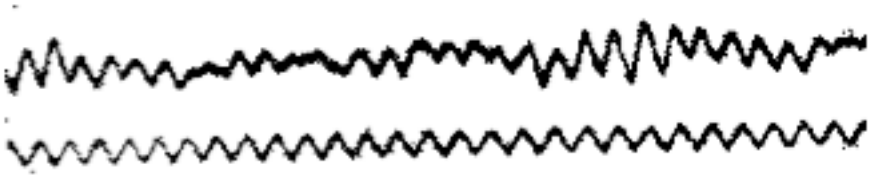
Models and Analysis of Brain Oscillations

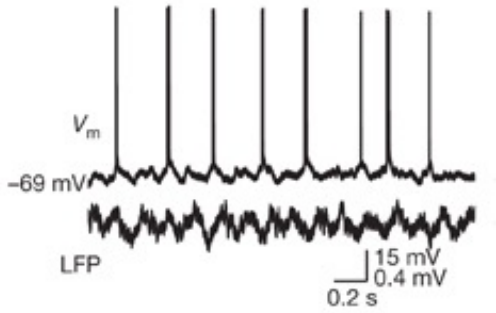
Boris Gutkin

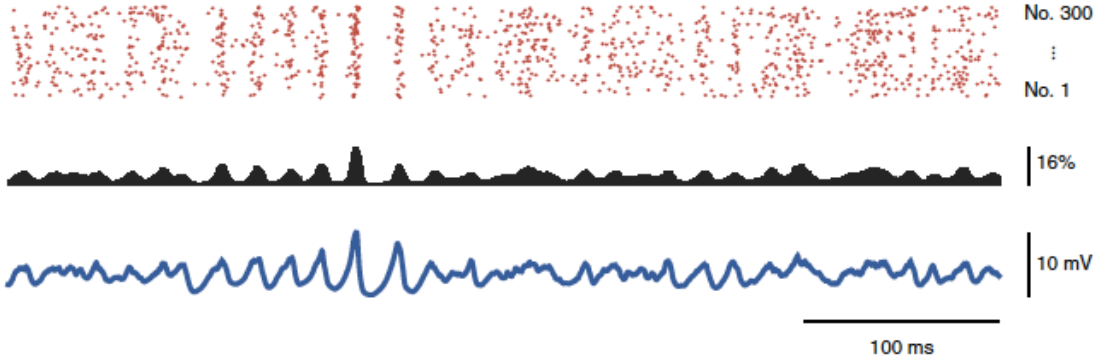
Group for Neural Theory, DEC-ENS-Paris

Oscillations is not a new thing

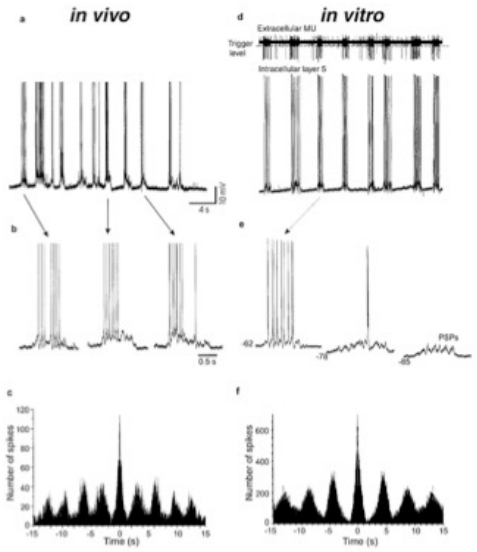


Hans Berger first published EEG (1929).



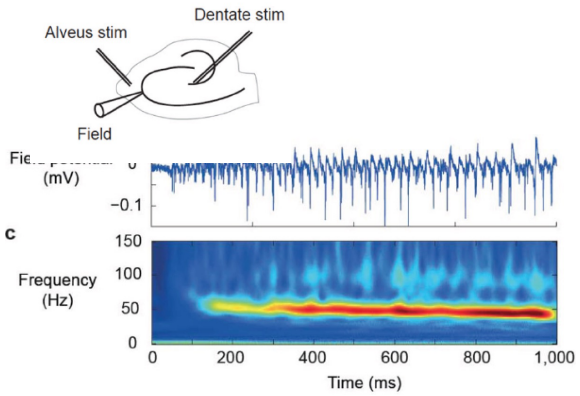


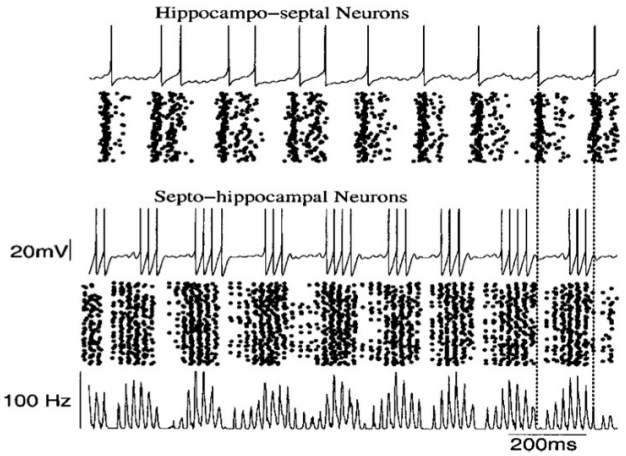
Up- states in neocortex in vivo and in vitro



Sanchez-Vives and McCormick, Nat. Neurosci. 3, 1027 (2000)

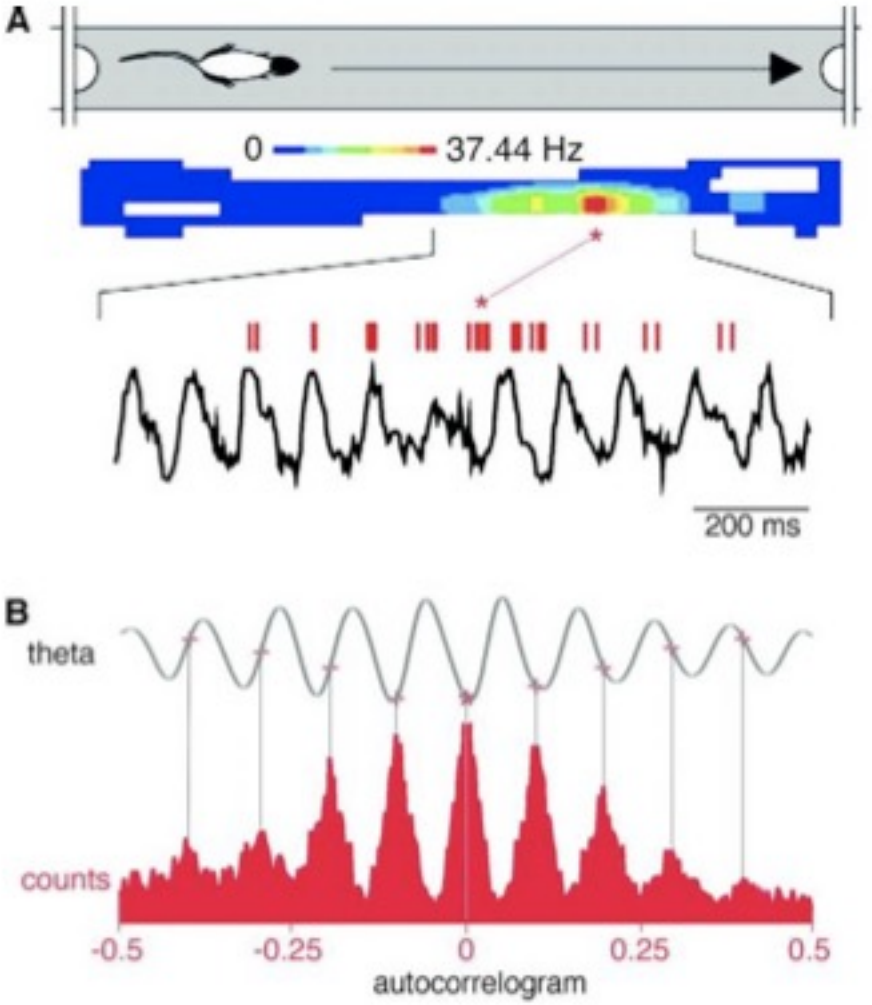
Gamma –oscillations in the hippocampal slice

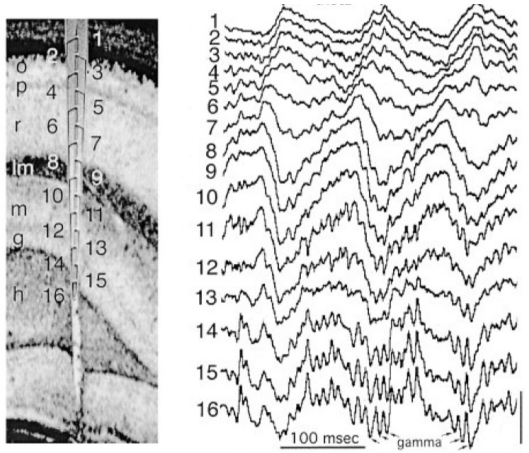




Wang (2002) – Pacemaker Neurons for the Theta Rhythm and Their Synchronization in the Septohippocampal Reciprocal Loop

Theta (5-10Hz) oscillations in the hippocampus

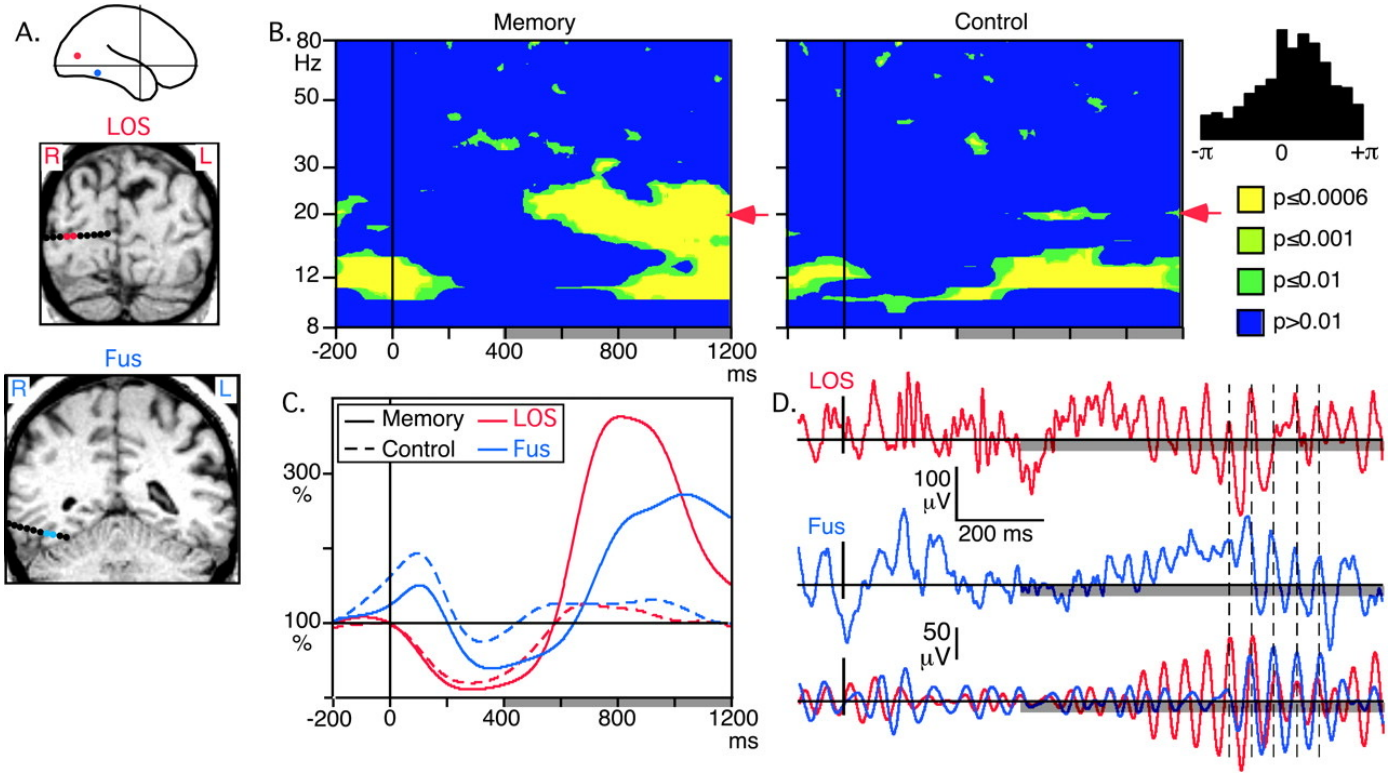




Buzsaki 2002

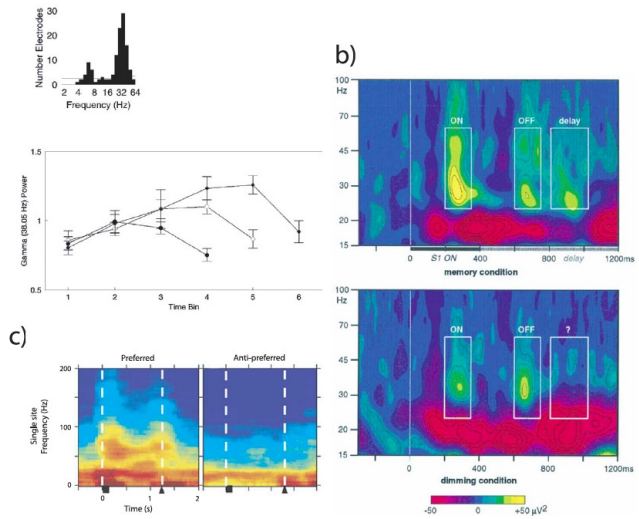
G. Buzsaki and A. Draguhn, Science 304, 1926 (2004)

