memory

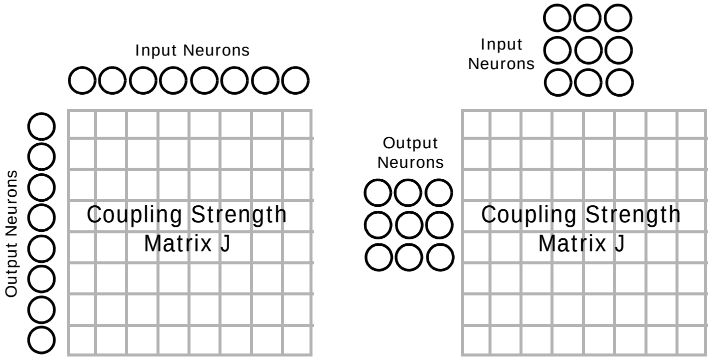


Fig. 1.12. The first eight letters of the alphabet, A–H (*top*), have been stored in a network of $N = 100$ formal neurons. After unlearning, corrupted versions (*middle*) with about 12% noise are presented to the network, which then retrieves the original patterns (*bottom*). Note that the retrieved patterns closely resemble but need not coincide with their prototypes

Capacity q of a fully connected network:

$$\mu = 1 \ldots q \qquad q \approx 0.14N$$

From: Models of Neural Networks I,
Domany, van Hemmen, Schulten, Springer 1995

Images of the size IxI are often used to show the memorizing capability of neural networks. Thus, the image is the pattern vector with length I^2 and the coupling strength matrix J has a size of I^2 x I^2.

For example we store 8 letters with I=10 using N=100 neurons and a coupling matrix of 100x100 weights. The retrieval from highly noisy input is possible, but shows some artefacts (F,G).

Retrieval is performed by starting at the noisy pattern, following the neuronal update dynamics to its fixpoint.

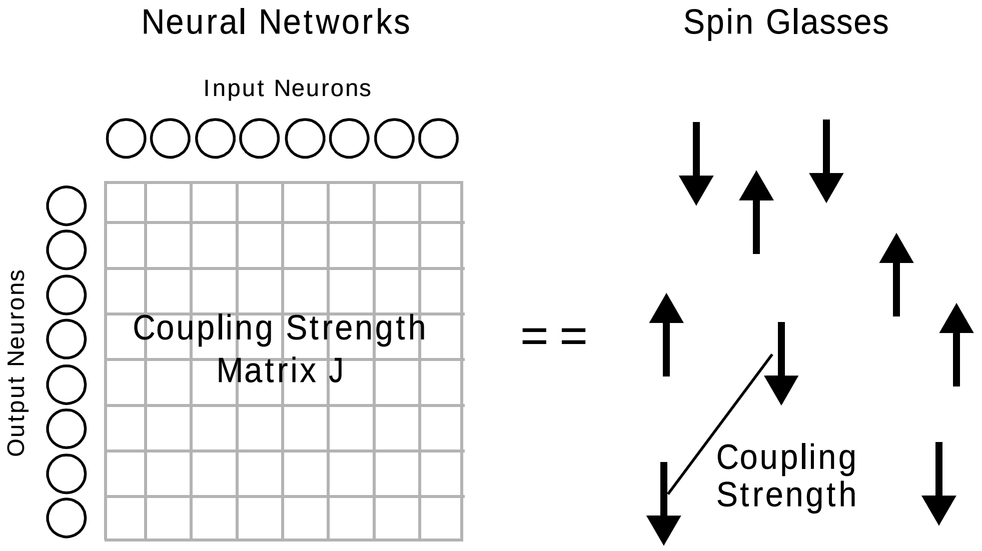
The capacity of a fully connected formal neural network scales with N. The number of patterns which can be stored is about 0.14xN. Thus in above network we can store about 9 letters.

An associative memory with the same number of synapses (10^{15}) than the brain could save $0.14 * 10^{7.5} = 5 x 10^6$ different patterns.

But the connections in the brain are much more complex.

Hopfield-Analogy to Spin Glasses

Hopfield:



Hamilton-Operator for Spin Glasses

$$H = -\frac{1}{2}\sum_{i \neq j} J_{ij} S_i S_j$$

J.J. Hopfield showed 1982 that formal neural networks are **analogous to spin glasses**.

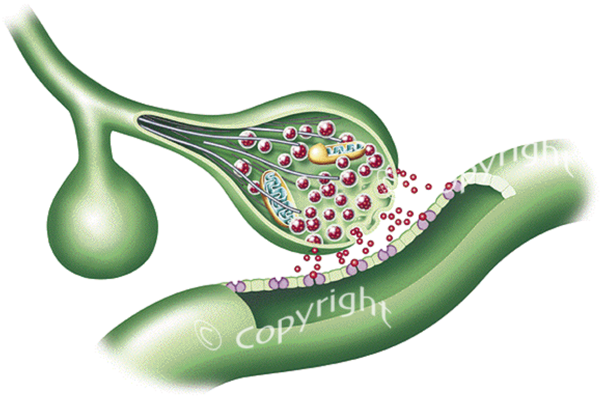
A spin glass is an amorphous material which fixes **spins** in a **3D matrix**. The spins can be oriented up or down.

The **magnetic field** from each spin influences the other spins. This **"crosstalk"** between spins is described by a coupling strength matrix J.

Such a spin glass is described by the **Hamilton operator H** to the left. The fixpoints are now simply the **ground states** of the system to which the dynamics converge.

The analogy made neuronal networks more accessible to theoretical physicists.

Towards realistic neurons

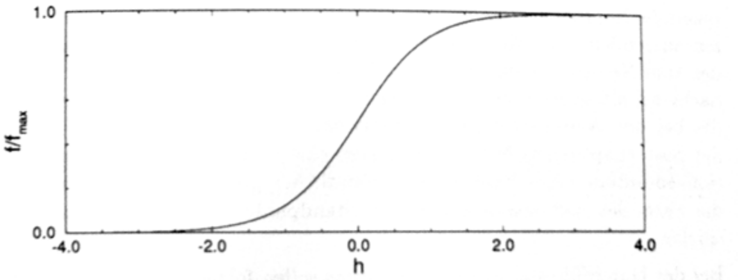


Deterministic:

$$\overrightarrow{S_{OUT}} = t(\underline{J}\,\overrightarrow{S_{IN}}) \qquad t(h) = \mathrm{sign}[h]$$

With Randomness:

$$\mathrm{Prob}[t(h)] = \frac{1 - \tanh[\beta h - \Theta]}{2}$$



Figur 2.11: *Gewinnfunktion eines Neurons im Standardmodell.* Im zeitlichen Mittel ist die Feuerrate eines Neurons, das durch einen konstanten Stimulus h angeregt wird, durch die Funktion $f(h) = (f_{max}/2)[1 + \tanh(\beta h)]$ gegeben, vgl. Gleichung (2.8). Das Standardmodell setzt voraus, daß die mittlere Feuerrate $f(h)$ genügt, um ein Neuron zu charakterisieren.

From: Gerstner, Ritz, van Hemmen,
Biol. Cybern. 68,363-374 (1993)

Synapses of real neural networks show intrinsic **noise**. For example, chemical synapses either release a synaptic vesicle, or they don't (**"quantal" noise**).

It is implemented into neuronal networks with a **probabilistic function of t(h)** with t being the probability to find the output neuron in the state $S_i = +1$.

As expected, noise does not change the property of neural networks dramatically.

As everywhere in biophysics, the inclusion of noise in a model is a good test for its robustness.

