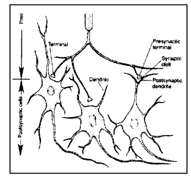
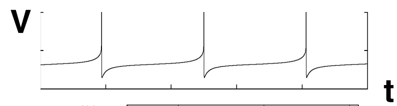
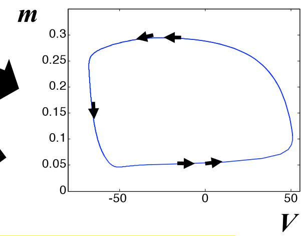
Reduction of neurons to phases

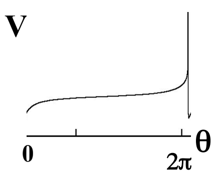
start with "biophysical" neuron model

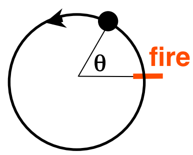


$$
\begin{aligned}
C\dot{V} &= -\bar{g}_{Na} m^3 h (V - E_{Na}) - \bar{g}_K n^4 (V - E_K) - \bar{g}_L (V - E_L) + I_a \\
\dot{m} &= \alpha_m(V)(1-m) - \beta_m(V) m \\
\dot{n} &= \alpha_n(V)(1-n) - \beta_n(V) n \\
\dot{h} &= \alpha_h(V)(1-h) - \beta_h(V) h
\end{aligned}
$$







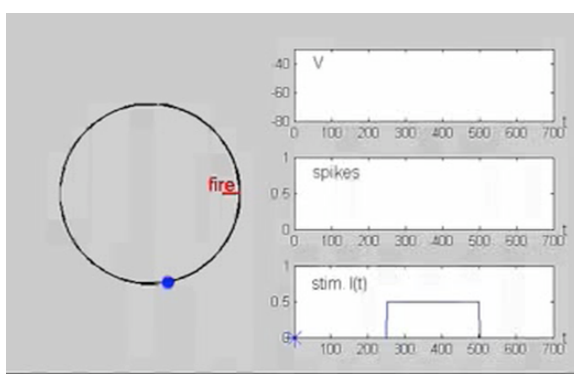


ALERT: Interesting methods here!

Winfree '74, Guckenheimer '75

1

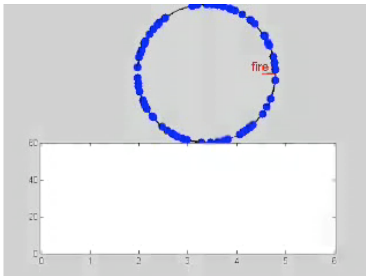
Phase dynamics



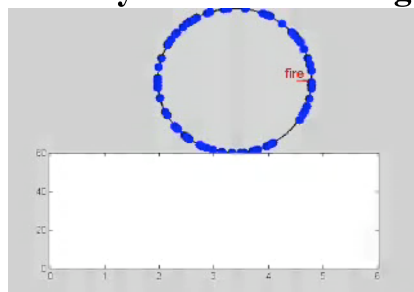
2

Spike trains, firing rates, and synchrony

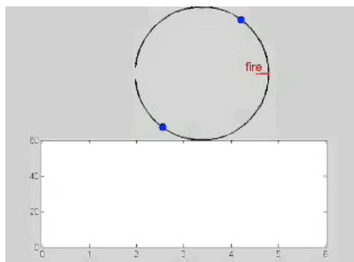
1. Decorrelated firing



2. Synchronized firing



3. Anti-synchronized, frequency-doubled firing

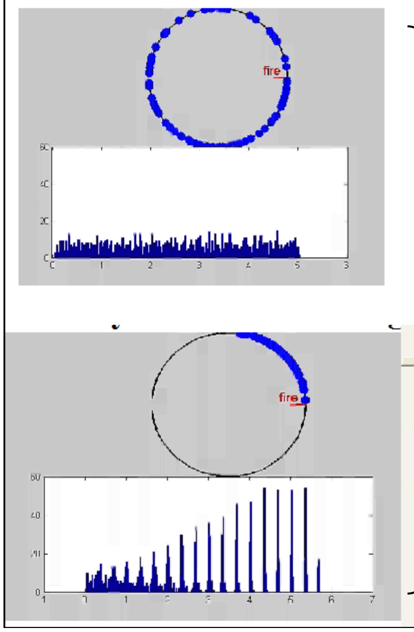


Roles for synchrony

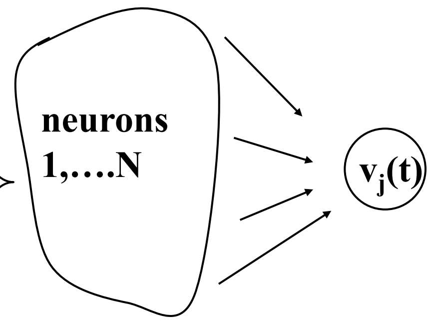
– 1) Synchrony allows information to propagate through “layers” of neurons

– 2) Synchrony enables new information processing strategies

A role for synchrony in signal transmission



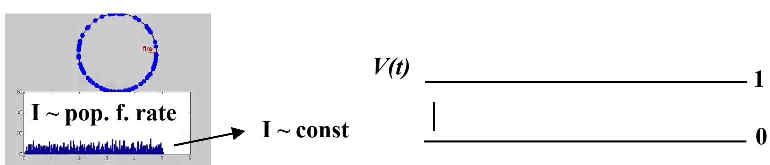
Can the population trigger upstream cells? Answer depends on synchrony … "synchrony controls salience of representation"

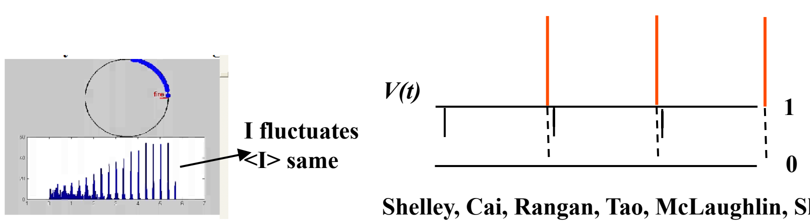


If average input <I> from upstream neurons insufficient to cause firing, need FLUCTUATIONS in I due to synchrony to drive V above spiking threshold ("detecting" upstream event)

$$\tau\frac{dV}{dt} = -V + k_1 + k_2 I \quad ; 0 \le V \le 1$$

plus "reset" condition: $V \to 0$ and spike when passes 1





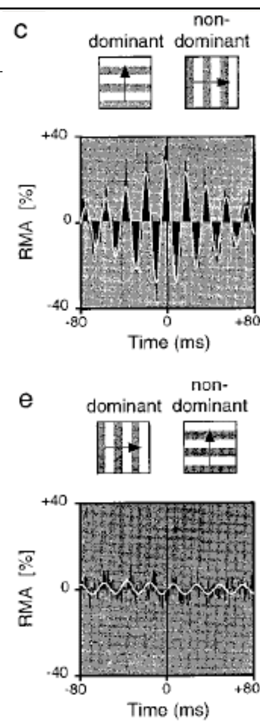
Shelley, Cai, Rangan, Tao, McLaughlin, Shapley – Fluctuation driven firing (related)

Another role for synchrony …

- **Hypothesis**
  - **alpha, beta, gamma rhythms set up "substrate" on which further neural computations are based.**
    - **gamma (30-80 Hz) … cognition ; synchrony at this frequency when "binding" together features of object, or in attention**
    - **beta (12-30 Hz) … intense mental activity**
    - **alpha (8-12 Hz) … wakefulness, reward?**
    - **delta (1-4 Hz) … sleep**

E.g. …

- **Measurements of synchrony in visual cortex during binocular rivalry task indicate *greater synchrony* among "currently" dominant neurons**



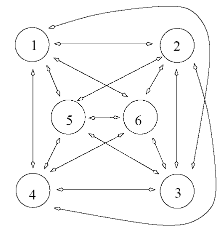
DOMINANT EYE

NON-DOMINANT EYE

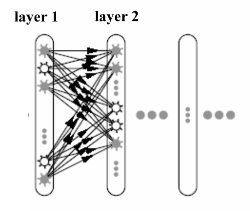
from Fries et al 1997

Three mechanisms for the generation of synchrony

1) Recurrent connections in a network



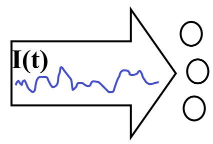
2) Feed-forward connections among layers



Diesmann, Gewaltig, Aertsen
Nature 402, 529, 1999.