Power and Synchrony Change Dynamically, In Task-Oriented Manner

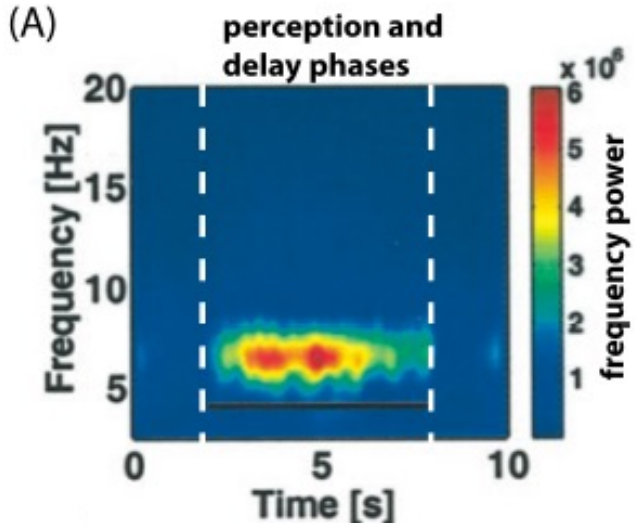


Tallon-Baudry et al , J. Neurosci 2001

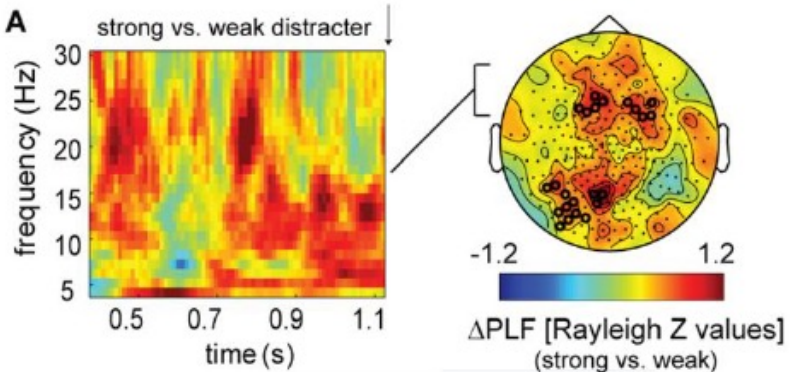
Oscillations in multiple bands seen during WM performance



Jensen 2006

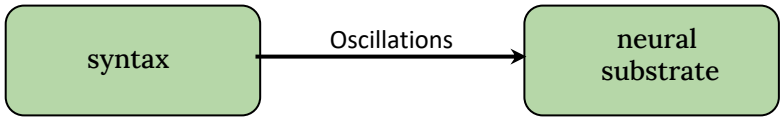


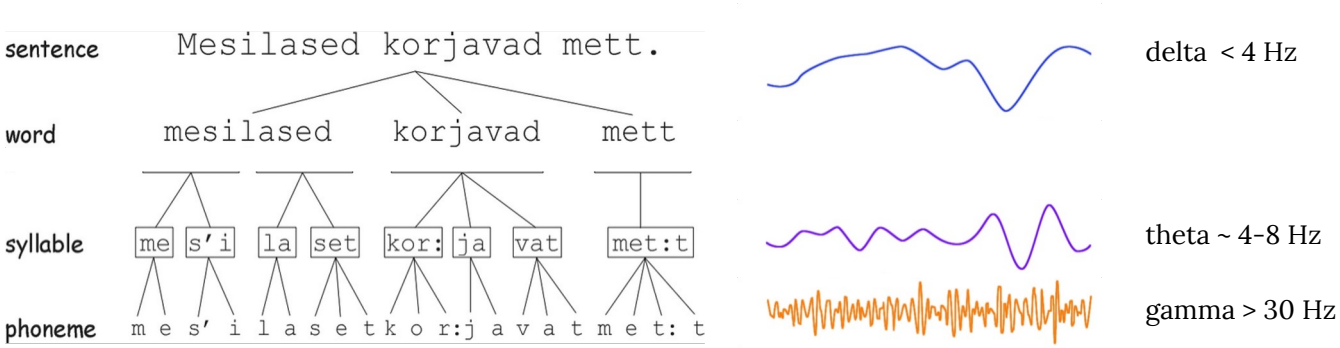
Human EEG
Raghavachariet al.
[2001]



Bennefond and Jensen Curr Biol 2012

Oscillations in Speech coding

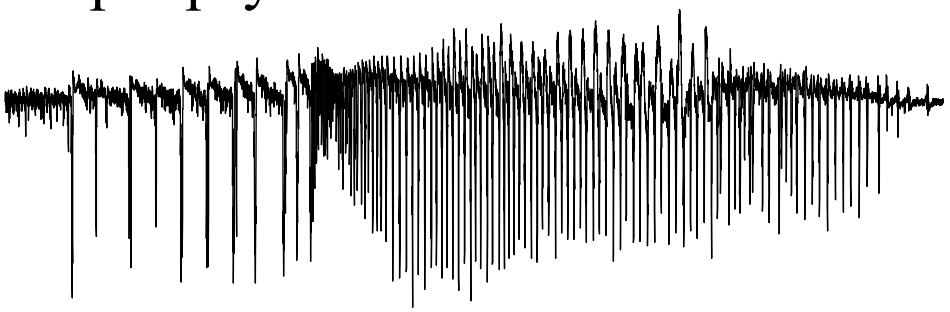




[1] D Poeppel, Cog. neuropsychology, 2012.
[2] AL Giraud & D Poeppel. Nature neuroscience, 2012.
[3] N Ding, et al. Nature neuroscience, 2016. ë

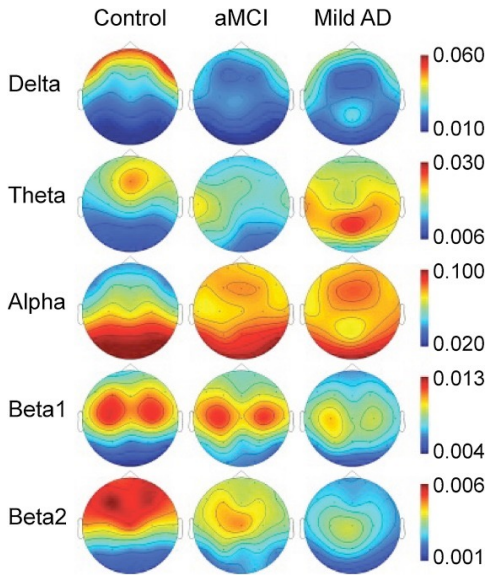
Oscillations in Pathologies

Epilepsy



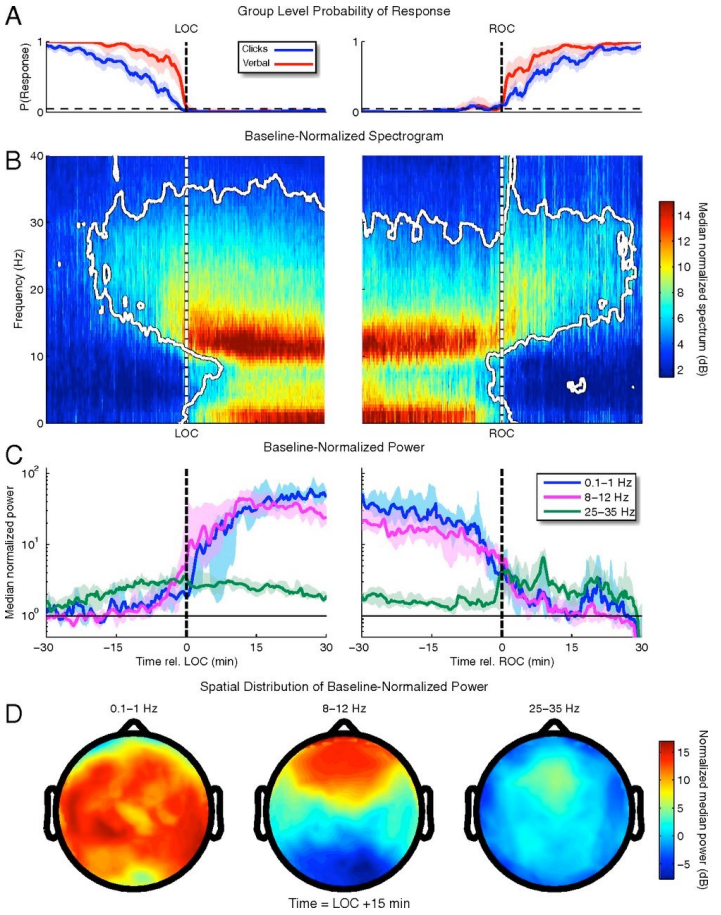
Huberfeld et al. 2011

MCI AD



Jafari et al 2020

Loss of consciousness



Purdon et al PNAS 2013

Alphabet Soup



- delta (1-4 Hz)
- theta (4-12)
- alpha (9-11) Hz
- beta (12-30)
- gamma (30-90)
- spindling (11-15)
- ripples (100 -200)
- slow (< 1 Hz)

- These are associated with a variety of cognitive states.
- Frequency ranges are somewhat arbitrary and overlapping.
- Needed: mechanisms! not (just) by frequency
- Needed: Interactions cross-rhythms
- Needed: Function?

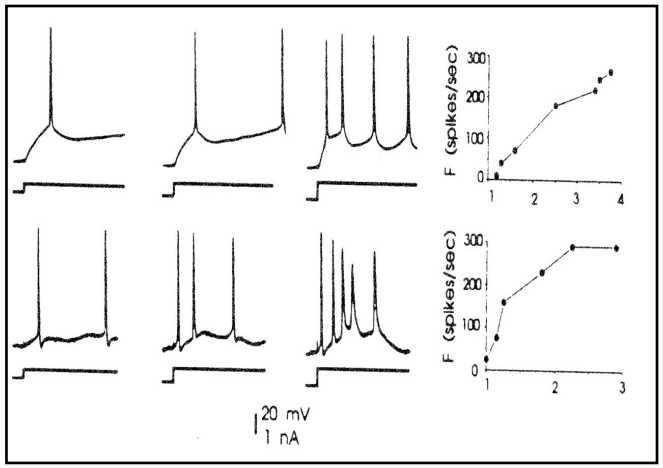
Game Plan:

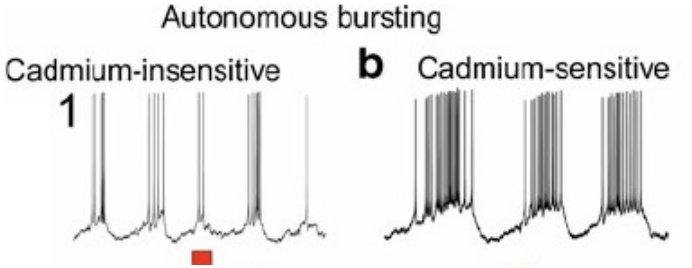
- Part 1: Mechanisms and basic analysis
  - Excitability in single neurons
  - Reduction to planar models
  - **Onset of oscillations: generic bifurcations**

- Part2: Oscillations in circuits and networks:
  - phase response approach
  - Weakly-coupled oscillator theory
  - Exact reduced models
  - Analysis of Communication Through Coherence

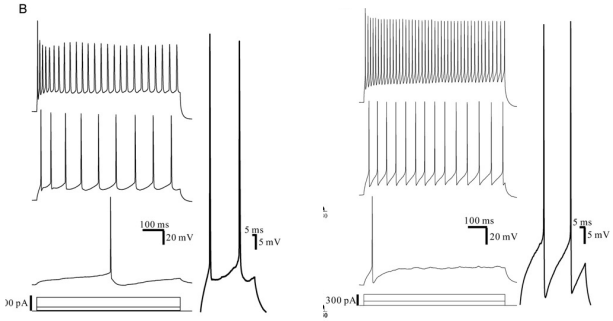
Neurons are non-linear oscillators:

when stimulated with current injection neurons respond with repetitive discharges