Towards realistic neurons
Sparse Firing and Oscillations

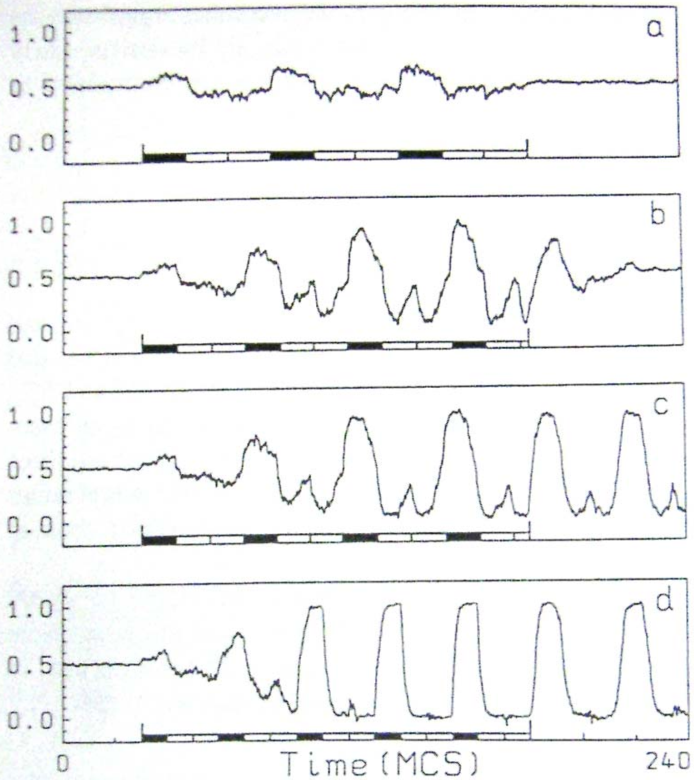


Fig. 1.9a–d. An external stimulus triggers the internal dynamics of a network with a broad, here uniform, *distribution* of delays between 0 and 30 Monte Carlo steps per spin (MCS). The network has been taught a cycle consisting of three patterns, each lasting $\Delta = 10$ MCS; the learning rule used was (4.1). Both the 3-cycle and the 3-symmetric state ($m_1 = m_2 = m_3 = 0.5$) are stable. The system starts in the latter and is triggered during $30 \leq t \leq 180$ MCS by an external signal which is the original cycle *distorted in time*; see (4.16). The external signal is weak: $\gamma = 0.20$. From top to bottom (**a** – **d**) we have a frequency distortion by factors 0.6, 0.75, 0.77, and 1.0, compared to the original frequency, which is d. Each track represents the overlaps $m_1(t)$ with the first pattern of the cycle. If in a and b one presented the distorted cycle to the network within a period of time beyond $t = 180$, then the response would no longer change

Until now, we have assumed **instantaneous propagation of signals** in neural networks. This is not the case and typical delays are on the 5-20ms time scale.

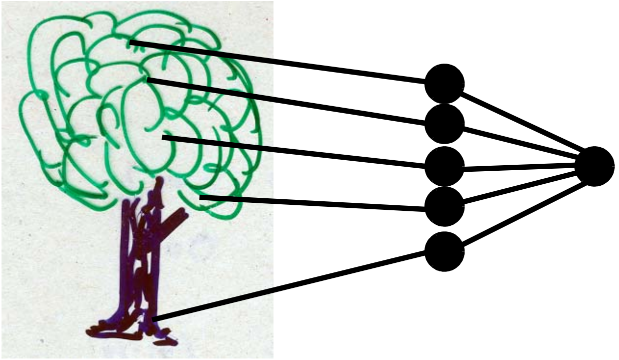
Delays leads to new dynamics of the network and can trigger **oscillations** (left).

We will discuss a compelling model which uses these delays in the following.

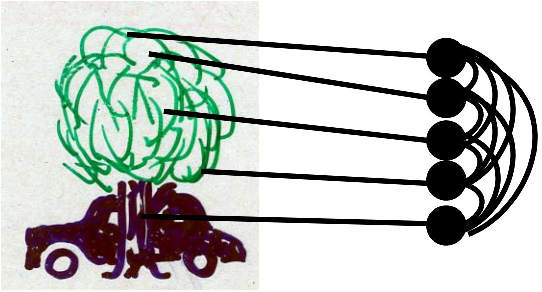
From: Models of Neural Networks I,
Domany, van Hemmen, Schulten, Springer 1995

Phase Locking and Pattern Recognition

A. Recognition by “grandmother cell”



B. Recognition by Cell Groups



Superposition
Catastrophy

One old theory of pattern gecognition is the so called “**grandmother cell**” proposal. It assumes that partial patterns converge to one cell and if that cell fires, the grandmother is seen. However this approach has severe problems:

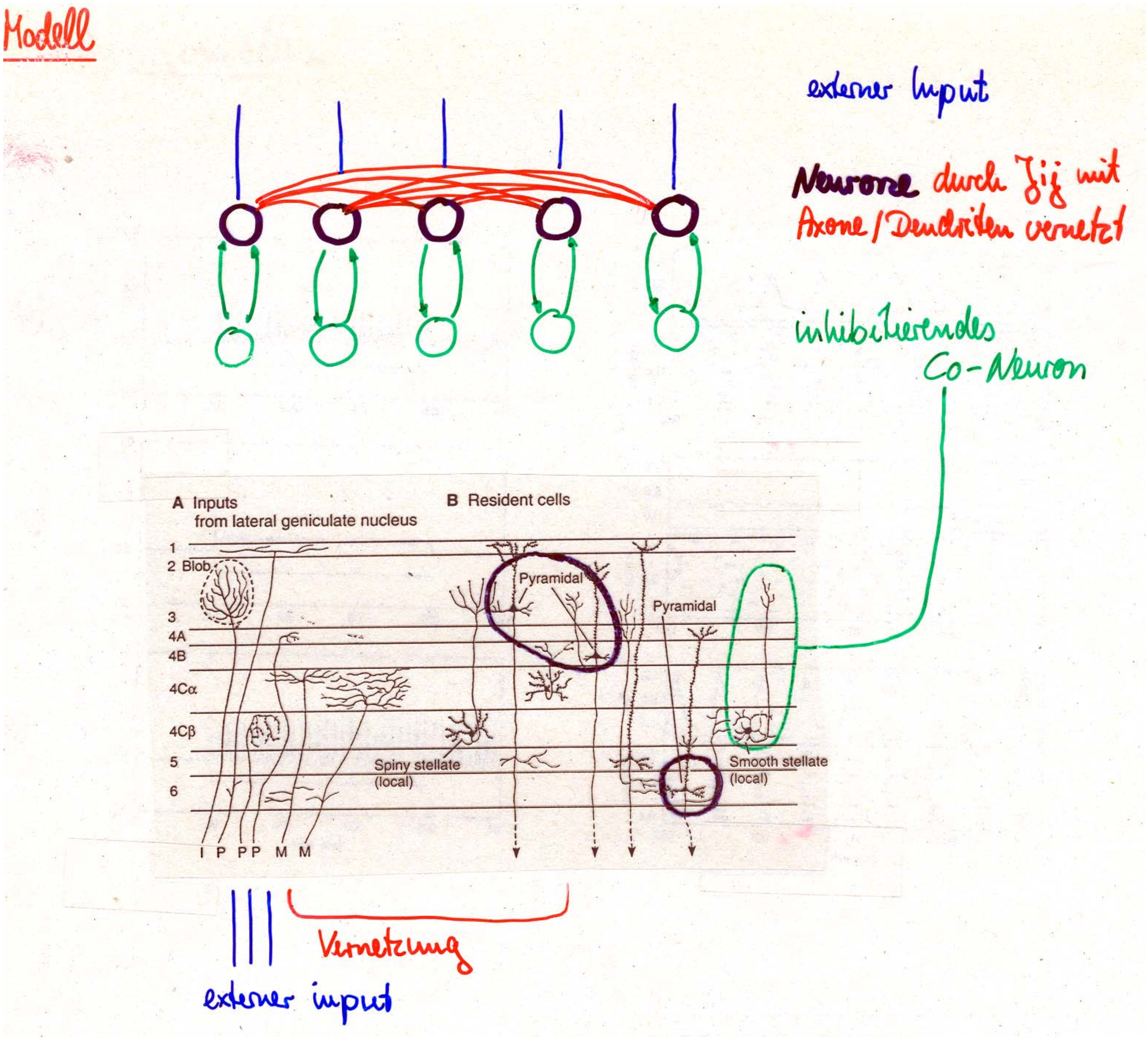
- What happens if this cell dies?
- Not much experimental evidence
- “**Combinatorical Explosion**”: any combination of patterns would require a novel grandmother cell, much more than even the brain can have.

The detection of patterns by cell groups as associated memory does not have that problem. Noisy signals can still be detected and the model is robust against death of single cells.

However there are two major problems:

- How should the pattern be **read out**?
- “**Superposition catastropy**”: a superposition of patterns is not recognized since it acts as novel pattern.

Phase Locking and Pattern Recognition



A biologically motivated and analytically soluble model of collective oscillations in the cortex.
Gerstner W, Ritz R, van Hemmen JL, Biol Cybern. 1993;68(4):363-74.