3) Shared, fluctuating inputs to a population

Entrainment -- no connections!



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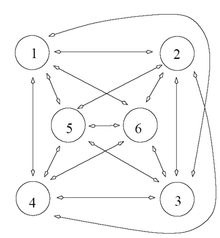
Three mechanisms for the generation of synchrony

1) Recurrent connections in a network



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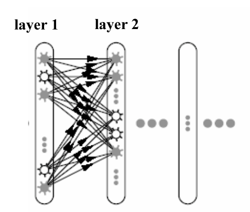

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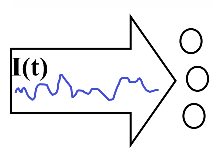
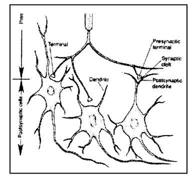


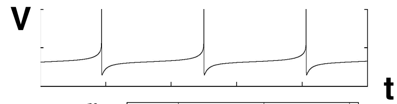
10

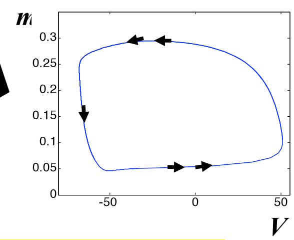
recall …. reduction of neurons to phases

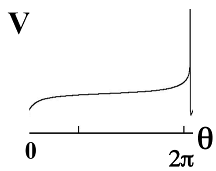
start with biophysically plausible neuron model

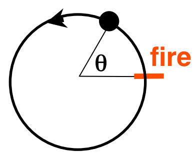


$$C\dot{V} = -\bar{g}_{Na} m^3 h(V - E_{Na}) - \bar{g}_K n^4 (V - E_K) - \bar{g}_L (V - E_L) + I_a \boxed{+\mathbf{I_{syn}(t)}}$$
$$\dot{m} = \alpha_m(V)(1-m) - \beta_m(V)m$$
$$\dot{n} = \alpha_n(V)(1-n) - \beta_n(V)n$$
$$\dot{h} = \alpha_h(V)(1-h) - \beta_h(V)h$$









ALERT: Interesting methods here!

Winfree '74, Guckenheimer '75

11

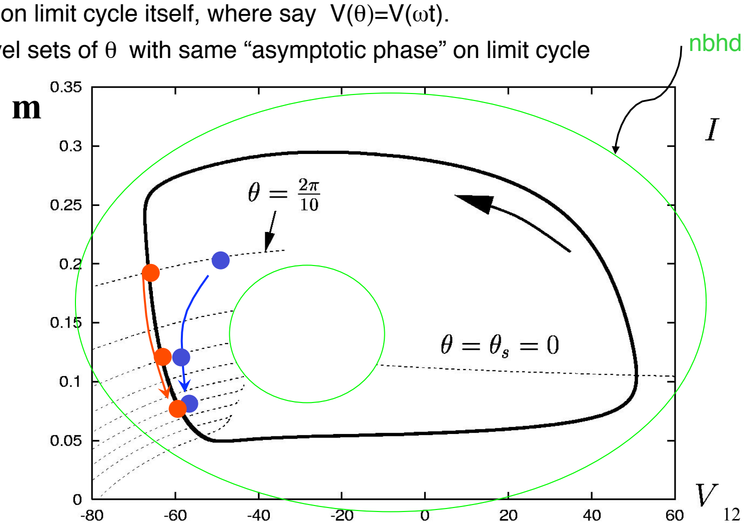
Reduction of neurons to phases

[Coddington and Levinson, 1955, Winfree, 1974, Guckenheimer, 1985]

In nbhd. of limit cycle, define variable $\theta(V,m,n,h)$ such that: $\boxed{\frac{d\theta}{dt} = \omega \text{ along trajectories, where } \omega = \frac{2\pi}{T}}$

Strategy: start on limit cycle itself, where say $V(\theta)=V(\omega t)$.

Then define level sets of θ with same "asymptotic phase" on limit cycle



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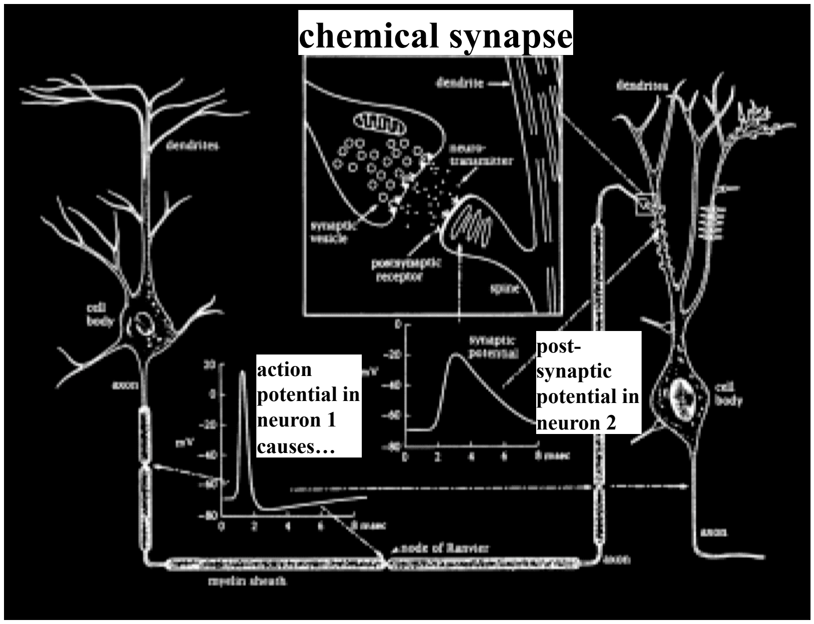
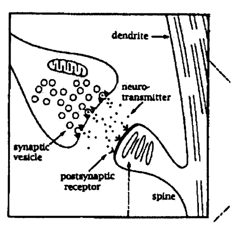
Recall how neurons communicate…



13



Kandel and Schwartz

(Chemical) Synapse

$$C\dot{V} = -\bar{g}_{Na} m^3 h (V - E_{Na}) - \bar{g}_K n^4 (V - E_K) - \bar{g}_L (V - E_L) + I_a \boxed{+ I_{syn}(t)}$$

$$\dot{m} = \alpha_m(V)(1-m) - \beta_m(V)m$$

$$\dot{n} = \alpha_n(V)(1-n) - \beta_n(V)n$$

$$\dot{h} = \alpha_h(V)(1-h) - \beta_h(V)h$$

$$I_{syn}(t) = g_{syn}(t) * (V_{syn} - V)$$

Excitatory synapse: $V_{syn} > V_{rest}$

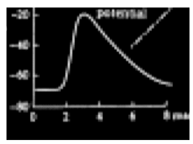
Inhibitory synapse: $V_{syn} < V_{rest}$