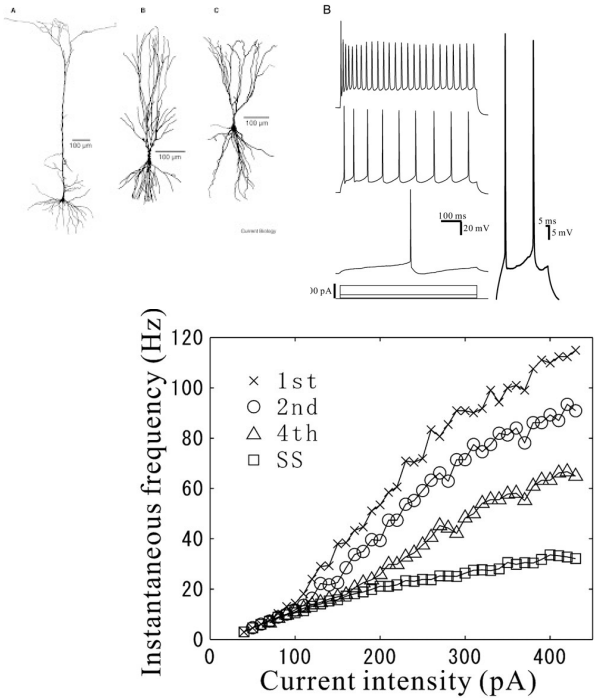


Ramirez et al 2011 JBP



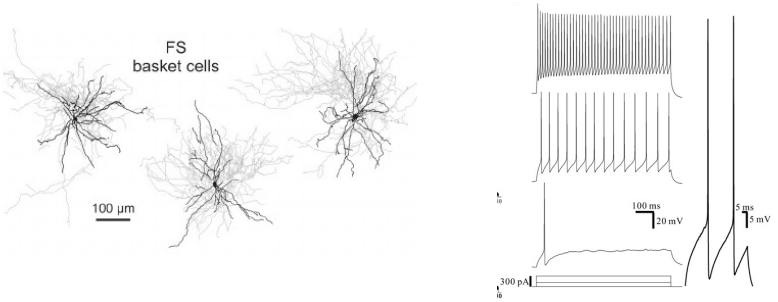
Tateno & Robinson 2005 JNeuroPhys

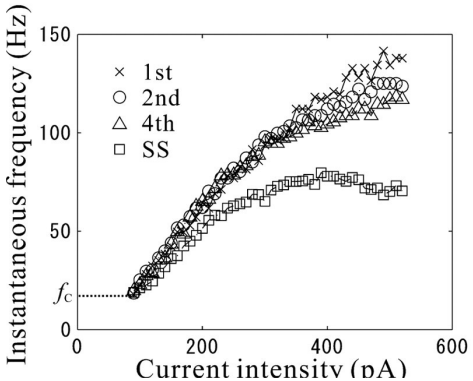
Pyramidal

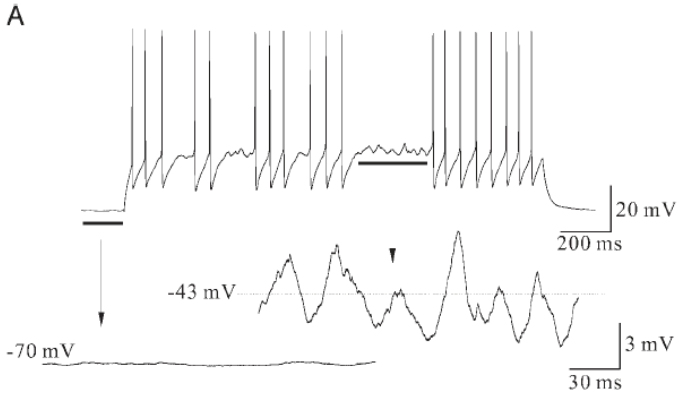


Tateno & Robinson 2005 JNeuroPhys

Fast-spiking interneurons





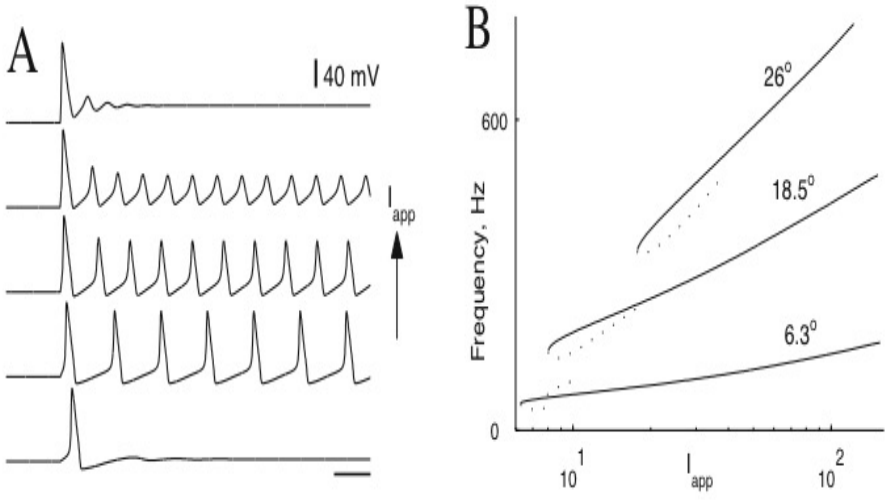


Historical Antecedents

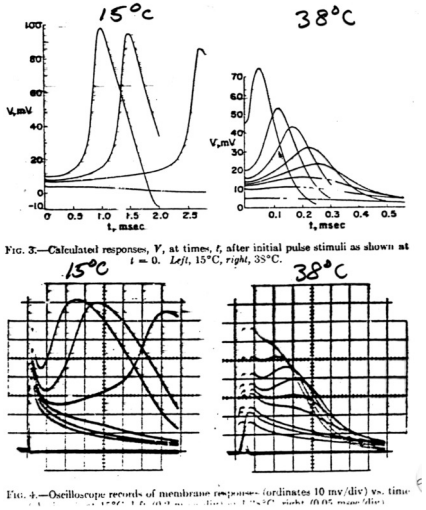
What Nobel-laureate giants saw in squid giant axons.

H  H

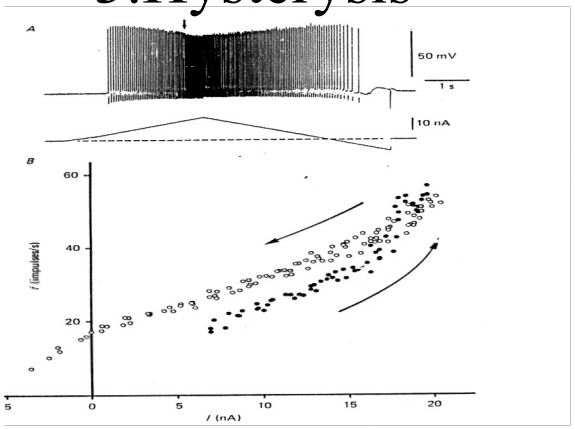
1.Minimal frequency



2.Graded responses

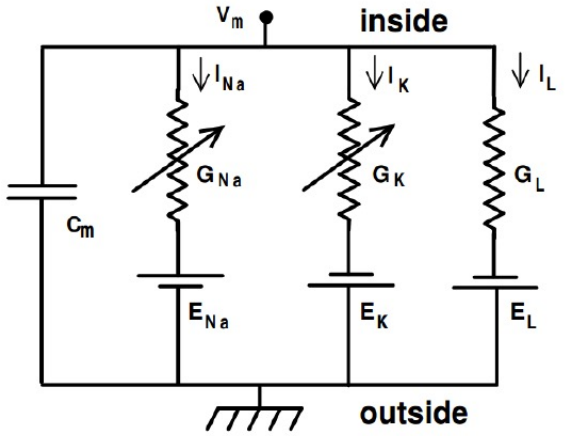


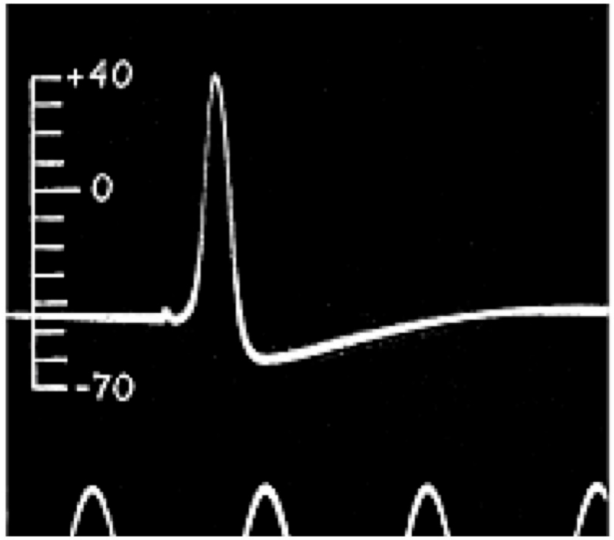
3.Hysterysis



The Hodgkin-Huxley (HH) Model

$$
\begin{aligned}
C\frac{dv}{dt} &= I - g_{Na}m^3h(V - V_{Na}) - g_K n^4(V - V_K) - g_L(V - V_L) \\
\frac{dm}{dt} &= a_m(V)(1-m) - b_m(V)m \\
\frac{dh}{dt} &= a_h(V)(1-h) - b_h(V)h \\
\frac{dn}{dt} &= a_n(V)(1-n) - b_n(V)n \\
a_m(V) &= .1(V+40)/(1-\exp(-(V+40)/10)) \\
b_m(V) &= 4\exp(-(V+65)/18) \\
a_h(V) &= .07\exp(-(V+65)/20) \\
b_h(V) &= 1/(1+\exp(-(V+35)/10)) \\
a_n(V) &= .01(V+55)/(1-\exp(-(V+55)/10)) \\
b_n(V) &= .125\exp(-(V+65)/80)
\end{aligned}
$$

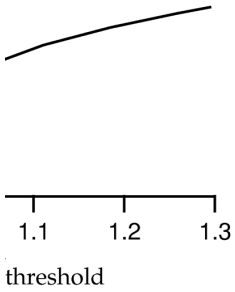


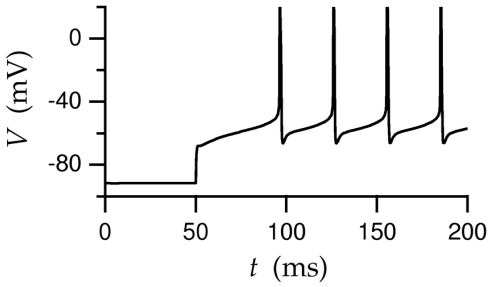


Dissection of AP:
See John Rinzel's talks

The Hodgkin-Huxley (HH) Model

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a_h(V) &= .07\exp(-(V + 65)/20) \\
b_h(V) &= 1/(1 + \exp(-(V + 35)/10)) \\
a_n(V) &= .01(V + 55)/(1 - \exp(-(V + 55)/10)) \\
b_n(V) &= .125\exp(-(V + 65)/80)
\end{aligned}
$$

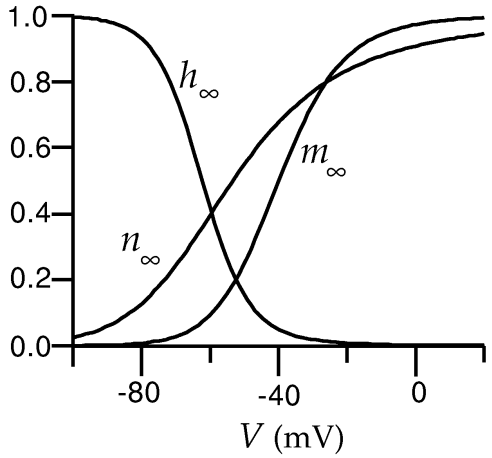


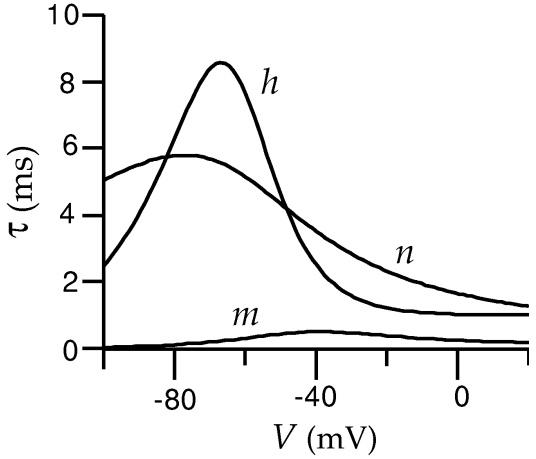


The Hodgkin-Huxley (HH) Model

Reduction to 2-d

$$
\begin{aligned}
C\frac{dv}{dt} &= I - g_{Na}m^3h(V - V_{Na}) - g_K n^4(V - V_K) - g_L(V - V_L) \\
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a_n(V) &= .01(V + 55)/(1 - \exp(-(V + 55)/10)) \\
b_n(V) &= .125\exp(-(V + 65)/80)
\end{aligned}
$$