Phase Locking and Pattern Recognition

Das Neuron i

Zustand: $S_i = 1$: Neuron feuert gerade Aktionspotential (1ms lang)

$= 0$: Neuron ruhig

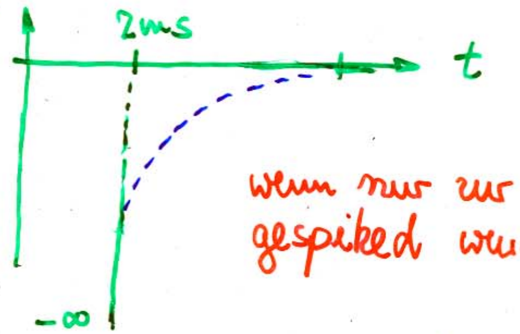
$h_i(t)$: Membranpotential am Axonhügel.

$$h_i(t) = h_i^{syn}(t) + h_i^{ref}(t) + h_i^{ext}(t)$$

Potential durch die Synapsen

externes Signal über extra Synapsen

$h_i^{ref}(t)$



wenn nur zur Zeit $t=0$ gespiked wurde

Überlagerung der h^{ref} aller älteren Spikezeitpunkte

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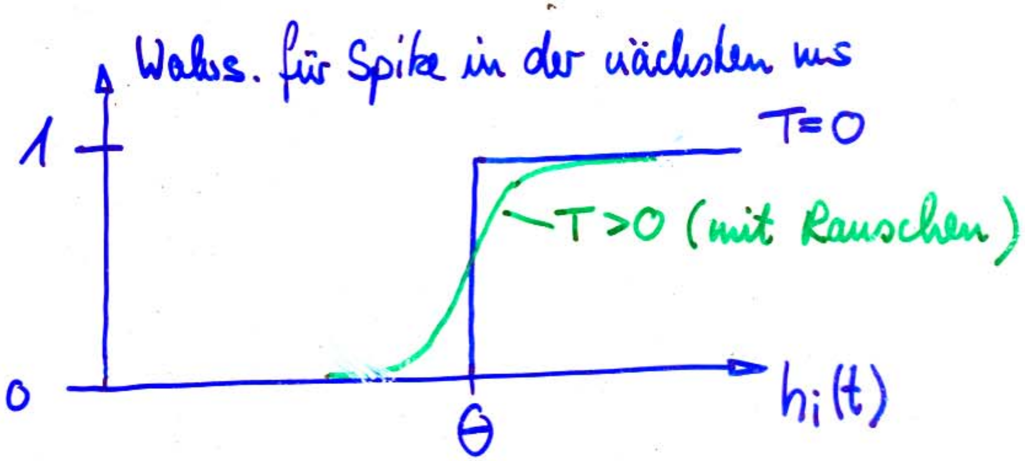
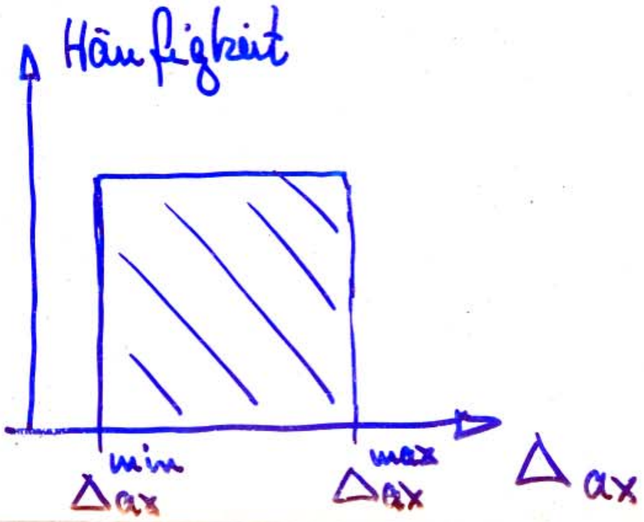
Phase Locking and Pattern Recognition

Auslösen eines Spikes



Axone

haben eine zufällig verteilte Verzögerungszeit Δ_{ax}



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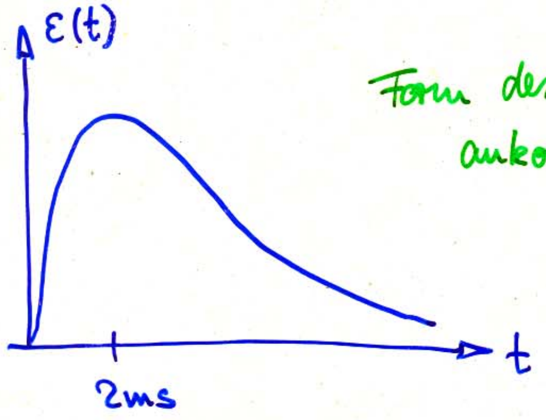
Phase Locking and Pattern Recognition

Synapsen

Ankommendes Aktionspotential löst postsynaptisches Potential aus – je nach Effektivität J_{ij} unterschiedlich groß. (j: Neuron, von dem AP kommt)



Form des PSP, wenn AP bei $t=0$ ankommt.

Die PSP's summieren im Dendritenbaum linear und „ohne Verzögerung" (d.h. Verzögerung wird in Axonverz. Δ_{ax} gepackt):

$$h_i^{syn}(t) = \sum_{\text{Neurone } j} J_{ij} \cdot \sum_{\text{Vergangenheit } s} \varepsilon(s)\, S_j(t-s-\Delta_j^{ax})$$

Form des PSP

=1, wenn damals AP ausgelöst

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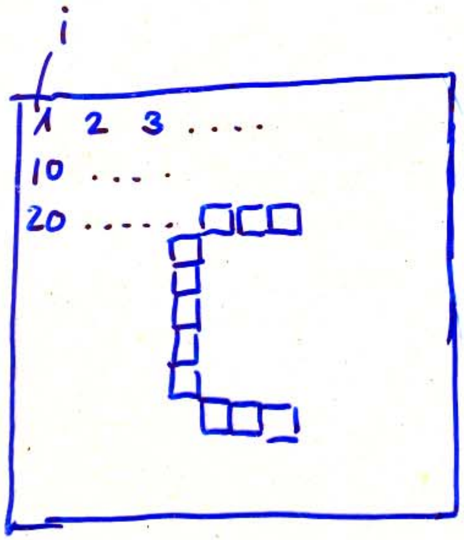
Phase Locking and Pattern Recognition

Muster



In den synaptischen Gewichten können vers. Muster abgelegt werden (assoz. Speicher)

Überlapp

gibt an, wie sehr der Feuerzustand der Neurone einem der Muster entsprechen:

$$m^{\mu} = \begin{cases} 1 & S_i \text{ stellen Muster dar} \\ 0 & \text{Muster nicht dargestellt} \end{cases}$$

Musternummer

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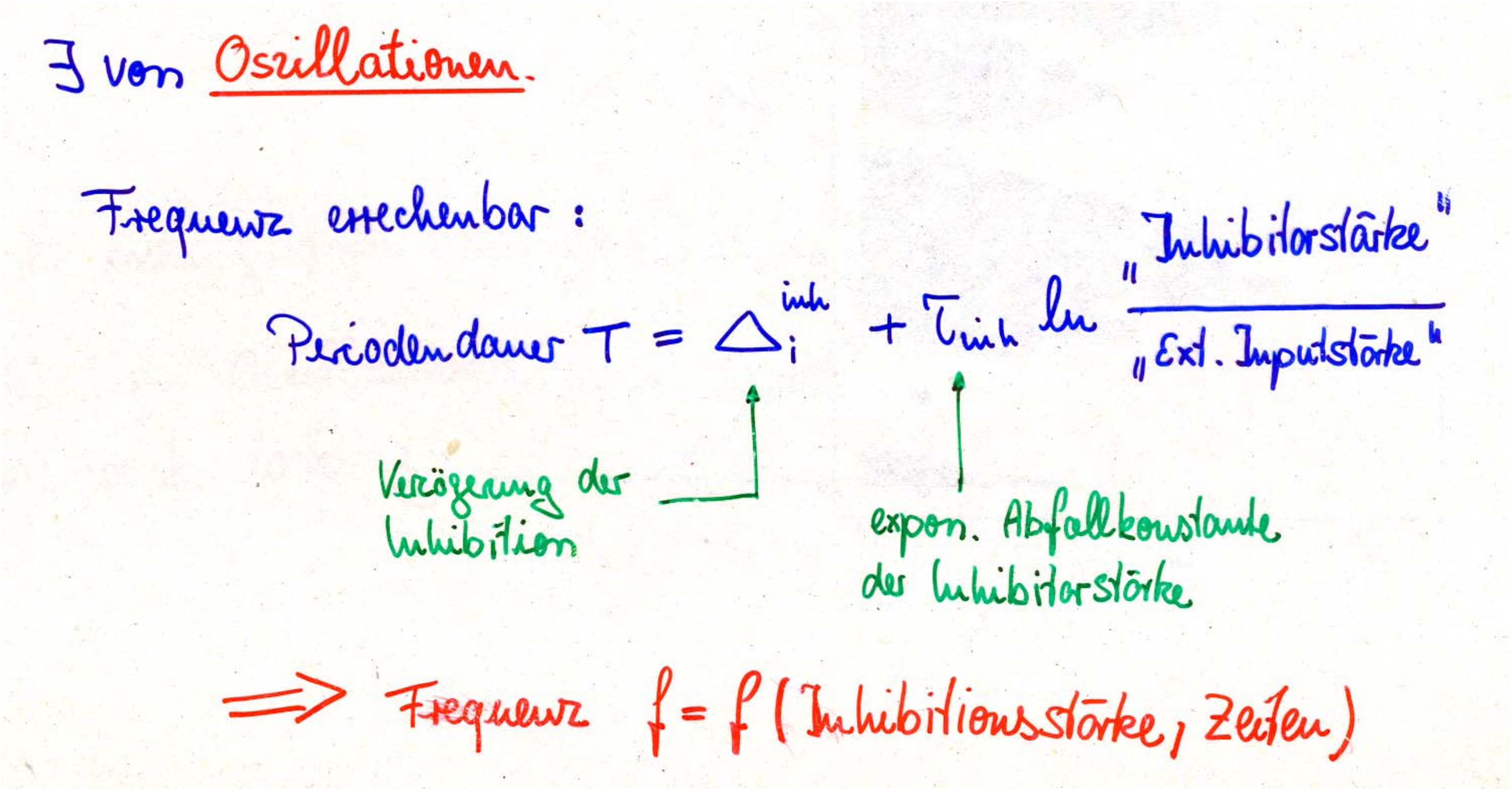
Phase Locking and Pattern Recognition