14

Approximating the synaptic current

$$\frac{dV}{dt} = \ldots \quad + \underbrace{I_{syn}(t)}_{g_{syn}(t)*(V_{syn}-V)}$$

assuming conductance impulse is brief, set $I_{syn}(t)=h*\delta(t)$



Excitatory synapse: $V_{syn} > V_{rest}$, $h>0$

Inhibitory synapse: $V_{syn} < V_{rest}$, $h<0$

$$\boxed{V \rightarrow V + h\Delta V}$$

15

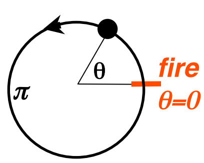
For phase dynamics …

$$\frac{d\theta}{dt} = \omega + z(\theta)\times\left[I_{syn}(t)\right]$$

natural frequency

phase response curve
(phase sensitivity curve)

$$\boxed{z(\theta) = \lim_{\Delta V \to 0} \frac{\Delta\theta}{\Delta V}}$$

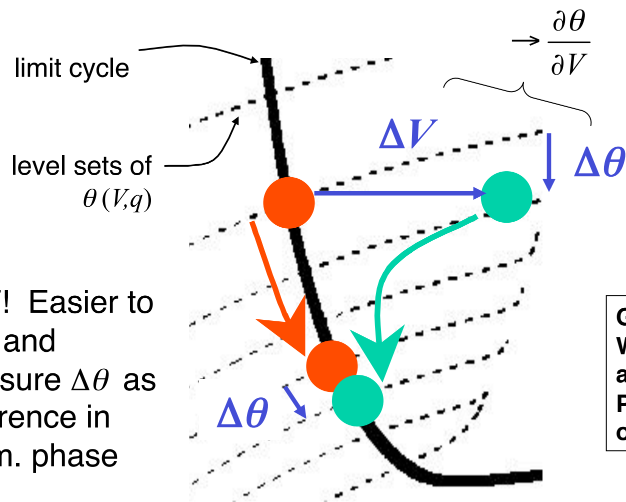


16

Finding z(θ) .

Asymptotic phase property of field $\theta\,(V,q)$ gives nice way to calculate

$$z(\theta) = \lim_{\Delta V \to 0} \frac{\Delta\theta}{\Delta V}$$



standard way to calculate partial deriv. -- must know $\theta\,(V,q)$ in nbhd. of lim cycle.

BUT! Easier to wait and measure $\Delta\theta$ as difference in asym. phase

Glass and Mackey, Winfree, Ermentrout and Kopell, Izhikevich, Park and Kim, and others

17

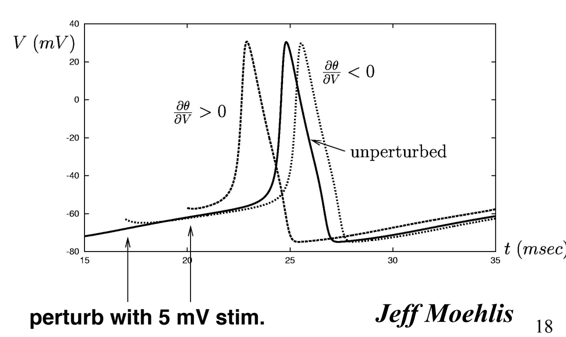
Finding z(θ) .

Calculating the phase response curve:

- Perturb an uncoupled neuron with a brief voltage stimulus ΔV at different times in its cycle, parameterized by θ
- Measure the resulting phase-shift $\Delta\theta$ with respect to the unperturbed system

For Hodgkin-Huxley neurons with $I = 10\mu A/cm^2$:

$$z(\theta) = \lim_{\Delta V \to 0} \frac{\Delta\theta}{\Delta V}$$



Jeff Moehlis

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Have phase dynamics … that you could directly derive from the laboratory !

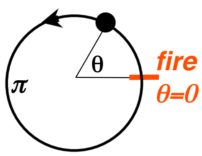
Glass and Mackey, Winfree

$$\frac{d\theta}{dt} = \omega + z(\theta) \times \left[I_{syn}(t) \right]$$

natural frequency

phase response curve
(phase sensitivity curve)

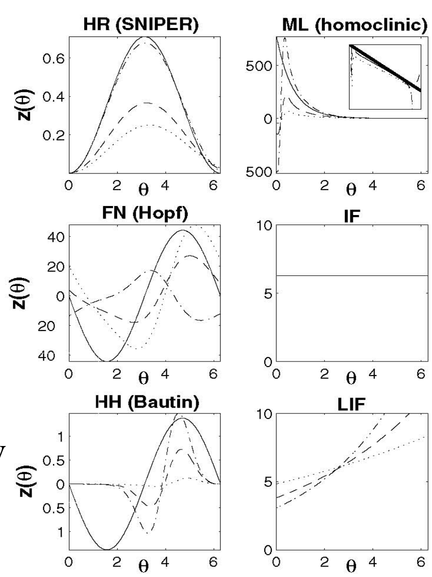
$$\boxed{z(\theta) = \lim_{\Delta V \to 0} \frac{\Delta\theta}{\Delta V}}$$



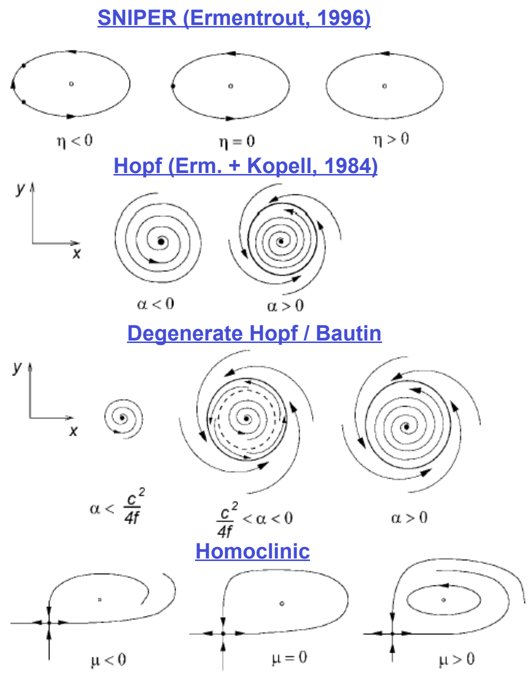
Phase response curves for different neurons look very different!

[Ermentrout and Kopell, Van-Vreeswick, Bressloff, Izhekevich, Moehlis, Holmes, S-B]

Hodgkin-Huxley



Leaky Integrate and Fire



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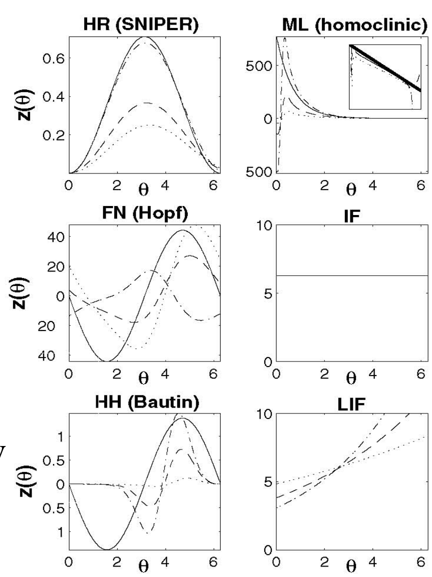
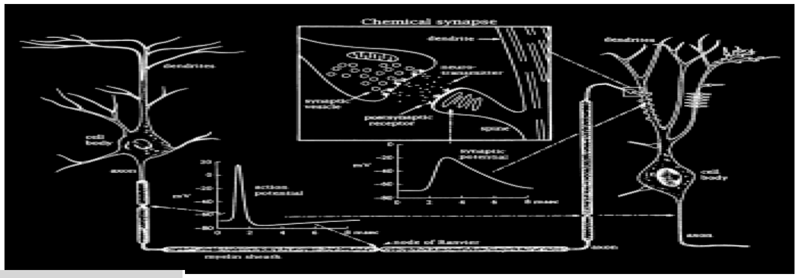