$$C\frac{dV}{dt} + I_{ion}(V, W_1, ..W_n) = I(t)$$

$$\frac{dW_i}{dt} = \phi\frac{W_{i,\infty} - W_i}{\tau_i(V)}$$

$$I_j = \bar{g}_j\sigma_j(V, W_1, ..., W_n)(V - V_j)$$

$m = m_\infty$

$n = b - an$

$\tau_m = 0$

$\tau_n = \tau_h$

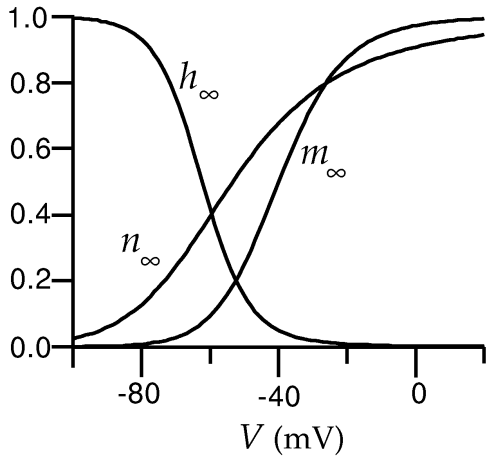
The Hodgkin-Huxley (HH) Model

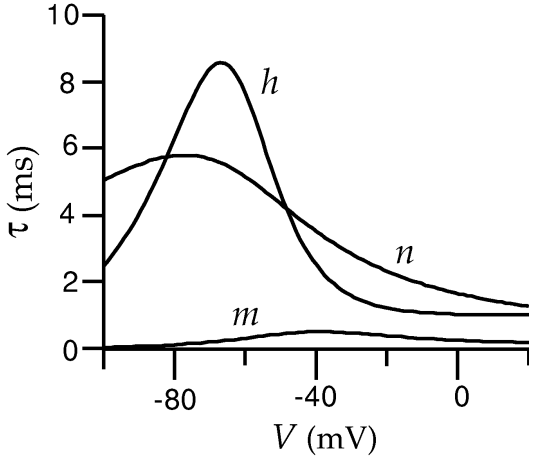
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a_n(V) &= .01(V + 55)/(1 - \exp(-(V + 55)/10)) \\
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\end{aligned}
$$

$$\frac{du}{dt} = \frac{1}{\tau}\left[F(u,w) + IR\right]$$

$$\frac{dW}{dt} = \frac{1}{\tau_w}G(u,w)$$





$m = m_\infty$

$n = b - an$

$w = b - h = a\,n$

$\tau_m = 0$

$\tau_n = \tau_h = \tau_w$

The Hodgkin-Huxley (HH) Model

Reduction to 2-d

$$
\begin{aligned}
C\frac{dv}{dt} &= I - g_{Na}m^3h(V - V_{Na}) - g_K n^4(V - V_K) - g_L(V - V_L) \\
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$$

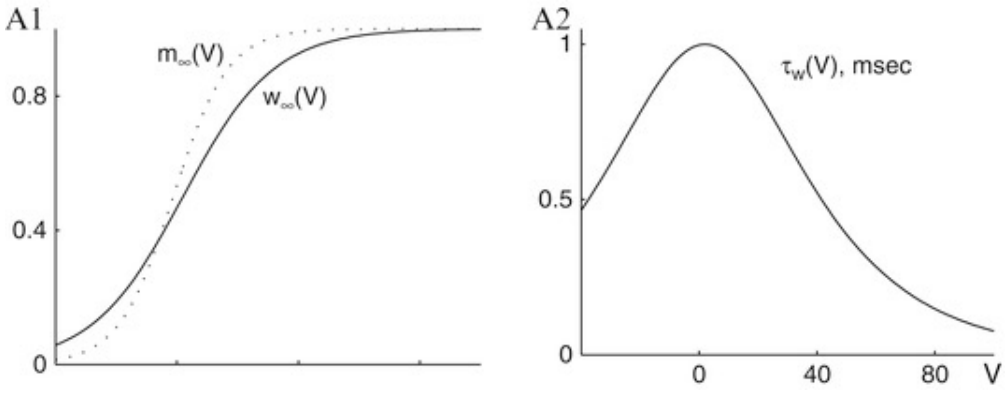
e.g. from Rinzel 1985

$$
\begin{aligned}
C\dot{V} = I_{\text{app}} &- \bar{g}_{\text{Na}} m_\infty^3(V)(1-W) \\
&\times (V - V_{\text{Na}}) - \bar{g}_{\text{K}}(W/S)^4(V - V_{\text{K}}) \\
&- g_L(V - V_L) \qquad (1)
\end{aligned}
$$

$$
\dot{W} = \phi[W_\infty(V) - W]/\tau(V) \qquad (2)
$$

$$
\frac{du}{dt} = \frac{1}{\tau}\left[F(u,w) + IR\right]
$$

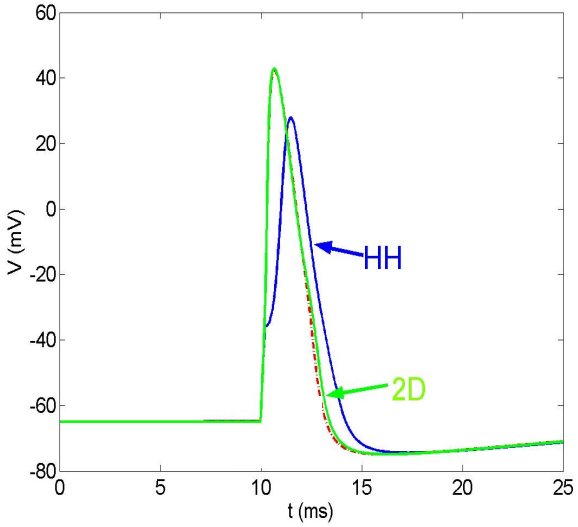
$$
\frac{dW}{dt} = \frac{1}{\tau_w}G(u,w)
$$



The HH Model

HH 4d to 2d

$$
\begin{aligned}
C\frac{dv}{dt} &= I - g_{Na}m^3h(V - V_{Na}) - g_K n^4(V - V_K) - g_L(V - V_L) \\
\frac{dm}{dt} &= a_m(V)(1-m) - b_m(V)m \\
\frac{dh}{dt} &= a_h(V)(1-h) - b_h(V)h \\
\frac{dn}{dt} &= a_n(V)(1-n) - b_n(V)n \\
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b_n(V) &= .125\exp(-(V+65)/80)
\end{aligned}
$$



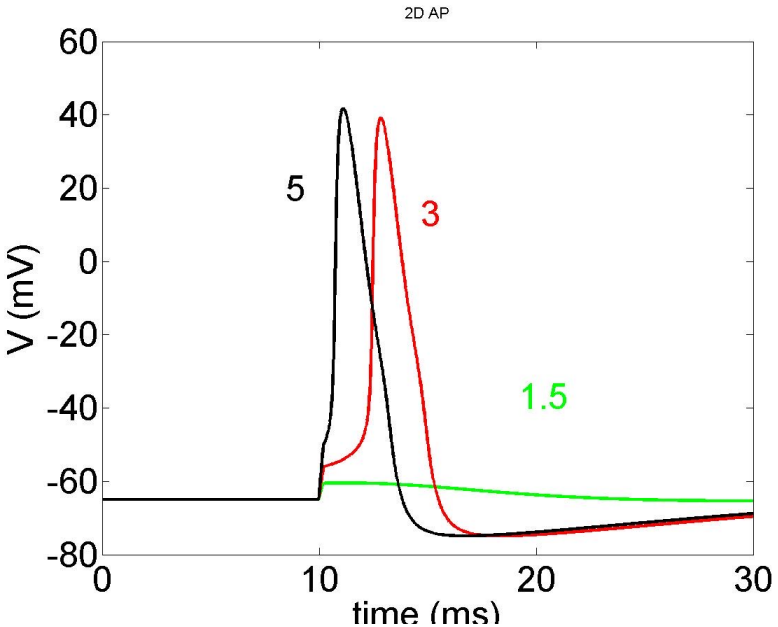
$$\frac{du}{dt} = \frac{1}{\tau}\left[F(u,w) + IR\right]$$

$$\frac{dW}{dt} = \frac{1}{\tau_w}G(u,w)$$

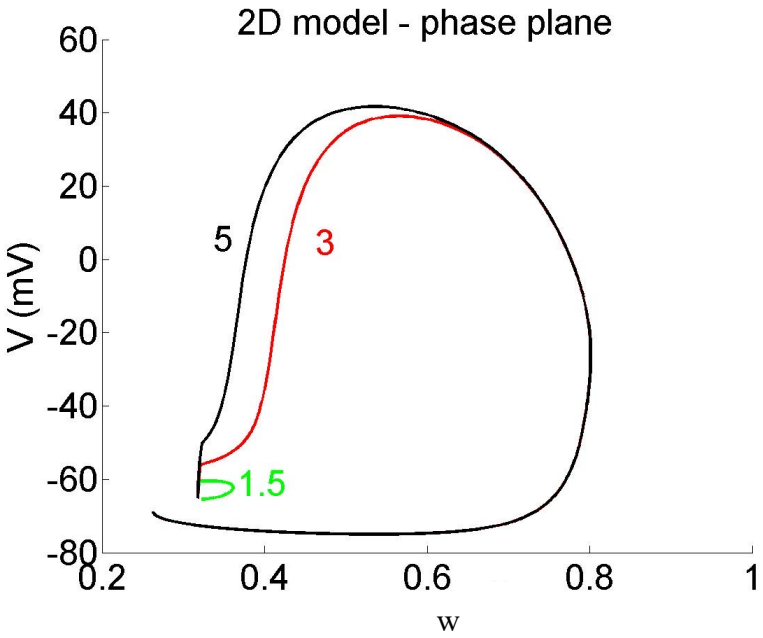
Different assumptions on *F(u,w), G(u,w)*
Different 2-d models

2-d HH: Phase Planes!

AP in time



Plot V vs. w



Change the initial voltage

Phase Plane!

Analysis: Phase plane

Lets consider a simple example

Notice that the RHS or the voltage equation is roughly cubic
Lets assume that the *G(u,w)* is linear; set τ_w to be constant

$$\frac{du}{dt} = \frac{1}{\tau}\left[F(u,w) + IR\right]$$

$$\frac{dW}{dt} = \frac{1}{\tau_w} G(u,w)$$

$$C\dot{V} = I_{\text{app}} - \bar{g}_{\text{Na}} m_\infty^3(V)(1 - W) \times (V - V_{\text{Na}}) - \bar{g}_{\text{K}}(W/S)^4(V - V_{\text{K}}) - g_L(V - V_L) \quad (1)$$

$$\dot{W} = \phi[W_\infty(V) - W]/\tau(V) \quad (2)$$

Fitzhugh-Nagumo (FHN)

Analysis: Phase plane

Fitzhugh-Nagumo (FHN)

$$\frac{du}{dt}=\frac{1}{\tau}\left[F(u,w)+IR\right] \qquad F(u,w)=u-\frac{1}{3}u^3-w,$$

$$\frac{dW}{dt}=\frac{1}{\tau_w}G(u,w) \qquad G(u,w)=b_o+b_1 u-w$$

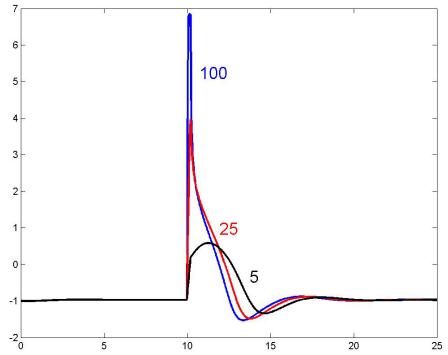
Analysis: Phase plane

Let's consider a simple example: Fitzhugh-Nagumo (FHN)

Notice that the RHS or the voltage equation is roughly cubic
Lets assume that the *G(u,w)* is linear

$$\frac{du}{dt} = \frac{1}{\tau}\left(u - \frac{1}{3}u^3 - w\right)$$

$$\frac{dW}{dt} = b_o + b_1 u - w$$



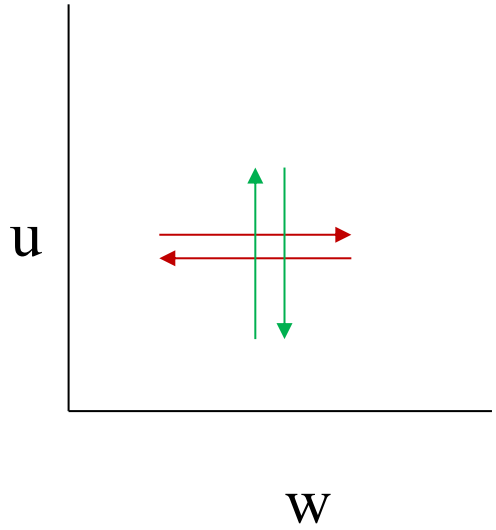
FHN Analysis: Phase plane

Plot characteristic curves: nullclines

$$\frac{du}{dt}=0$$ *u* does not change!

$$\frac{dw}{dt}=0$$ *w* does not change!

$$\frac{du}{dt}=\frac{dw}{dt}=0$$ Fixed point!