∃ von Oszillationen.

Frequenz errechenbar :

$$\text{Periodendauer } T = \Delta_i^{inh} + \tau_{inh} \ln \frac{\text{„Inhibitorstärke“}}{\text{„Ext. Inputstärke“}}$$

Verzögerung der Inhibition

expon. Abfallkonstante der Inhibitorstärke

$\Longrightarrow$ Frequenz $f = f(\text{Inhibitionsstärke, Zeiten})$

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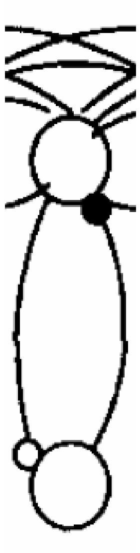
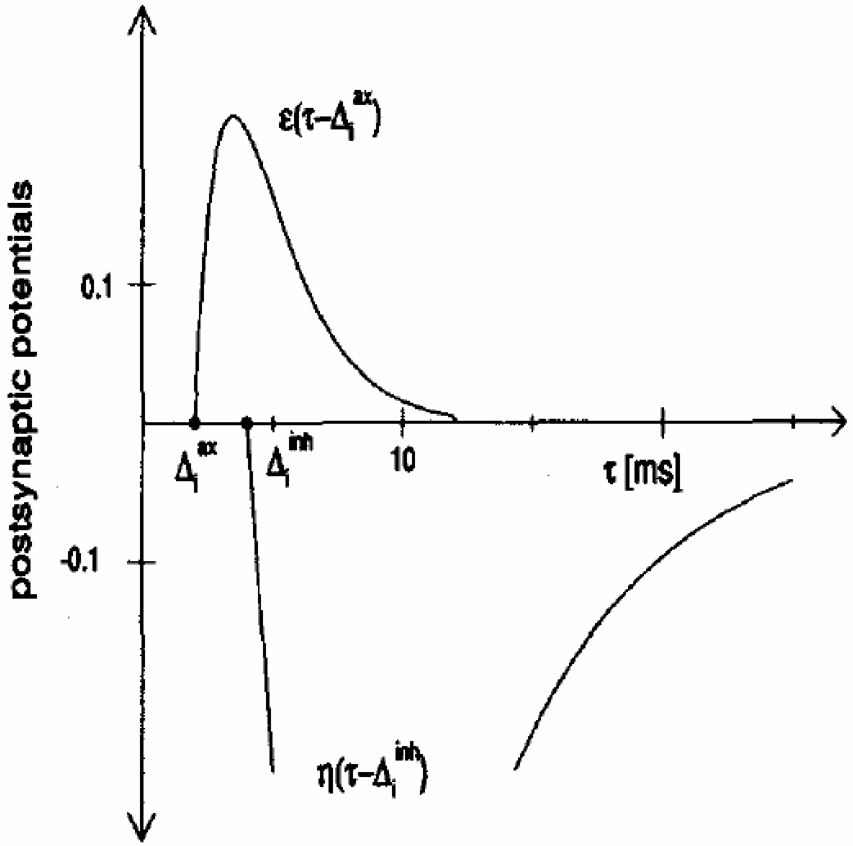
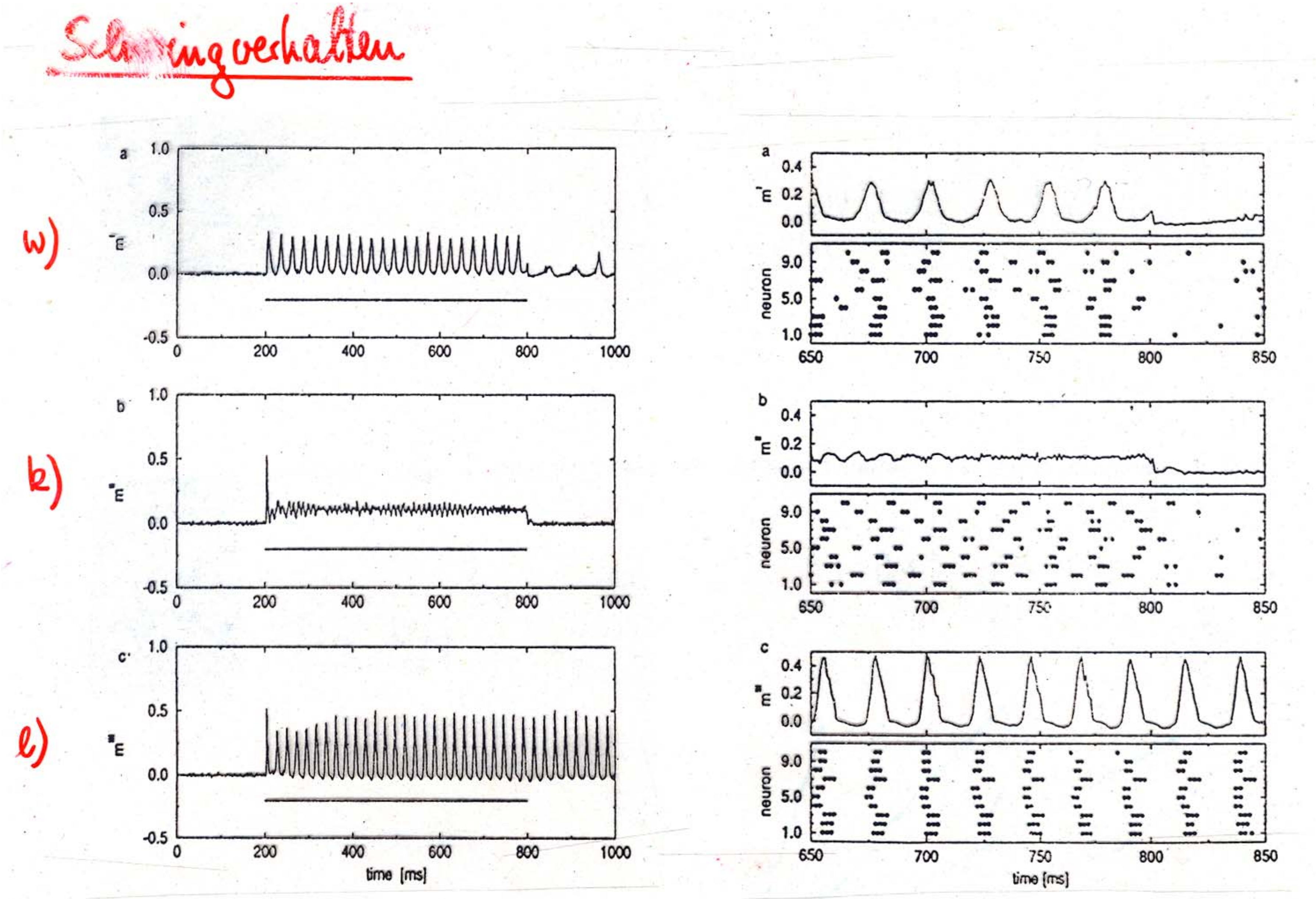
Inhibition