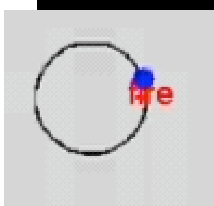
Hodgkin-Huxley

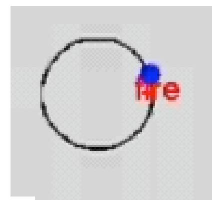
Leaky Integrate and Fire

22

Study synchrony in “network” of two coupled neurons







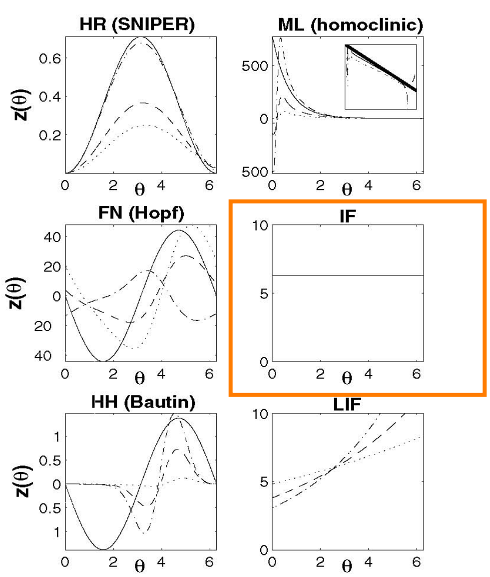
I$_{syn}$(t)

$$\frac{d\theta_1}{dt} = \omega + z(\theta_1) * h\delta(t - t_2^j)$$

$$\frac{d\theta_2}{dt} = \omega + z(\theta_2) * h\delta(t - t_1^j)$$

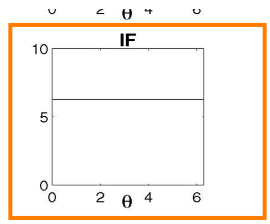
23

OK … let’s take the simplest imaginable case …



24

OK … let's take the simplest imaginable case …



$$\frac{dV}{dt} = I_b \quad ; 0 \leq V \leq 1 \qquad (1)$$

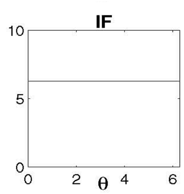
plus "reset" condition: $V \to 0$ and spike when passes 1

25

OK … let's take the simplest imaginable case for z -- IF

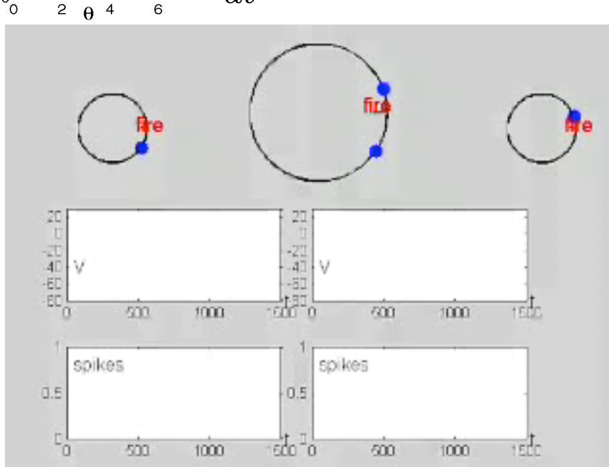
PRC

z(θ)



$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$

$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$



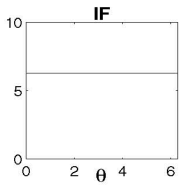
h>0
excit.
synapse

26

OK … let's take the simplest imaginable case for z -- IF

PRC

z(θ)



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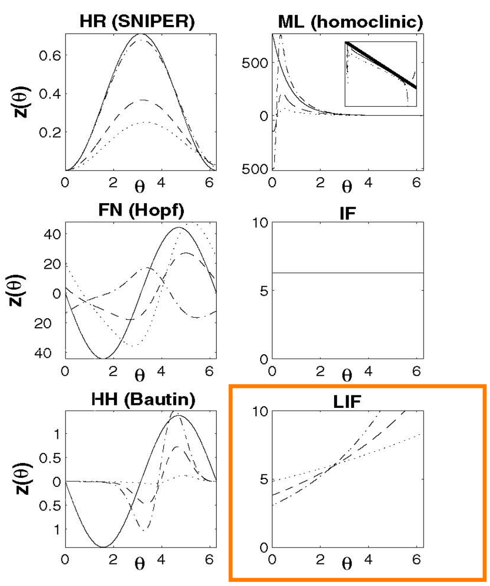
$$\frac{d\theta_2}{dt} = \omega + z(\theta_2)\delta(t - t_1^j)$$

Moral: coupling two neurons together does nothing if this coupling is not voltage (phase) dependent

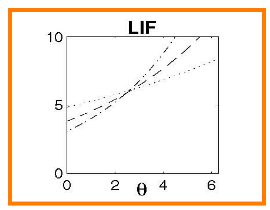
Next, consider the *leaky* integrate and fire model



The Leaky integrate and fire model

$$\tau \frac{dV}{dt} = -V + I_b + I_{ext}(t) \quad ; 0 \leq V \leq 1 \qquad (1)$$

plus "reset" condition: $V \to 0$ and spike when passes 1



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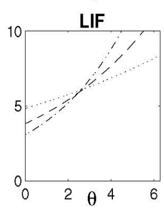
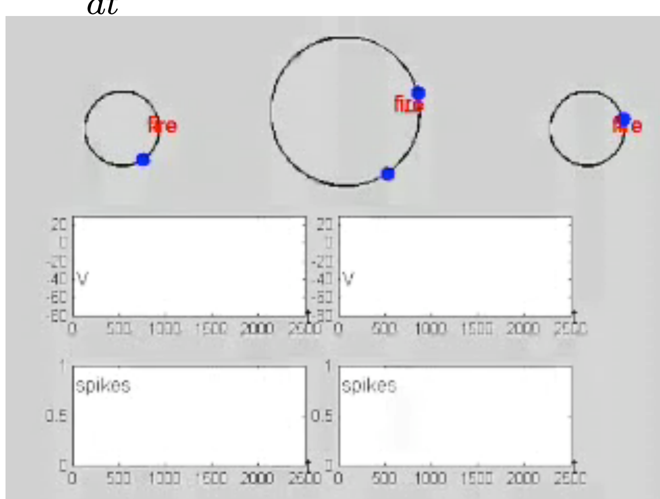
The Leaky integrate and fire model

PRC

z(θ)

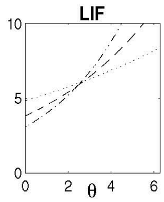


$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$



PRC

z(θ)



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$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$

Moral: "Fast" excitatory coupling can synchronize LIF neurons …

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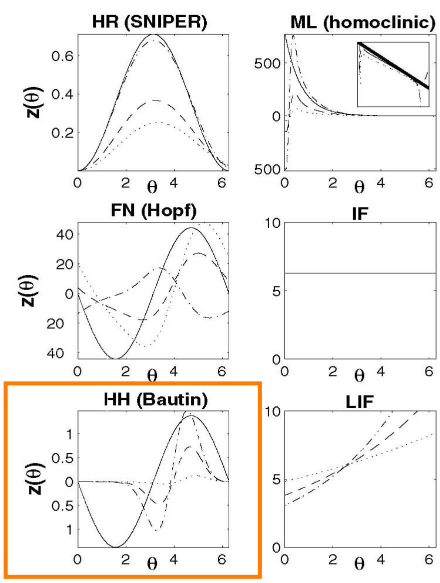
Next, back to Hodgkin-Huxley!