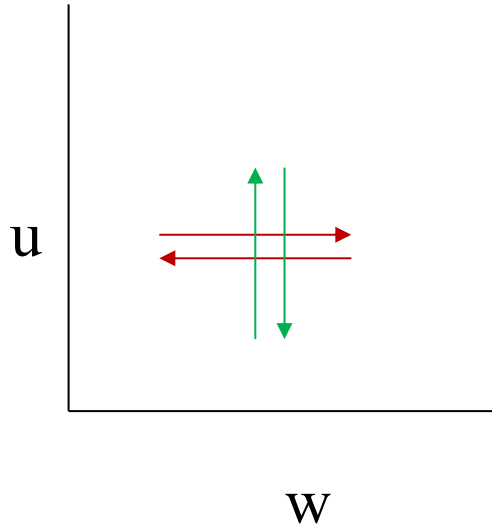
FHN Analysis: Phase plane

Plot characteristic curves: nullclines

$$\frac{du}{dt}=0$$ *u* does not change!

$$\frac{dw}{dt}=0$$ *w* does not change!

$$\frac{du}{dt}=\frac{dw}{dt}=0$$ Fixed point!



FP Question: which are stable? Unstable?

FHN Analysis: Phase plane

Plot characteristic curves: nullclines

$$\frac{du}{dt}=0 \quad \frac{dw}{dt}=0$$

$$(1)\, w = u - \frac{1}{3}u^3$$

$$(2)\, w = b_0 + b_1 u$$

FHN Analysis: Phase plane

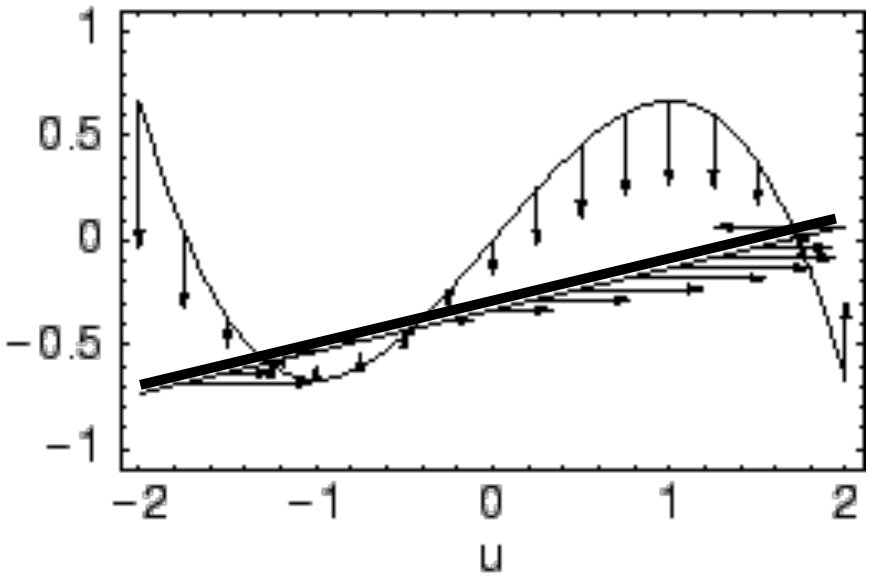
Plot characteristic curves: nullclines

$$\frac{du}{dt}=0 \quad \frac{dw}{dt}=0$$

$$(1)\, w = u - \frac{1}{3}u^3$$

$$(2)\, w = b_0 + b_1 u$$

Stable points
Saddles
Unstable Points
Oscillations



FHN Analysis: Phase plane

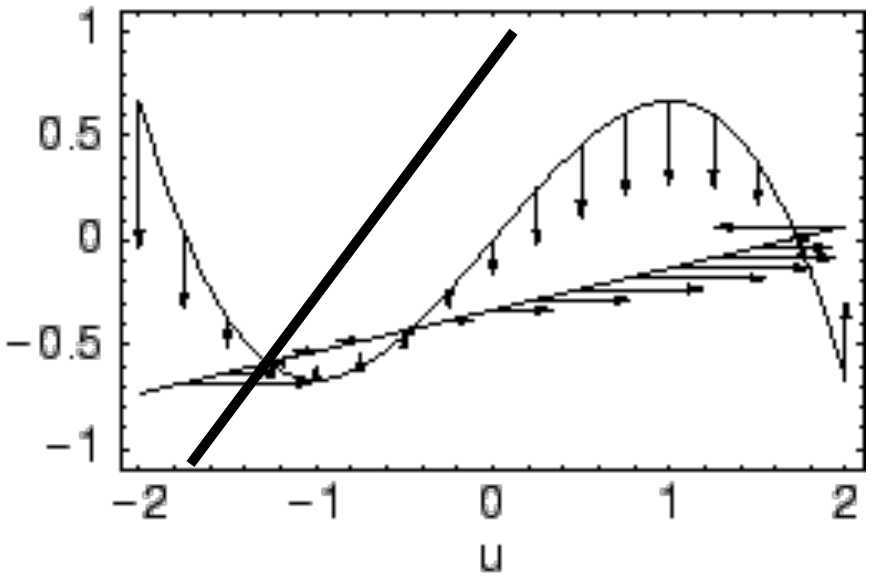
Plot characteristic curves: nullclines

$$\frac{du}{dt}=0 \quad \frac{dw}{dt}=0$$

$$(1)\, w = u - \frac{1}{3}u^3$$

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Stable points
Saddles
Unstable Points
Oscillations



FHN Analysis: Phase plane

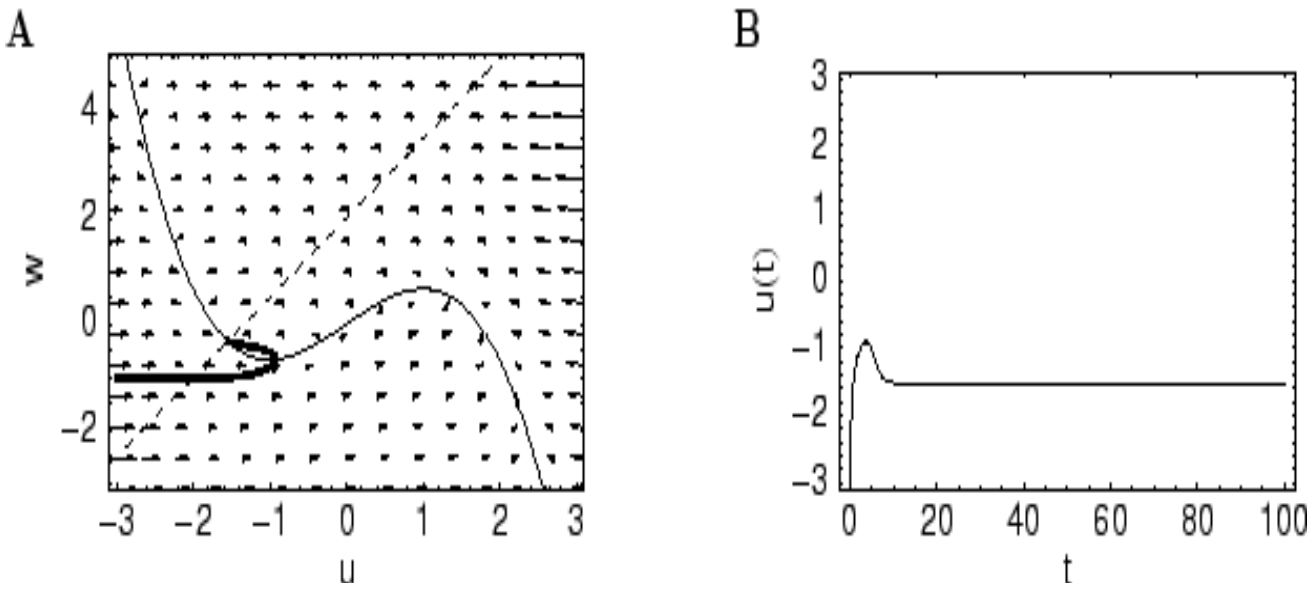
Plot characteristic curves: nullclines

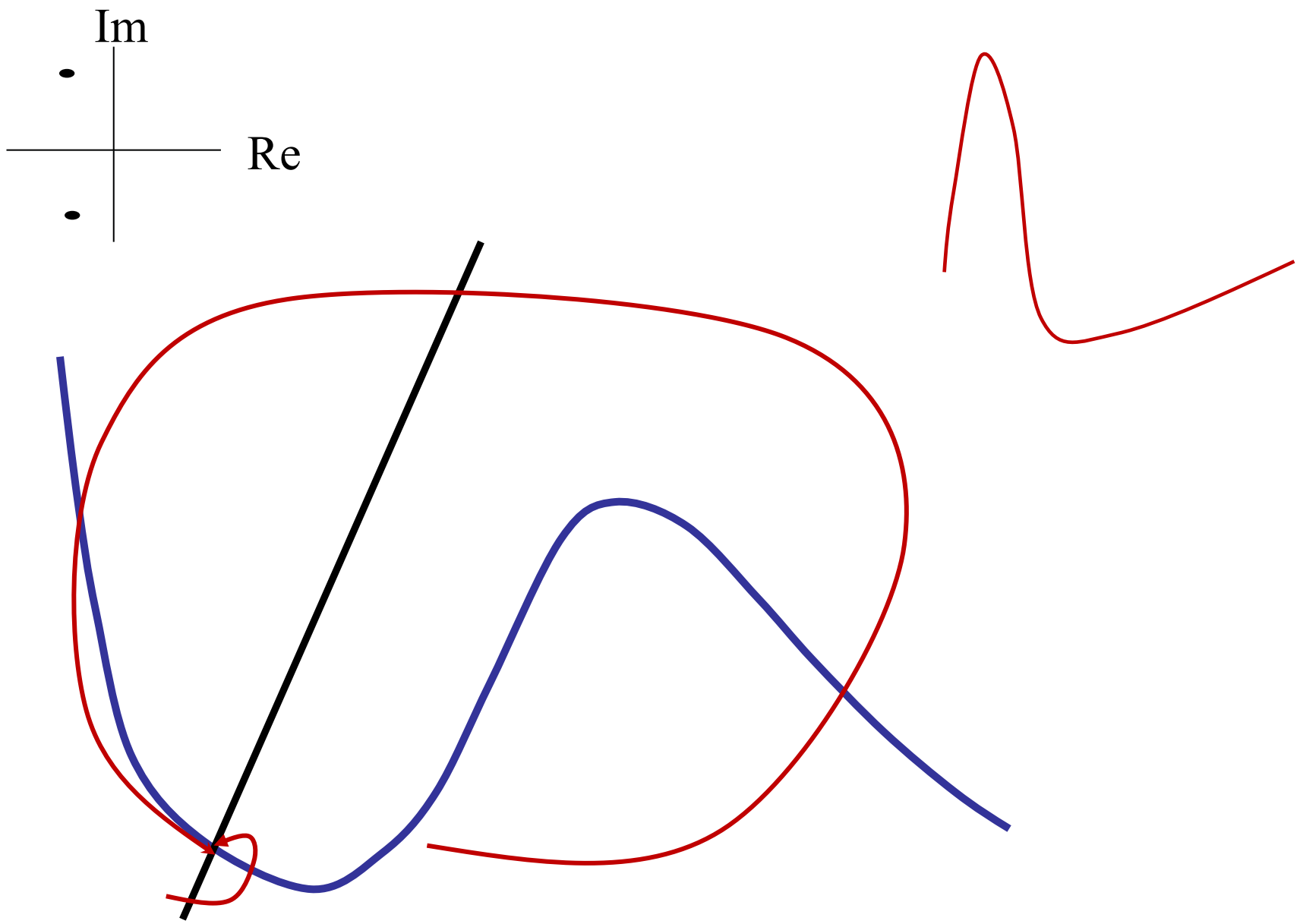
$$\frac{du}{dt}=0 \quad \frac{dw}{dt}=0$$