- Delayed
- Fires a few pulses followed by a pause



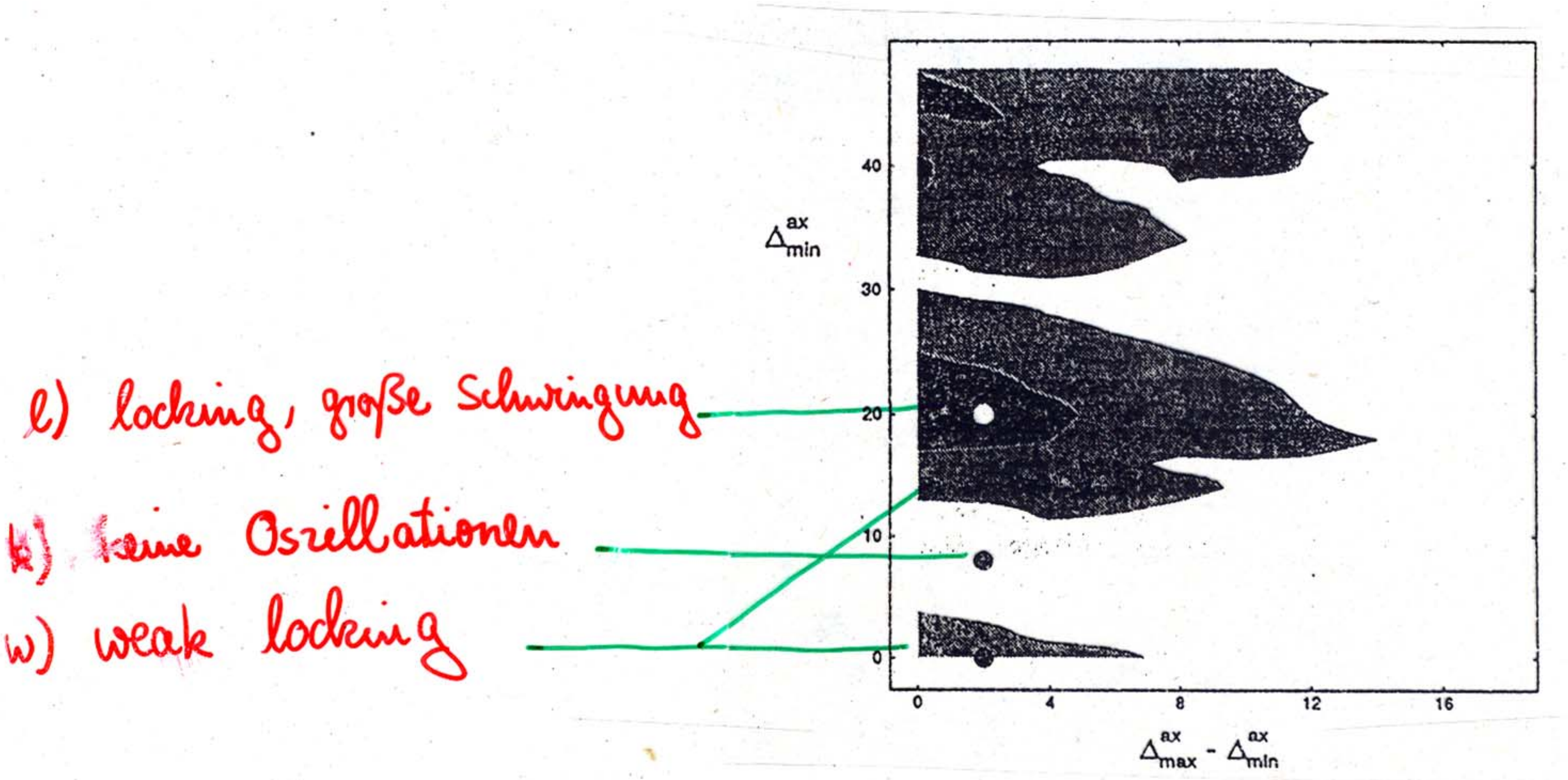


Phase Locking and Pattern Recognition



A biologically motivated and analytically soluble model of collective oscillations in the cortex.
Gerstner W, Ritz R, van Hemmen JL, Biol Cybern. 1993;68(4):363-74.

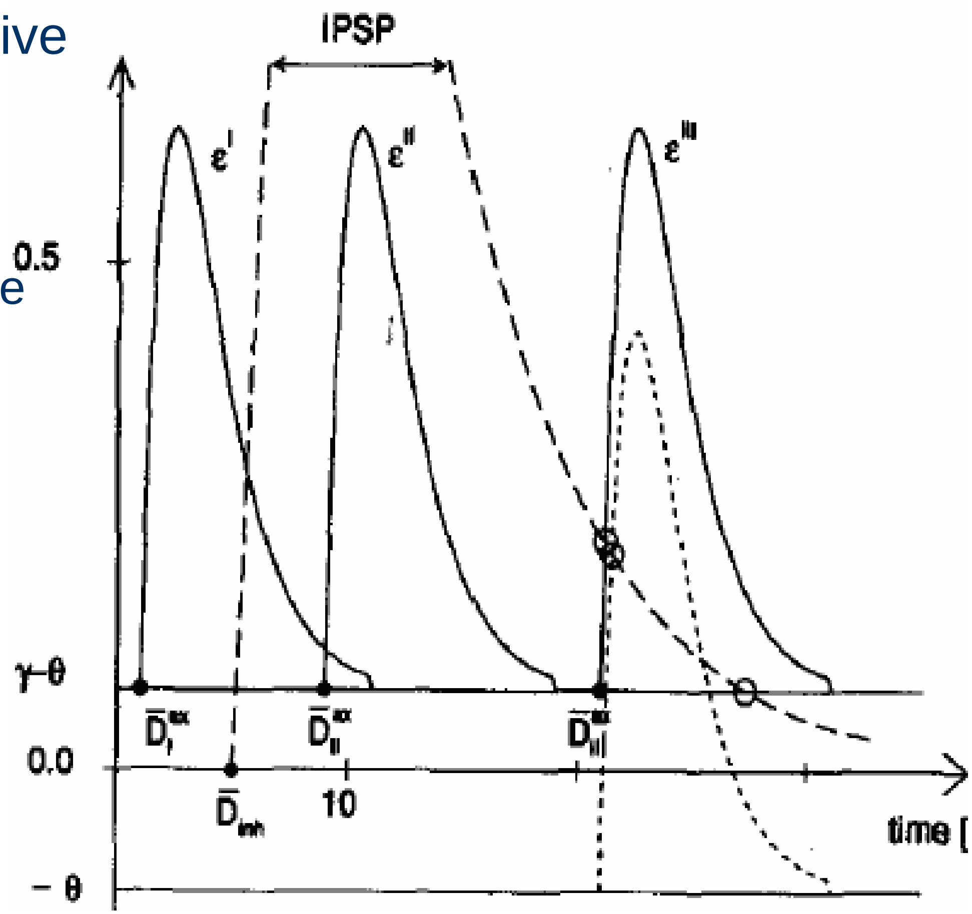
Phase Locking and Pattern Recognition



A biologically motivated and analytically soluble model of collective oscillations in the cortex. Gerstner W, Ritz R, van Hemmen JL, Biol Cybern. 1993;68(4):363-74.

Explanation

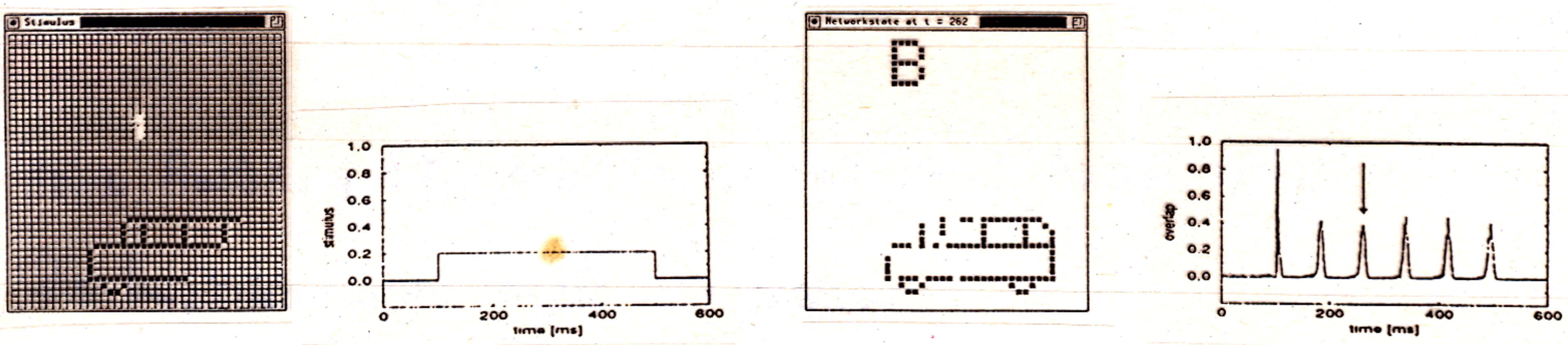
- Behaviour depends of relative timing of inhibition and excitation
- Low delay:
  - Quick feedback, immediate growth
- Medium delay:
  - Excitation while neuron is inhibited
- Long delay:
  - Excitation from previous oscillation stimulates the following excitation