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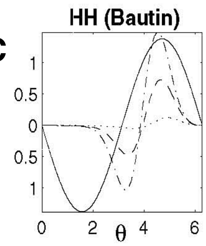
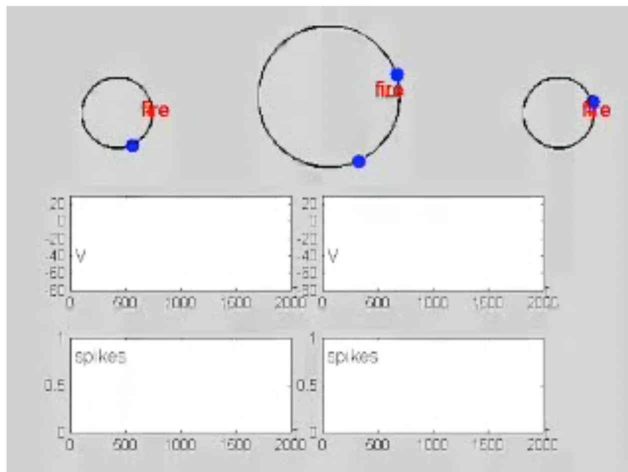
The Hodgkin-Huxley model

PRC

z(θ)



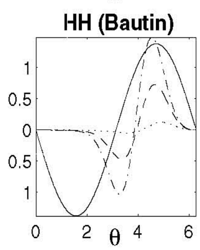
$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$



The Hodgkin-Huxley model

PRC

z(θ)



$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$

Moral: (again) “Fast” excitatory coupling can synchronize HH neurons …

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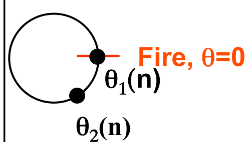
Analyze via Poincare map between firing times of θ_1

$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$

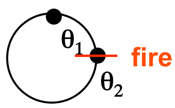
Nancy Kopell, Bard Ermentrout,
-- "weak coupling theory"

Let $\theta_{12} = \theta_1 - \theta_2$

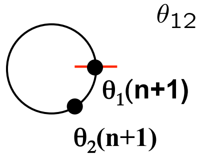


$\theta_2 \mapsto \theta_2 + hz\,(-\theta_{12}(n)))$, or:

$$\theta_{12}(n) \mapsto \underbrace{\theta_{12}(n) - hz\,(-\theta_{12}(n))}$$



$$\theta_1 \mapsto \theta_1 + hz\,[\overbrace{\theta_{12}(n) - hz\,(-\theta_{12}(n))}]$$



$$\begin{aligned}\theta_{12}(n+1) &= \theta_{12}(n) \\ &\quad -hz\,(-\theta_{12}(n)) \\ &\quad +hz\,[\theta_{12}(n) - hz\,(-\theta_{12}(n))] \\ &= \theta_{12}(n) + h\,\{z(\theta_{12}(n)) - z(-\theta_{12}(n))\} + \mathcal{O}(h^2)\end{aligned}$$

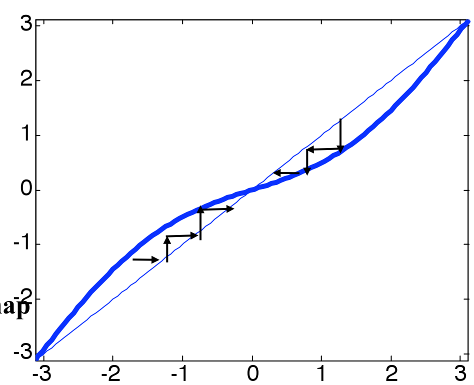
Phase-difference map $\theta_{12} = \theta_1 - \theta_2$

$$\theta_{12}(n+1) = \theta_{12}(n) + h\,\{z(\theta_{12}(n)) - z(-\theta_{12}(n))\} + \mathcal{O}(h^2)$$

E.g., for HH neuron, $z(\theta) \sim -\sin(\theta)$, so

$$\boxed{\theta_{12}(n+1) \approx \theta_{12}(n) - 2h\sin(\theta_{12})}$$

$\theta_{12}(n+1)$

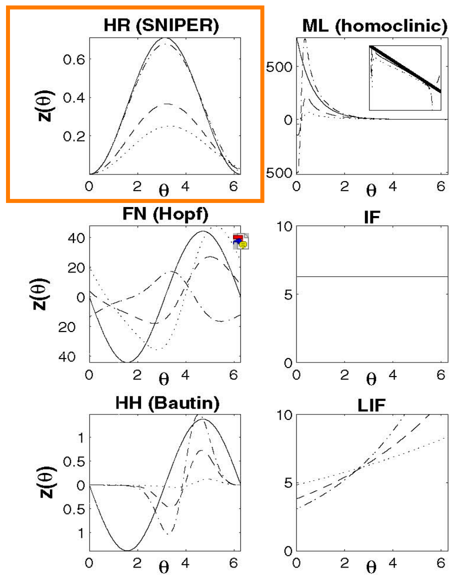


$\theta_{12}(n)$

See: synchronized state $\theta_{12}=0$ is stable fixed point for map

Let’s try (as our last example)

Very common in neural models …



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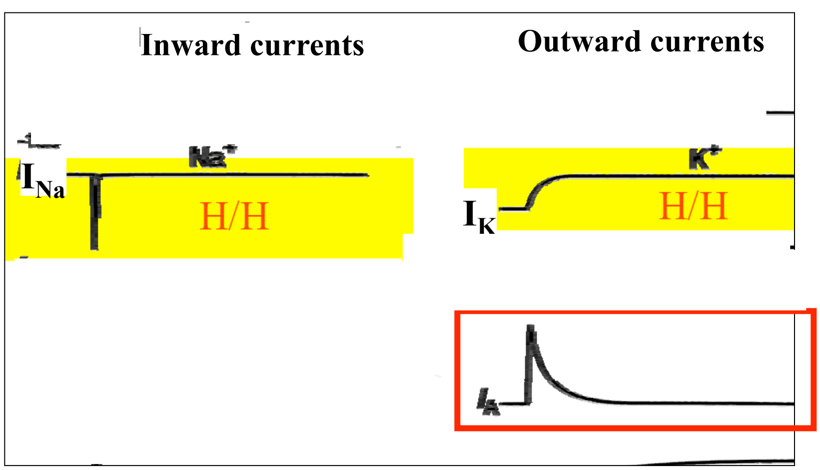
The “Hodgkin Huxley plus A current” model



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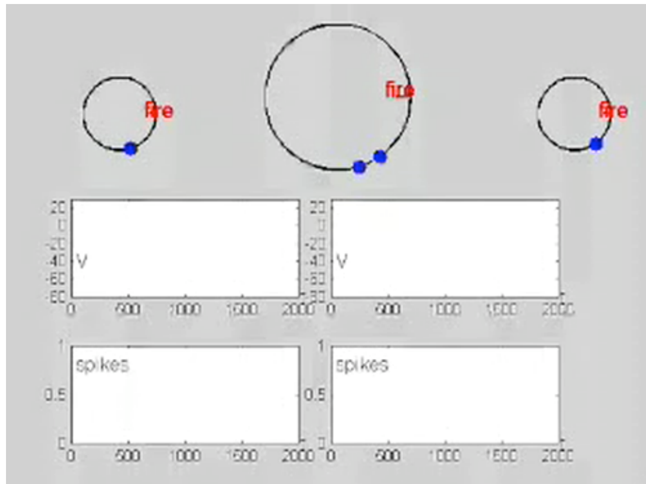
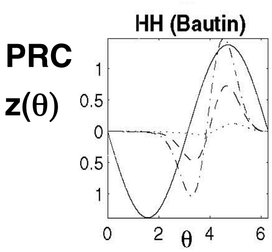
The "Hodgkin Huxley plus A current" model

$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$



The "Hodgkin Huxley plus A current" model



$$\frac{d\theta_1}{dt} = \omega + h * z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + h * z(\theta_2)\delta(t - t_1^j)$$

Moral: Excitatory coupling actually DEsynchronizes HH neurons with A currents

Stable "anti-synchronized" state

However, inhibition *does* synchronize …

"When inhibtion, not excitation, synchronizes…" Van Vreeswijk et al 1995

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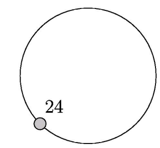
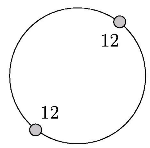
In fact, there are other types of stable antisynchronous states