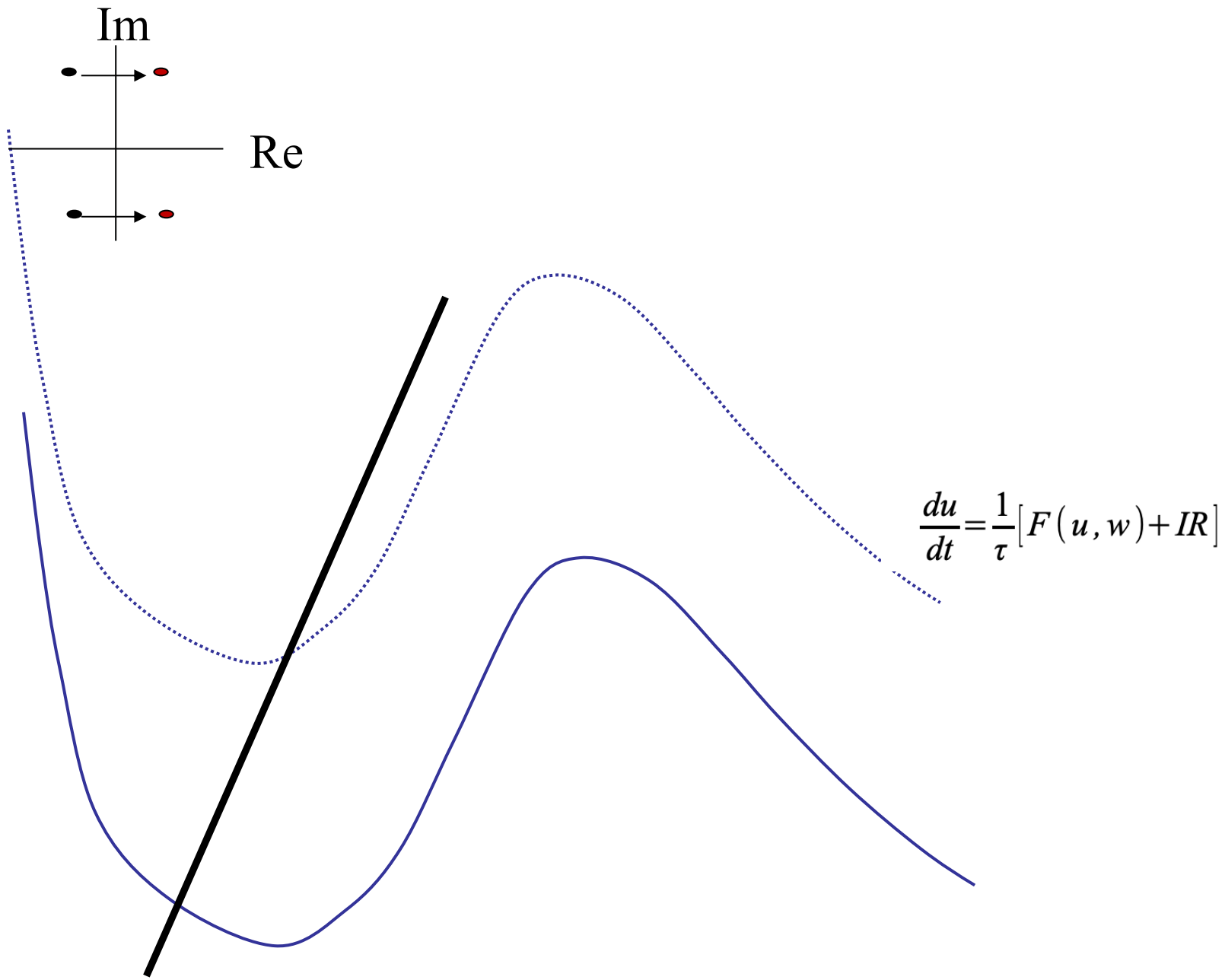
$$(1)\, w = u - \frac{1}{3}u^3$$

$$(2)\, w = b_0 + b_1 u$$

Stable points

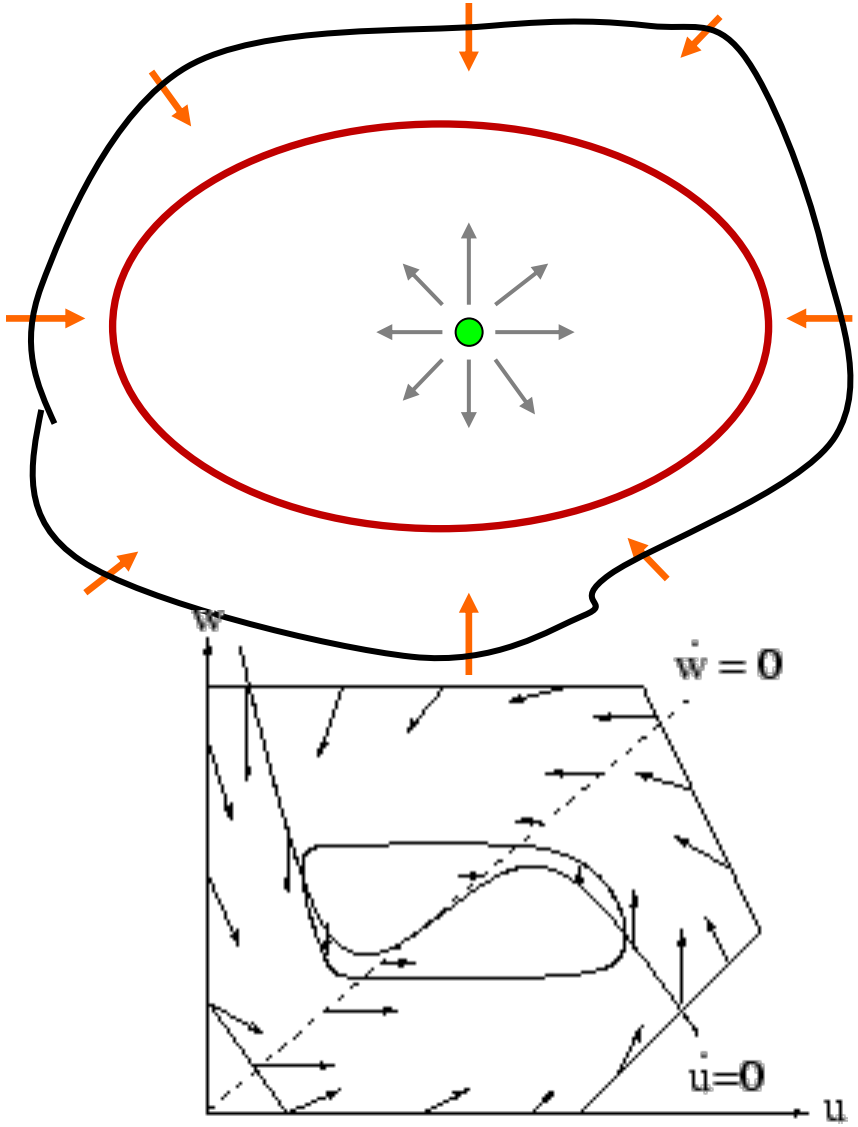


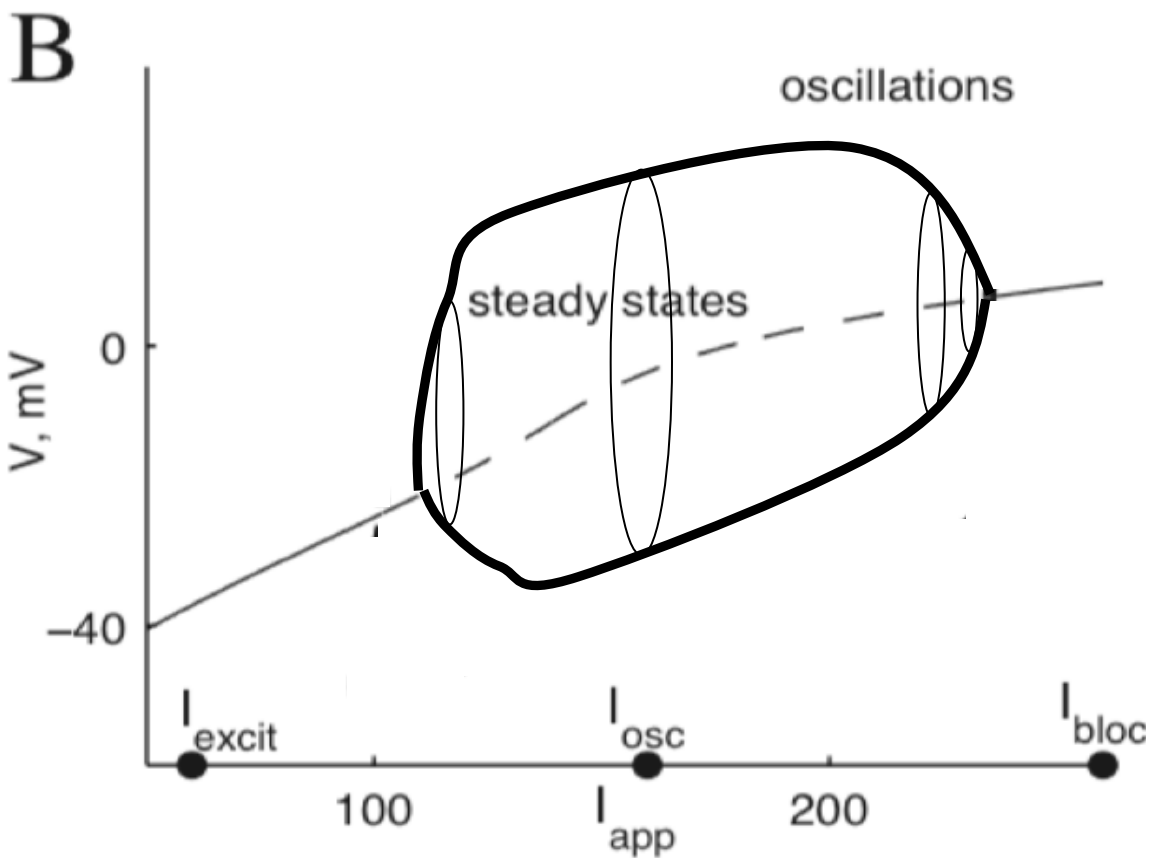

Im
Re

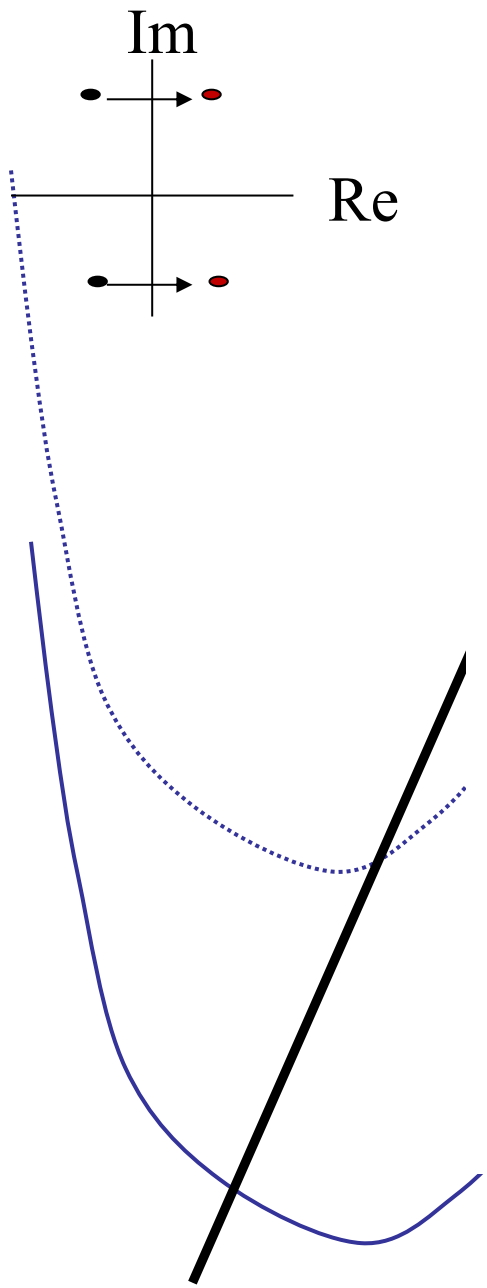

Im
Re
$\frac{du}{dt} = \frac{1}{\tau}\left[F(u,w) + IR\right]$

Poincare-Bendixon Theorem

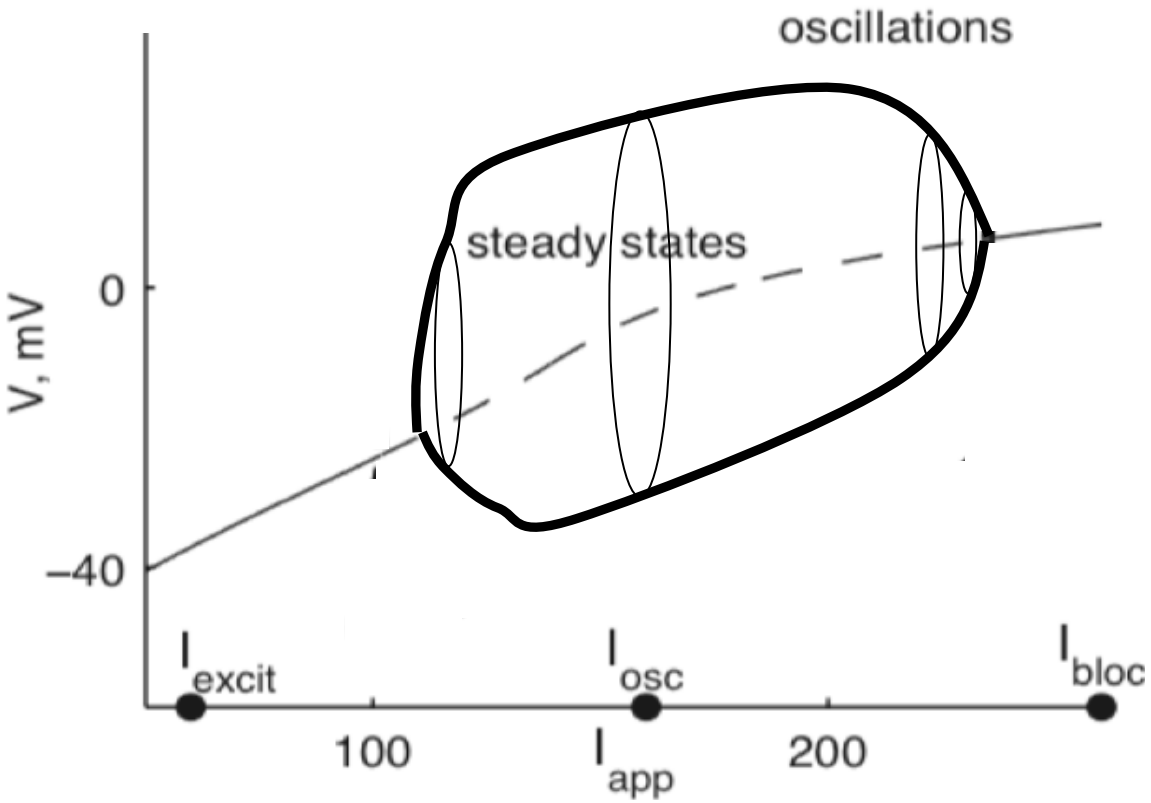
- Dynamical System
- Closed Domain
- No stable point
- Must have a stable solution