Phase Locking and Pattern Recognition

Bei weak locking:

(Σ : 7 Muster)



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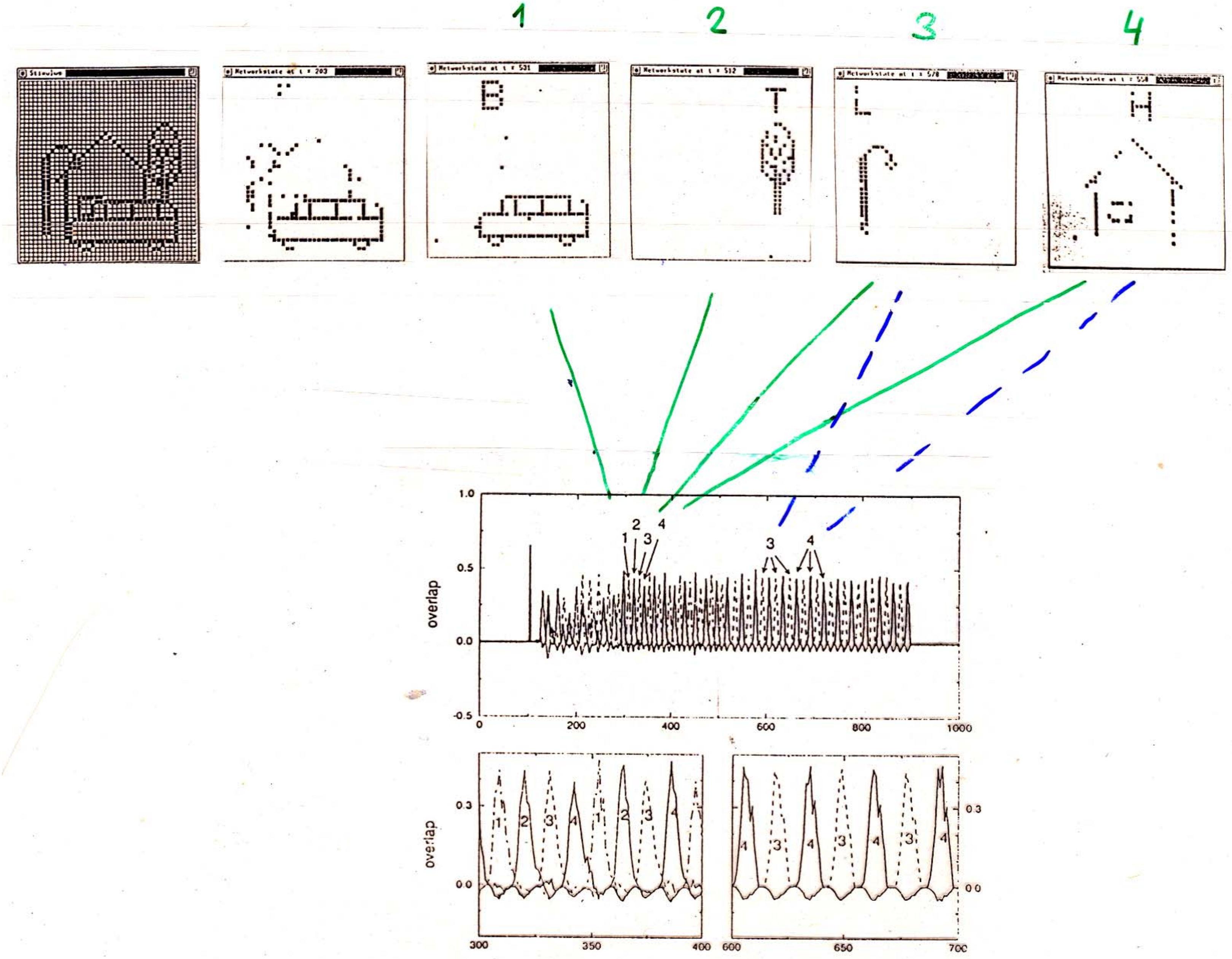
Phase Locking and Pattern Recognition



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Gerstner W, Ritz R, van Hemmen JL, Biol Cybern. 1993;68(4):363-74.

Ausblick

Dadurch

- ○ Keine Änderung der Synapsen während dem Erkennen nötig!
- ○ Die Inhibitionsstärke regelt
  - Frequenz
  - Anpassung auf Stärke des Reizes
- ○ Orts- und Drehinvarianz damit noch nicht erklärt
- ○ Bei Oszillationen mit 30Hz kann „man" rd. 4 Dinge gleichzeitig erkennen (→ Analogie zum Denken?)

Auslesen des erkannten Musters

Etwas erkennen heißt, darauf zu reagieren.

(Wie kann ich sonst wissen, ob jemand etwas erkannt hat?)

↝ Erkennungsvorgang muß nur Reaktion auslösen können d.h. andere Zellgruppen anregen. Synchron feuernde Zellgruppen können das wahrscheinlich gut.

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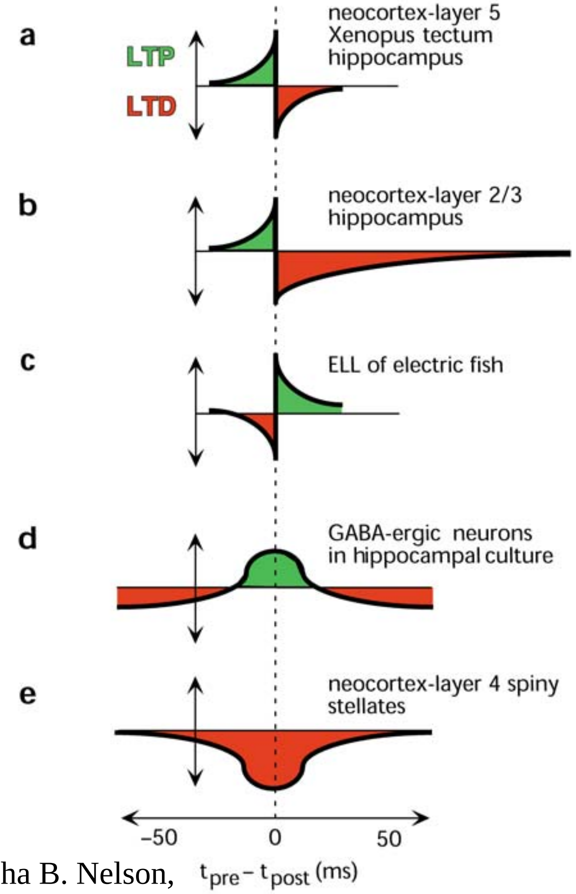
Towards realistic neurons: Temporal Learning

Hebbian in time

$$\Delta J_{ij} = \frac{1}{NT}\int_{0}^{T} e_{ij}(t) S_i(t) S_j(t-\tau)\,dt$$

Shapes of $e_{ij}(t)$



From: L. F. Abbott and Sacha B. Nelson,
Nature Neuroscience Suppl., 3:1178 (2000)

How does the Hebbian learning paradigm keep up with experiments?

Single neurons before and after a synaptic transmission are excited externally with different time delays. The efficiency of the synapse is recorded before and after the learning protocol.

This allows to infer the time resolution and direction of learning increment ΔJ_{ij} for a synapse (left).

These results would for sure have pleased Hebb. Indeed, the precise timing of neuron modulates the learning of a synapse with a very high time resolution on the ms time scale.

Temporal Patterns

FIG. 3. Sequential retrieval of three embedded patterns.

If we start from a distribution of axonal lengths, different synapses transport the information of both time delay and strength.

This can actually be used to extend the associative memory of networks onto the temporal domain: a sequence of patterns can be stored. If triggered, movie of patterns is generated (left).

from:

Retrieval of spatio-temporal sequence in asynchronous neural network, Hidetoshi Nishimori and Tota Nakamura, Physica Review A, 41, 3346-3354 (1990)

Sensory Maps: Kohonen Network

Finding: **sensory maps** are found in the brain with a high large scale organization.

Problem: **how** does the brain map and **wire similar outputs next to each other** although there is **no master** to order the things?