- **N neurons, (slow) *inhibitory* synapses, Hodgkin-Huxley model:**

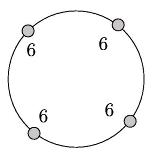
N=24



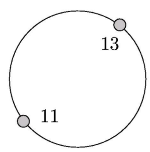
S_{24}
oscillators in phase



$(S_{12})^2 \otimes Z_2$
oscillators π out of phase



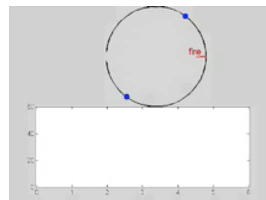
$(S_6)^4 \otimes Z_4$
oscillators $\pi/2$ out of phase



$S_{13} \times S_{11}$
oscillators $\delta \neq \pi$ out of phase

Multiple stable states

Each corresponds to a different effective frequency for the N neurons



Used by Rinzel to explain co-existence of delta (1-4 Hz) and "spindling" (8-14) Hz. rhythms [deep vs light sleep] – thalamo – cortical cells

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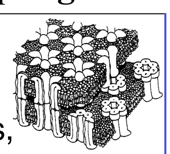
Beyond impulse coupling



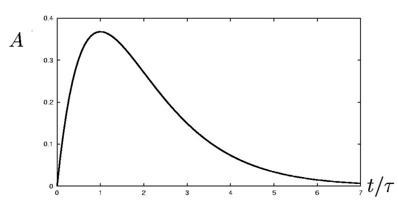
Brain has gap junctions,

$$\dot{V}_i = \cdots + \tfrac{\alpha_e}{N} \textstyle\sum_{j=1}^{N} (V_j - V_i)$$

N = number of neurons
α_e = electrotonic coupling strength

as well as slow chemical synapses.

$$\dot{V}_i = \cdots + \tfrac{\alpha_s}{N} (V_K - V_i) \textstyle\sum_{j=1}^{N} A(t - t_j - \tau_d)$$



$$\frac{d\theta}{dt} = \omega + z(\theta) \times \left[\underbrace{I_{syn}(t)}_{} \right]$$

$$\frac{d\theta_i}{dt} = \omega + z(\theta_i) \textstyle\sum_{j=1}^{n} I_j(\theta_j)$$

Kuramoto, Kopell, Ermentrout -- average coupling functions:

$$f_{e,s}(\theta) = \frac{1}{2\pi} \int_0^{2\pi} z(\phi) I_{e,s}^{coup}(\theta + \phi) d\phi$$

get a system depending on phase differences only

$$\frac{d\theta_i}{dt} = \omega + \underbrace{\frac{\alpha}{N} \sum_j f_e(\theta_j - \theta_i)}_{\text{electrotonic}} + \underbrace{\frac{\beta}{N-1} \sum_{j \neq i} f_s(\theta_j - \theta_i)}_{\text{synaptic}}$$

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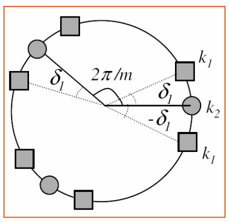
$$\frac{d\theta}{dt} = \omega + z(\theta)\times\left[\overbrace{I_{syn}(t)}\right]$$

$$\frac{d\theta_i}{dt} = \omega + z(\theta_i)\sum_{j=1}^{n} I_j(\theta_j)$$

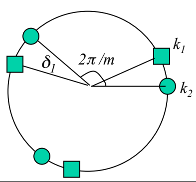
Symmetry arguments: exists huge variety of rotating equilibria

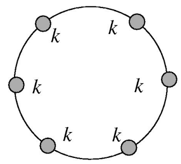
[Ashwin, Swift, Okuda, S-B.]

Proposition. f odd, satisfy inequality → solutions of form



Also, in general get:





Kuramoto, Kopell, Ermentrout -- average coupling functions:

$$f_{e,s}(\theta) = \frac{1}{2\pi}\int_0^{2\pi} z(\phi) I_{e,s}^{coup}(\theta+\phi)\,d\phi$$

get a system depending on phase differences only

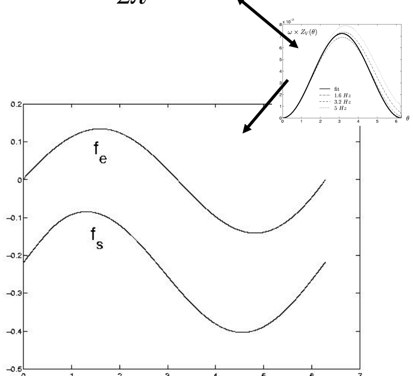
$$\frac{d\theta_i}{dt} = \omega + \underbrace{\frac{\alpha}{N}\sum_j f_e(\theta_j-\theta_i)}_{\text{electrotonic}} + \underbrace{\frac{\beta}{N-1}\sum_{j\neq i} f_s(\theta_j-\theta_i)}_{\text{synaptic}}$$

Contrasts situation for sin coupling

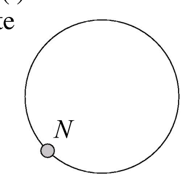
Coupling Functions

$$\frac{d\theta_i}{dt} = \omega + \underbrace{\frac{\alpha}{N}\sum_j f_e(\theta_j-\theta_i)}_{\text{electrotonic}} + \underbrace{\frac{\beta}{N-1}\sum_{j\neq i} f_s(\theta_j-\theta_i)}_{\text{synaptic}}$$

$$f_{e,s}(\theta) = \frac{1}{2\pi}\int_0^{2\pi} z(\phi) I_{e,s}^{coup}(\theta+\phi)\,d\phi$$



Proposition.
For f(.) = sin(.)
in-phase state



S_N
oscillators in phase

is globally stable

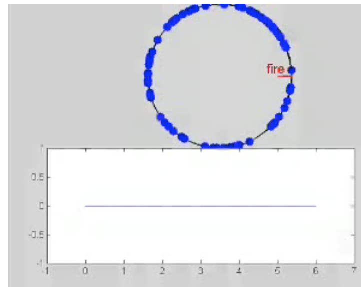
[Use gradient dynamics]

[Strogatz, S-B, Kuramoto,Okuda]

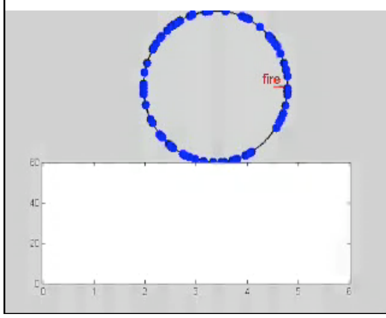
Synchronized firing requires similar frequencies

ω_i = 3 +- 0.5 Hz

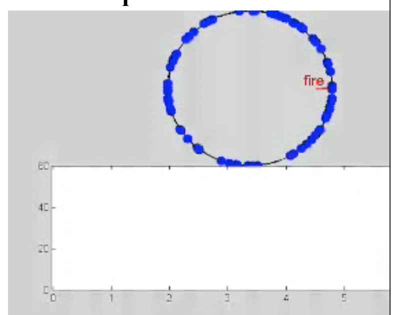
$$\frac{d\theta_i}{dt} = \omega_i + h\sum_{j=1}^{n} f(\theta_j - \theta_i)$$
$$i = 1, \ldots, N$$