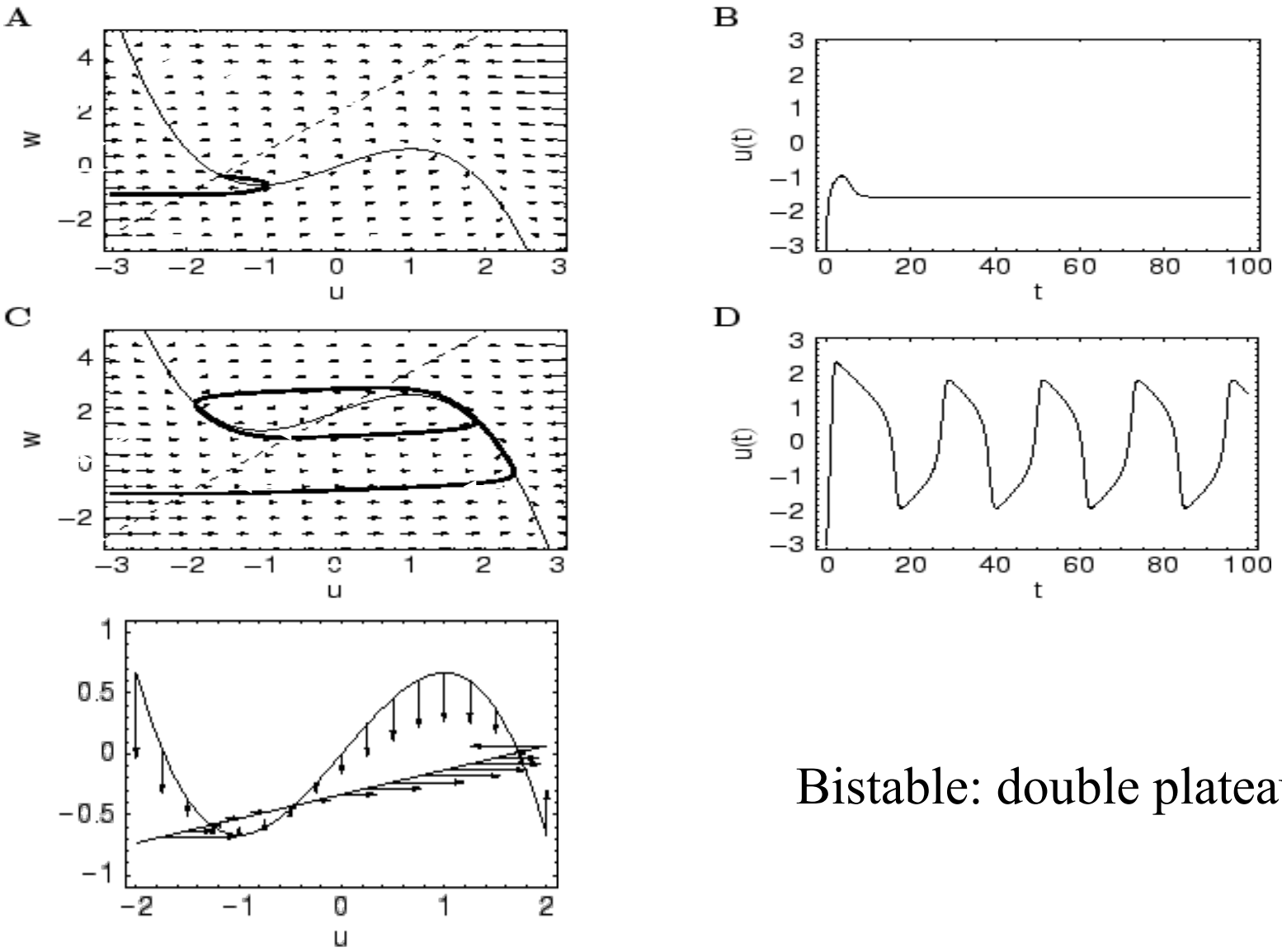

B
oscillations
steady states
0
V, mV
–40
I excit
I osc
I bloc
100
I app
200



Super-critical Andronov-Hopf



FHN Analysis: Phase plane



Bistable: double plateau

Lets relax the linear *G(u,w)* assumption: another 2-d model

$$C\frac{dV}{dt} = -g_{ca} * m_\infty(V) * (V - V_{Ca}) - g_K * w * (V - V_K) - g_L * (V - V_L) + I_{app}$$

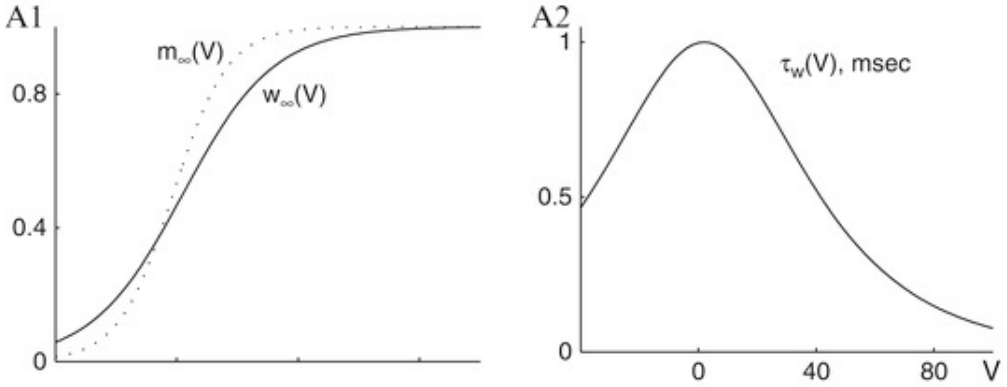
$$\frac{dw}{dt} = f * \frac{w_\infty(V) - w}{\tau(V)}$$

V denotes the membrane potential and w the fraction of open K^+ channels. The functions $m_\infty(V), w_\infty(V), \tau(V)$ are defined as follows:

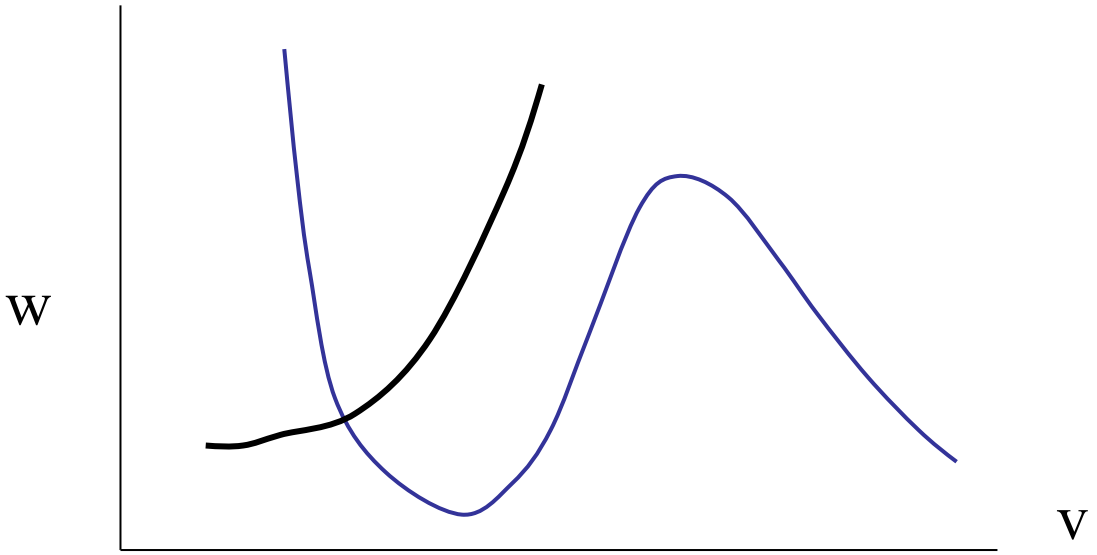
$$m_\infty(V) = 0.5 * [1 + \tanh(\frac{V - v_1}{v_2})]$$

$$w_\infty(V) = 0.5 * [1 + \tanh(\frac{V - v_3}{v_4})]$$

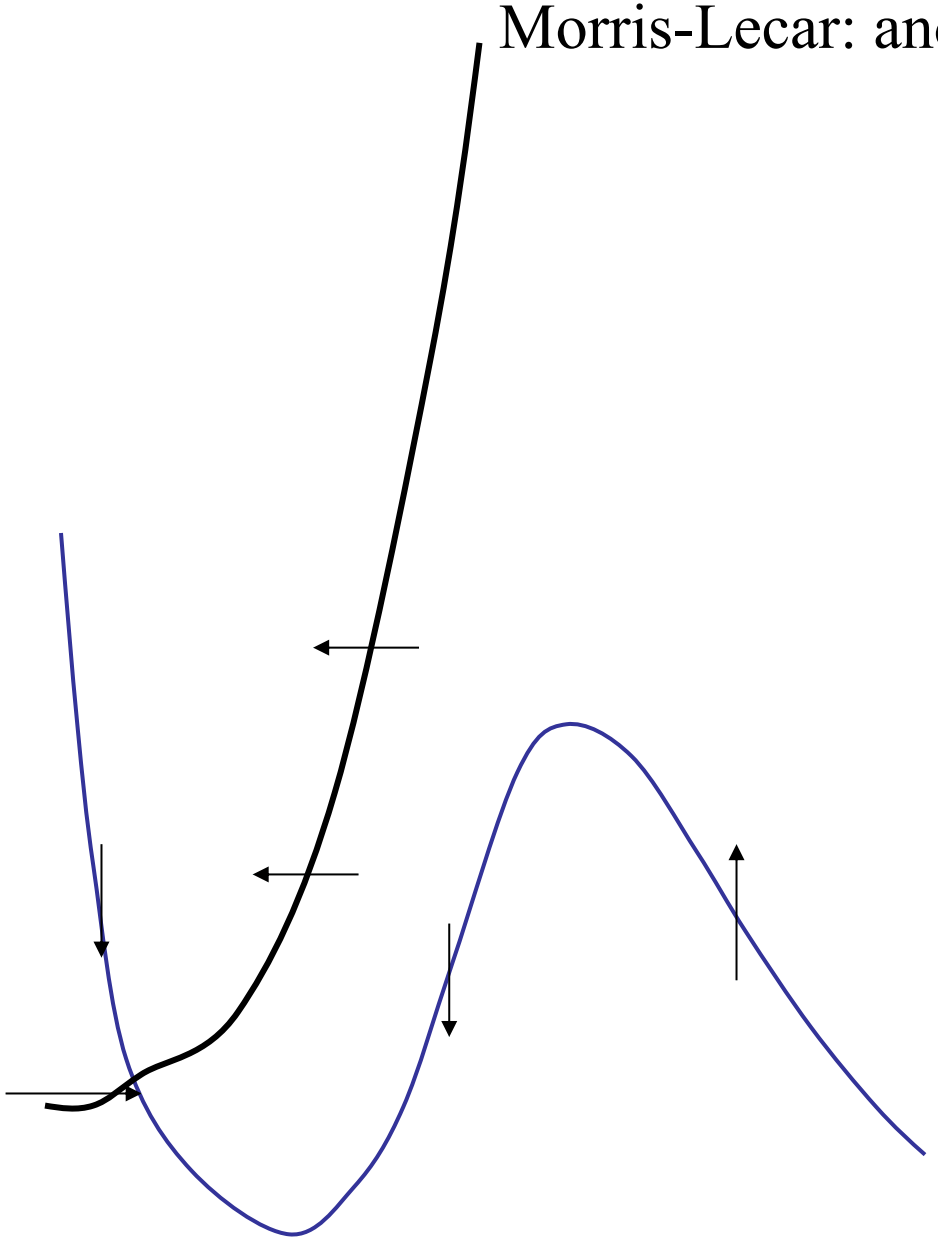
$$\tau(V) = \frac{1}{\cosh\left(\frac{V - v_3}{2 * v_4}\right)}$$