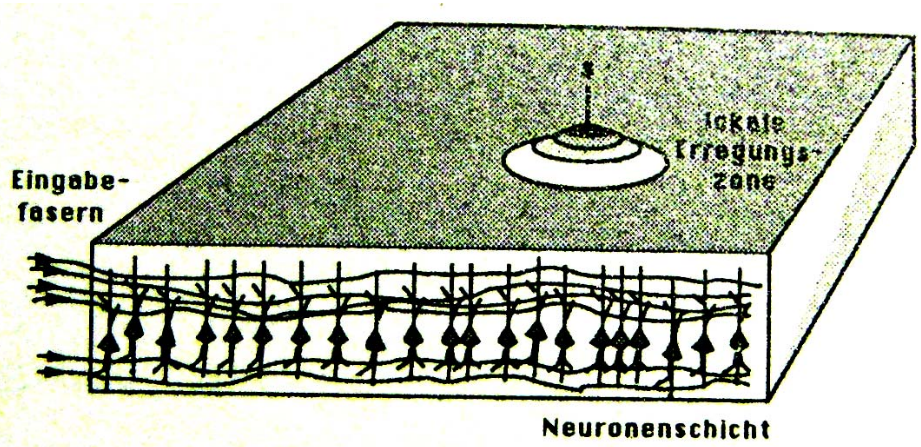


Abb.4.1 Schematische Darstellung der Neuronenschicht in Kohonens Modell. Die horizontal verlaufenden Nervenfasern führen das Eingabesignal heran und erregen die Schichtneuronen über synaptische Verbindungen. Laterale Wechselwirkungen zwischen den Neuronen begrenzen die Reaktion der Neuronen auf eine räumlich begrenzte "Erregungszone". Die Schicht wirkt als "topographische Merkmalskarte", wenn der Ort **s** der Erregungszone Aufschluß über den Anteil wichtiger Merkmale im Eingabesignal gibt.

Approach: Kohonen **assumed a "winner takes all"** approach where direct **neighbors profit from a mapping** and more distant ones are punished. With this, a simple algorithm (next page) generates beautiful sensory maps.

Disadvantage: We can only **guess the real microscopic algorithm** behind the approach since it appears that we need a master to determine the winner.

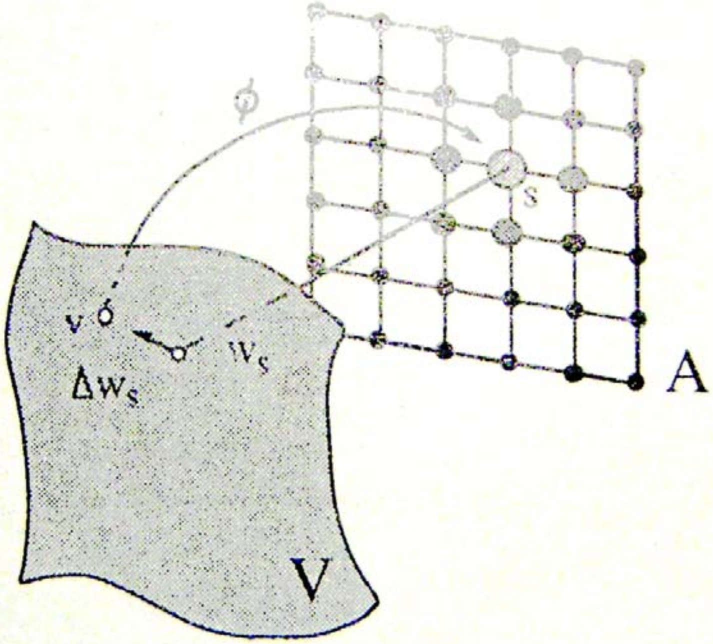
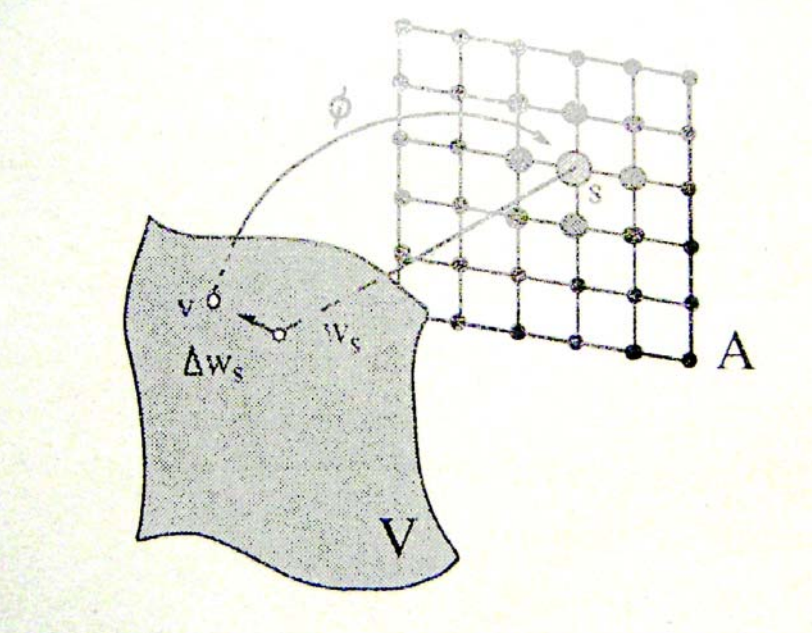


Abb.4.2 Der Adaptationsschritt in Kohonens Modell: der Eingangswert **v** selektiert ein Zentrum **s**, in dessen Nachbarschaft alle Neuronen ihre Gewichtsvektoren $\mathbf{w_s}$ in Richtung auf **v** "verschieben". Das Ausmaß der Verschiebung nimmt mit wachsendem Abstand vom Zentrum **s** ab, und ist im Bild durch unterschiedliche Grauwerte angedeutet.

Kohonen Network Algorithm



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Input stimulus vector v with index l

Target map location $\vec{a}$

Synaptic weight $J_{\vec{a},l}$ from V to A

Input of $\vec{a}$ given by $\sum_l J_{\vec{a},l} v_l$

Step 0: **Initialization**.
Synaptic weights J_{vl}=random.

Step 1: **Stimulus**
Choice of Stimulus Vector v.

Step 2: **Find Winner**
Find Stimulation-Winner location a with minimal weight vector - distance from stimulus v.

$$\|v - J_{\vec{a}'}\| \le \|v - J_{\vec{a}}\|$$

Step 3: **Adaptation**
Move the weights of winner (and its surrounding h) towards the stimulus v

$$J_{\vec{a}}^{(new)} = J_{\vec{a}}^{(old)} + \varepsilon h_{\vec{a},\vec{a}'}[v - J_{\vec{a}}^{(old)}]$$

and **go to Step 1**. This will converge towards a mapping given by:

$$v \rightarrow \vec{a} \quad \text{with} \quad \|v - J_{\vec{a}}\| \text{ minimal.}$$

Kohonen Example: 2D to 2D mapping

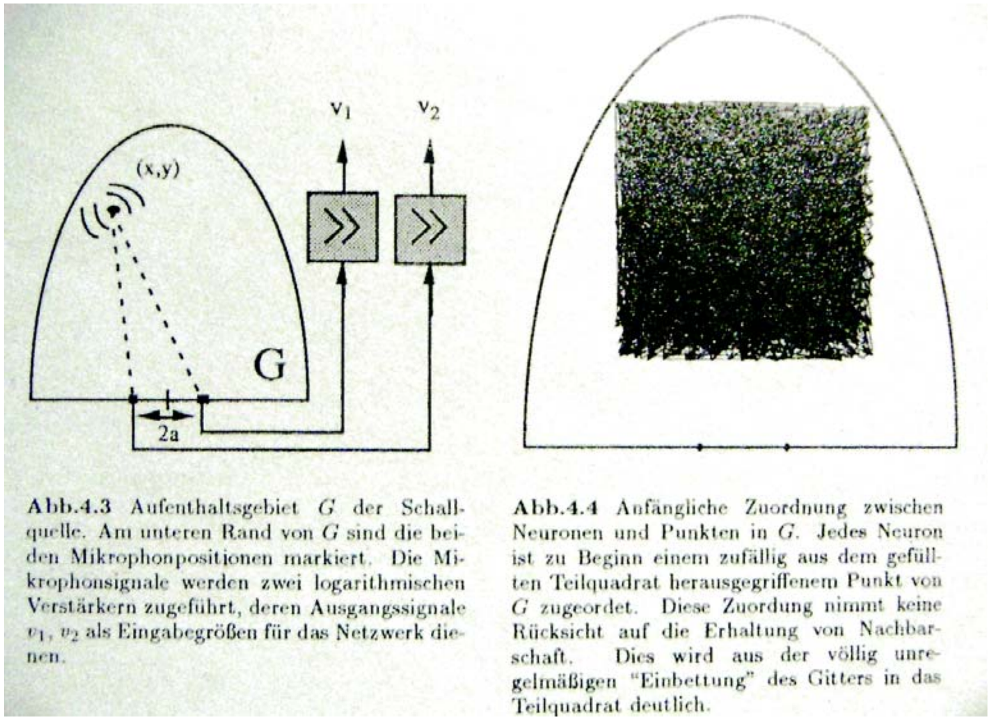

Abb.4.3 Aufenthaltsgebiet *G* der Schallquelle. Am unteren Rand von *G* sind die beiden Mikrophonpositionen markiert. Die Mikrophonsignale werden zwei logarithmischen Verstärkern zugeführt, deren Ausgangssignale v_1, v_2 als Eingabegrößen für das Netzwerk dienen.