ω_i = 3 +- 1.0 Hz

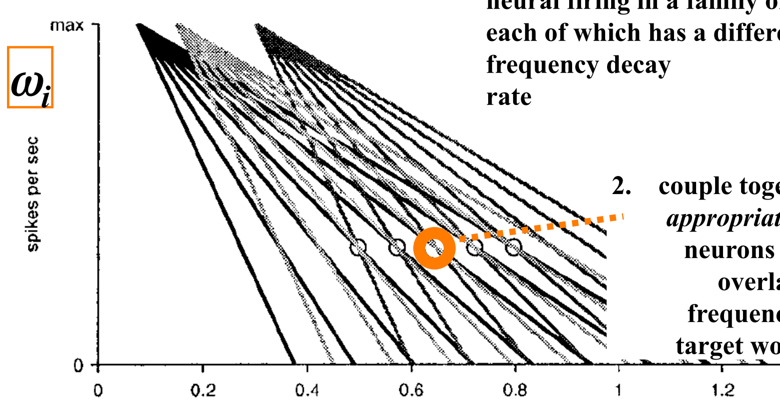


ω_i = 3 +- 1.5 Hz



- **this fact (!) allowed J. Hopfield and C. Brody to develop a new theory of speech recognition**

"o – n – e"



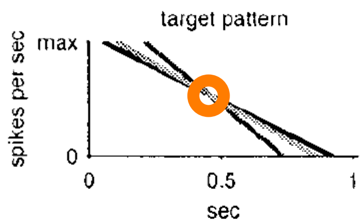
time (sec)

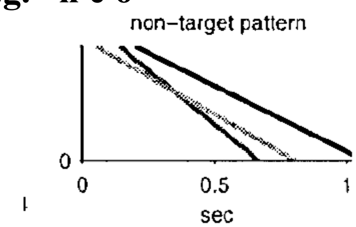
1. **(features of) incoming word trigger neural firing in a family of neurons, each of which has a different frequency decay rate**
2. **couple together -- *with appropriate synapses*- neurons that have overlapping frequencies when target word spoken**

PNAS **(2001)** vol. 98, **1282–1287**

- **“only” get overlap (among frequencies of selected neurons) when target word is presented**

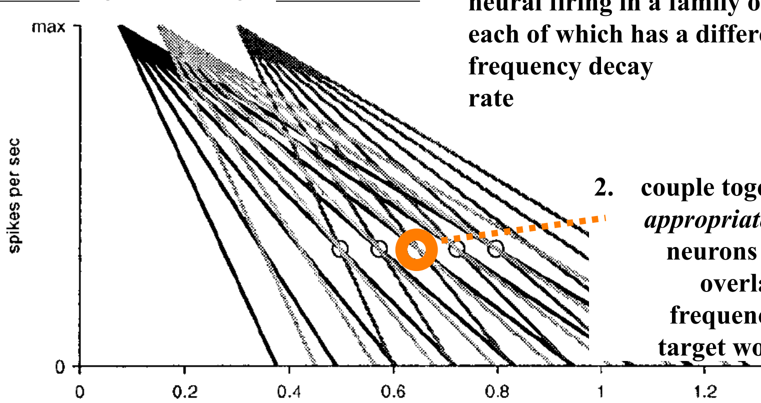
“o n e ” **e.g. “n e o ”**





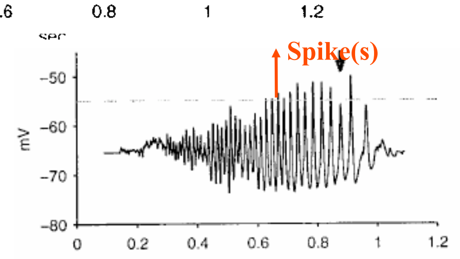
“o – n – e ”

1. **(features of) incoming word trigger neural firing in a family of neurons, each of which has a different frequency decay rate**



2. **couple together -- *with appropriate coupling*-- neurons that have overlapping frequencies when target word spoken**

3. Once the selected neurons synchronize, they drive “detector” cell above its threshold



SUMMARY

- 1) Synchrony allows information to propagate through "layers" of neurons
  - Synchronized activity can be necessary to trigger "downstream" cells
- 2) Coupling and rhythms yield new computational strategies
  - Diverse neurons give diverse results
    - frequency-doubling and antiphase states
    - significance for computation and beautiful mathematics (N. Kopell)
  - Speech recognition (Hopfield and Brody) and may other applications!

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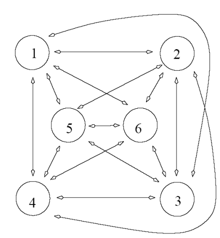
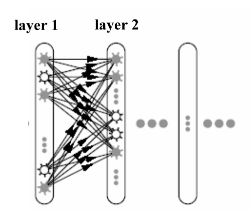
Three mechanisms for the generation of synchrony

1) Recurrent connections in a network

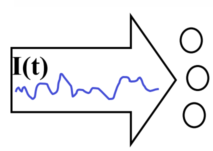


2) Feed-forward connections among layers

Diesmann, Gewaltig, Aertsen
Nature 402, 529, 1999.



3) Shared, fluctuating inputs to a population
Entrainment -- no connections!



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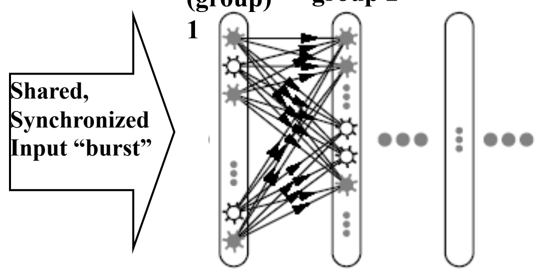
A key question…Diesmann, Gewaltig, Aertsen; Nature 402, 529, 1999

- ONCE a synchronized "burst" of activity has been generated, can it be stably propagated through *layers* of cortical tissue? Or will it "dissipate"?

Feed-fwd. ONLY connections between successive neural groups



Diesmann, Gewaltig, Aertsen
Nature 402, 529, 1999.

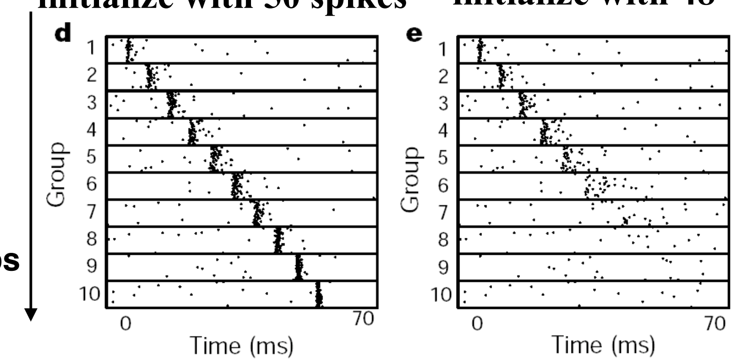
ABELES 1993 "synfire chains"

A key question…

- ONCE a synchronized "burst" of activity has been generated, can it be stably propagated through *layers* of cortical tissue? Or will it "dissipate"?

Feed-fwd. ONLY connections between successive neural groups

initialize with 50 spikes   initialize with 48



Diesmann, Gewaltig, Aertsen
Nature 402, 529, 1999.

ABELES 1993 "synfire chains"

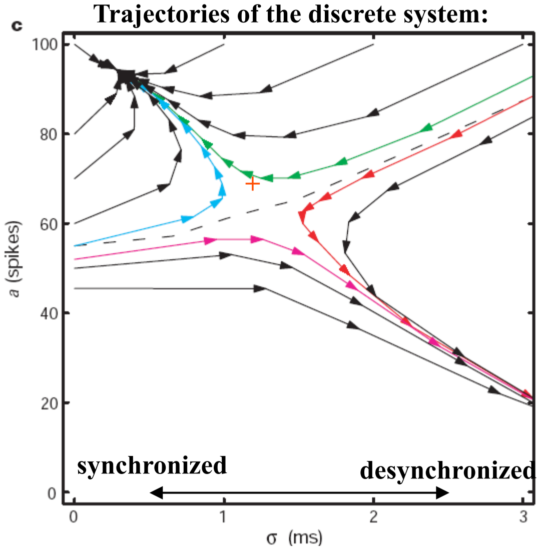
Answer: YES, if initiating spike volley suffic. large and synchronized

Explain --

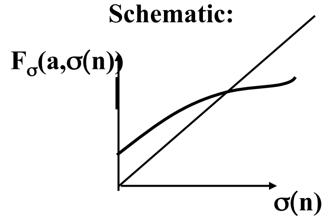
Numerically define a two dimensional map.
$a(n)$, activity (number of cells that spikes in layer n)
$\sigma(n)$, dispersion (spread in spike times at layer n).

$$(a(n+1), \sigma(n+1)) = F\,(a(n), \sigma(n))\ .$$

Trajectories of the discrete system:



Schematic:



See – synchrony DEVELOPS across layers

Diesmann, Gewaltig, Aertsen Nature 402, 529, 1999.

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- **Synchrony and feed forward networks at NYU:**
  - **Alex Reyes and collaborators**
    - **Build virtual feed forward networks out of REAL NEURON(s)**
    - **Analyze using phase or voltage density equations (Fokker-Planck, etc.)**

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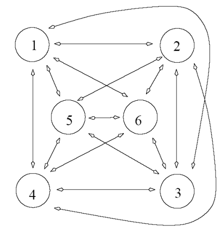
Three mechanisms for the generation of synchrony

1) Recurrent connections in a network



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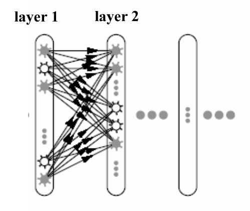

Diesmann, Gewaltig, Aertsen
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3) Shared, fluctuating inputs to a population

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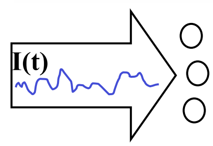

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Neurons produce reliable responses to fluctuating, but NOT constant (stepped) input

stepped input   fluctuating input

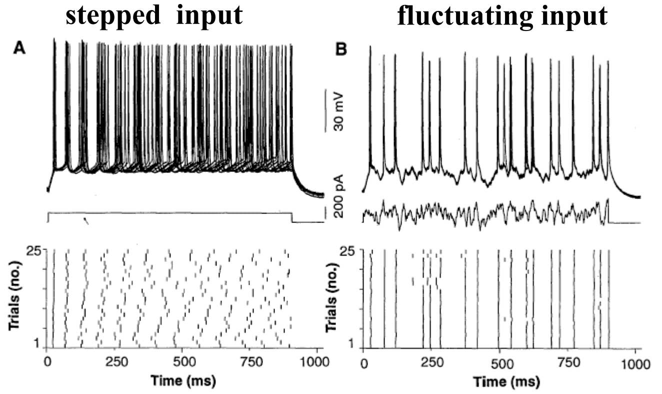

Fig. 1. Reliability of firing patterns of cortical neurons evoked by constant and fluctuating current. (**A**) In

Mainen and Sejnowski *data* from rat cortical neurons   Science 268 (1995) p. 1503

Spectrum of forcing matters – Hunter, Milton, and Cowan 1998
Explanation via Lyap. Exponents and phase models – Ritt 2003

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SUMMARY -- synchrony

- Can arise in several ways:
  - Recurrent coupling
  - Feedforward coupling
  - Coordinated inputs
- Uses:
  - Synchronized activity can be necessary to trigger "downstream" cells
  - Coupling and rhythms yield new computational strategies
    - See paper "We've got rhythm" by Nancy Kopell
    - Frequency-doubling and antiphase states (Rinzel, Golomb, Kopell)
    - Speech recognition (Hopfield and Brody) and may other applications!

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- extras

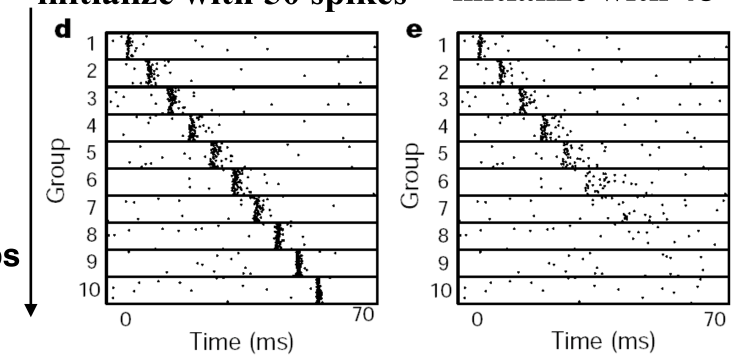
58

A key question…

- **ONCE a synchronized “burst” of activity has been generated, can it be stably propagated through *layers* of cortical tissue? Or will it “dissipate”?**

initialize with 50 spikes **initialize with 48**

Feed-fwd. ONLY connections between successive neural groups



Diesmann, Gewaltig, Aertsen
Nature 402, 529, 1999.

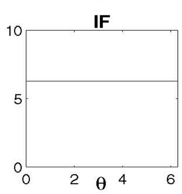
Answer: YES, if initiating spike volley suffic. large and synchronized

ABELES 1993 “synfire chains”

OK … let’s take the simplest imaginable case for z -- IF

PRC

z(θ)



$$\frac{d\theta_1}{dt} = \omega + z(\theta_1)\delta(t - t_2^j)$$
$$\frac{d\theta_2}{dt} = \omega + z(\theta_2)\delta(t - t_1^j)$$

Moral: coupling two neurons together does nothing if this coupling is not voltage (phase) dependent

Note that, if we introduced reversal potentials

$$I_{syn} = \delta\,(t-t^j)\,(V_{syn}-V)$$

into the above, would recover voltage dependence and hence coupling would have some synchronizing effect

Goal: simple phase description

natural frequency

$$\frac{d\theta}{dt} = \omega$$

- Let $\mathbf{x} = \begin{pmatrix} V \\ q \end{pmatrix}$. Then we have defined coordinate change so that:

$$\frac{d\mathbf{x}}{dt} = \mathbf{F}(\mathbf{x}) \quad \rightarrow \quad \frac{d\theta}{dt} = \omega$$

where $\mathbf{F}(\mathbf{x})$ is 'original' neural vectorfield giving oscillations at freq. ω.

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Goal: simple phase description

natural frequency

$$\frac{d\theta}{dt} = \underline{\omega + \cdots}$$

- Let $\mathbf{x} = \begin{pmatrix} V \\ q \end{pmatrix}$. Then we have defined coordinate change so that:

$$\frac{d\mathbf{x}}{dt} = \mathbf{F}(\mathbf{x}) \quad \rightarrow \quad \frac{d\theta}{dt} = \omega$$

where $\mathbf{F}(\mathbf{x})$ is 'original' neural vectorfield giving oscillations at freq. ω.

- We're actually interested in effects of additional currents: $J(\mathbf{x},t)$

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Goal: simple phase description

$$\frac{d\theta}{dt} = \underset{\text{natural frequency}}{\omega} + \cdots$$

- Let $\mathbf{x} = \begin{pmatrix} V \\ q \end{pmatrix}$. Then we have defined coordinate change so that:

$$\frac{d\mathbf{x}}{dt} = \mathbf{F}(\mathbf{x}) \quad \rightarrow \quad \frac{d\theta}{dt} = \omega$$

where $\mathbf{F}(\mathbf{x})$ is 'original' neural vectorfield giving oscillations at freq. ω.

- We're actually interested in effects of additional currents: $J(\mathbf{x},t)$ "perturbation"

$$\frac{d\mathbf{x}}{dt} = \mathbf{F}(\mathbf{x}) + \begin{pmatrix} J(\mathbf{x},t) \\ 0 \end{pmatrix} \quad \rightarrow \quad \frac{d\theta}{dt} = \frac{\partial\theta}{\partial\mathbf{x}} \cdot \left[\mathbf{F}(\mathbf{x}) + \begin{pmatrix} J(\mathbf{x},t) \\ 0 \end{pmatrix}\right]$$

$$\frac{d\theta}{dt} = \omega + \frac{\partial\theta}{\partial V} \; J(\mathbf{x},t)$$

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