Abb.4.4 Anfängliche Zuordnung zwischen Neuronen und Punkten in *G*. Jedes Neuron ist zu Beginn einem zufällig aus dem gefüllten Teilquadrat herausgegriffenem Punkt von *G* zugeordet. Diese Zuordung nimmt keine Rücksicht auf die Erhaltung von Nachbarschaft. Dies wird aus der völlig unregelmäßigen "Einbettung" des Gitters in das Teilquadrat deutlich.

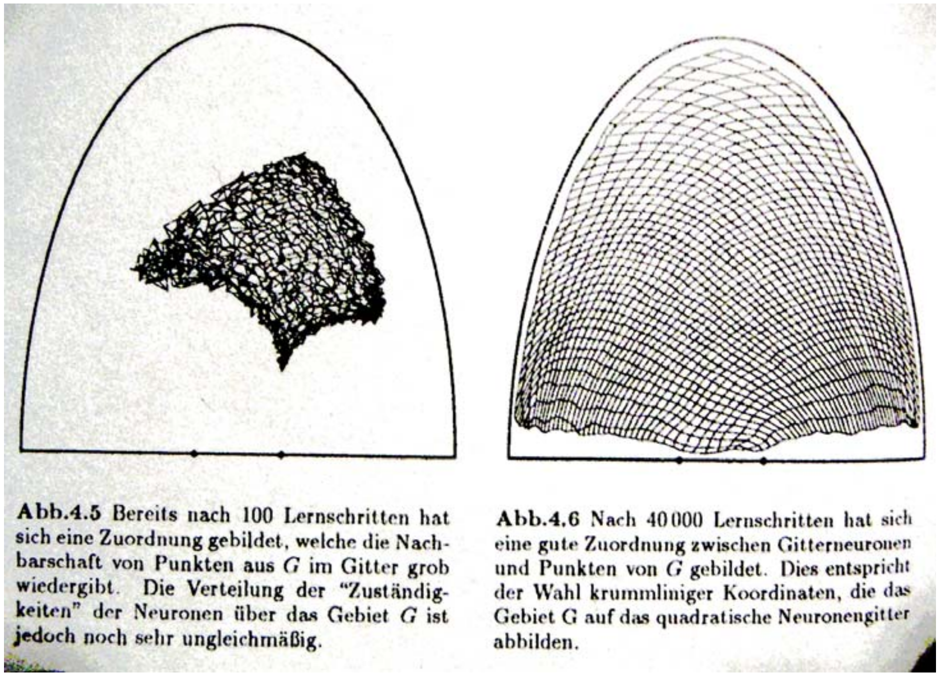

Abb.4.5 Bereits nach 100 Lernschritten hat sich eine Zuordnung gebildet, welche die Nachbarschaft von Punkten aus *G* im Gitter grob wiedergibt. Die Verteilung der "Zuständigkeiten" der Neuronen über das Gebiet *G* ist jedoch noch sehr ungleichmäßig.

Abb.4.6 Nach 40 000 Lernschritten hat sich eine gute Zuordnung zwischen Gitterneuronen und Punkten von *G* gebildet. Dies entspricht der Wahl krummliniger Koordinaten, die das Gebiet G auf das quadratische Neuronengitter abbilden.

Example.

Input vector v is the **logarithmic amplitude of two microphones** which record a **sound in a 2D space.**

We start with random weights J.

The Kohonen algorithm results in a **map that reflects** the setting of the sound location in 2D space.

The Kohonen-map has **memorized neighborhood information into their synaptic weights**.

Kohonen Example: 2D to 1D mapping

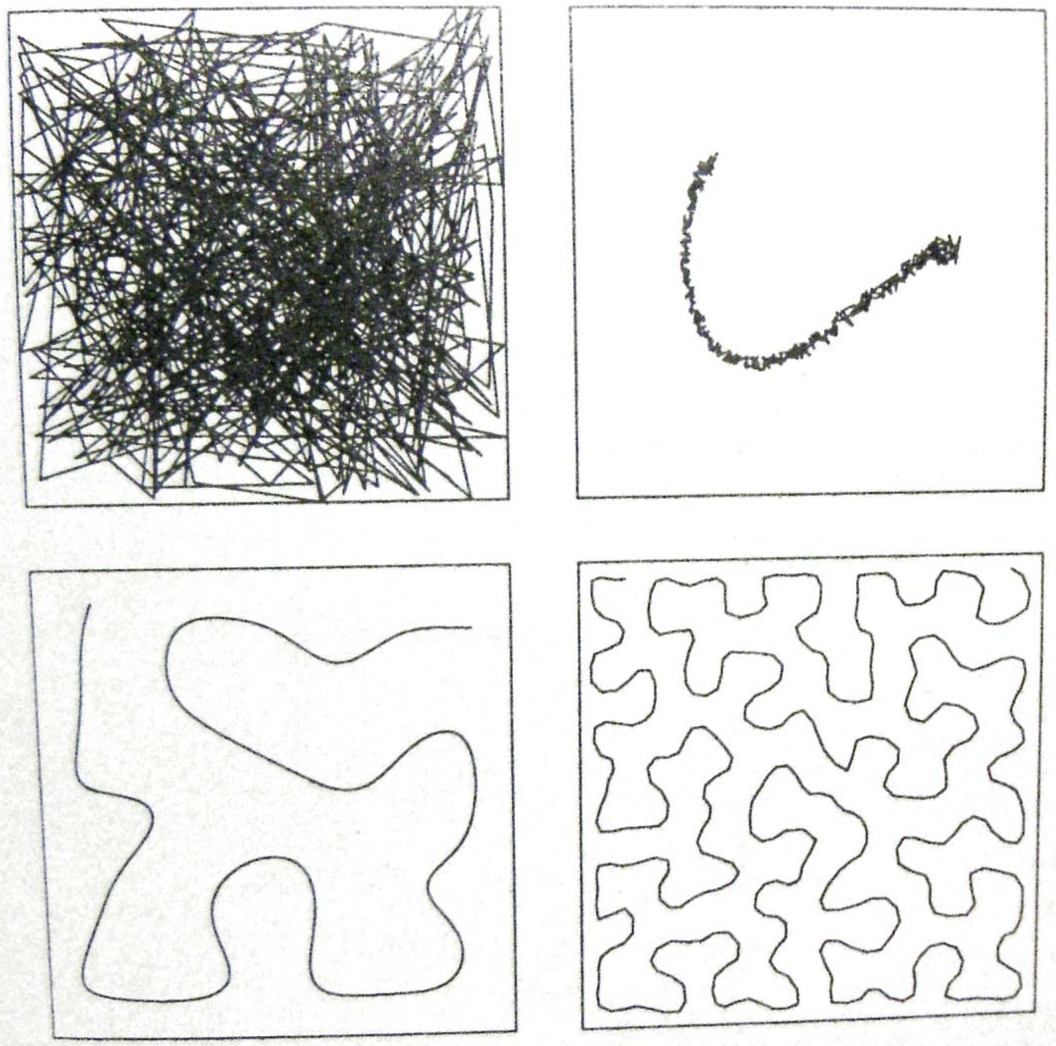

Abb.4.9 Abbildung zwischen einer Kette und einem Quadrat. Von links oben nach rechts unten: a) zufällig gewählte Anfangszuordnung; b) Grobzuordnung nach 200 Markov-Schritten; c) nach 50 000 Markov-Schritten; d) Nach 100 000 Markov-Schritten ähnelt die erhaltene Zuordnung einer "Peano-Kurve".

Quite impressive is the Kohonen mapping **between different dimensions** - in this case between the locations in 2D to a 1D receptive Kohonen map.

The mapping problem in this case is similar to the **traveling salesman-problem**.