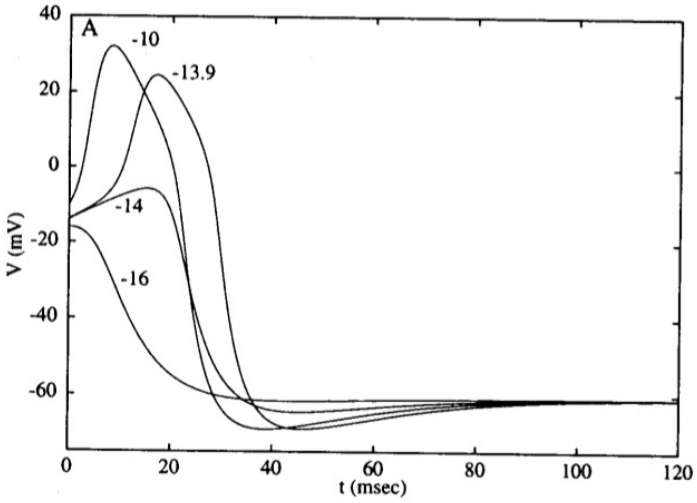


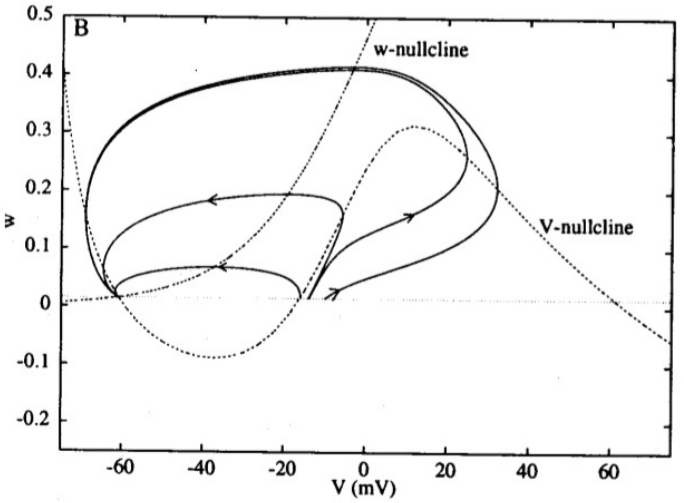
Morris-Lecar



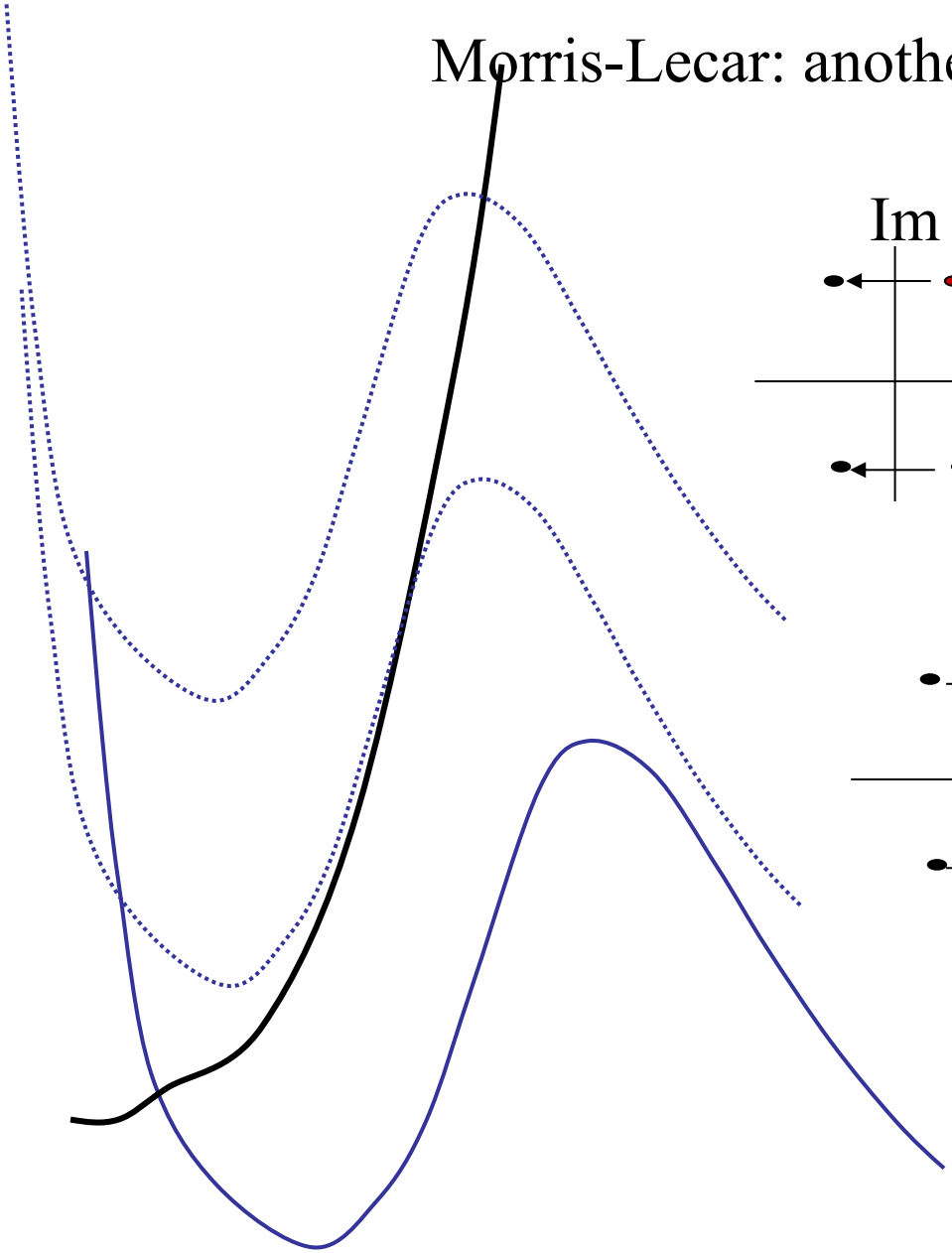
Morris-Lecar: another 2-d model

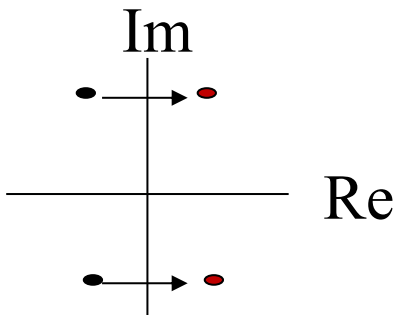




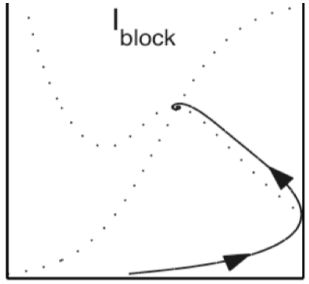


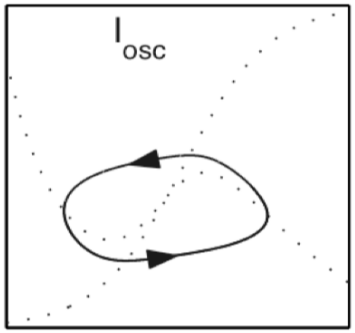
Morris-Lecar: another 2-d model

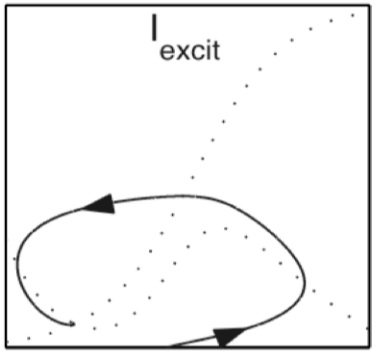




Plot: voltage states as a function of the I

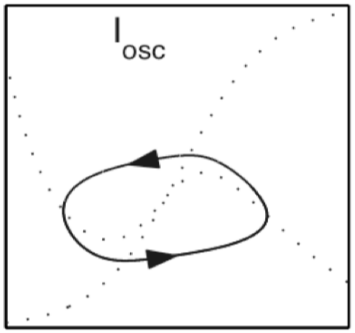






Morris-Lecar: another 2-d model

oscillatory

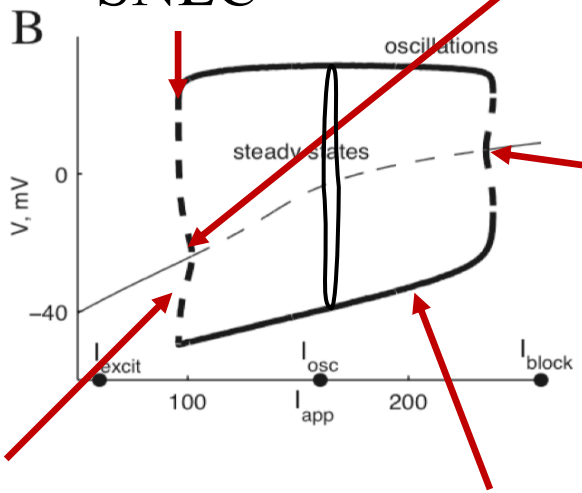


Im

Re

Hopf Bifurcations

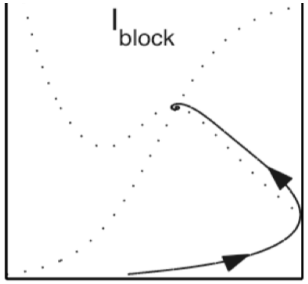
SNLC



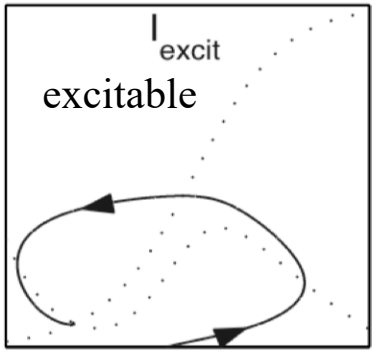
Im

Re

depolarization block

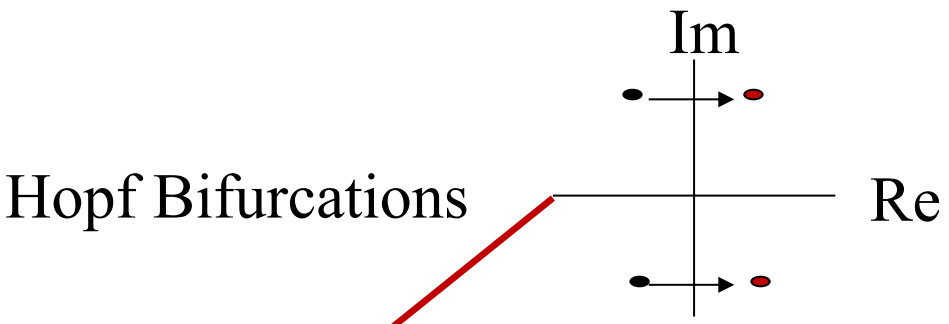


Unstable LC

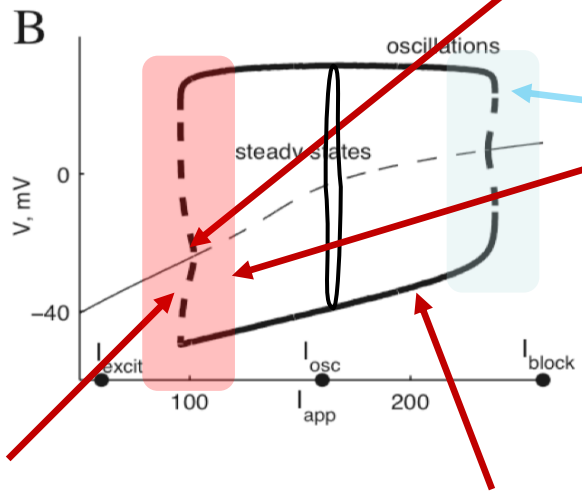


Stable Limit Cycle

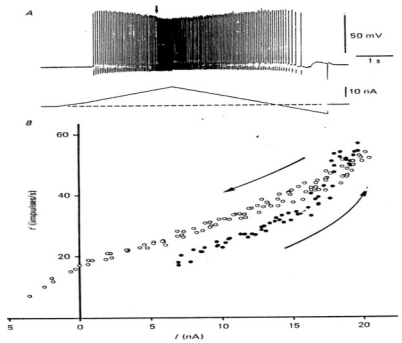
Morris-Lecar: another 2-d model

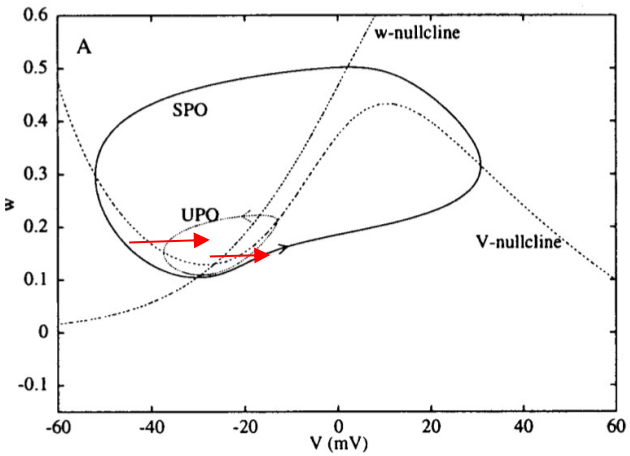


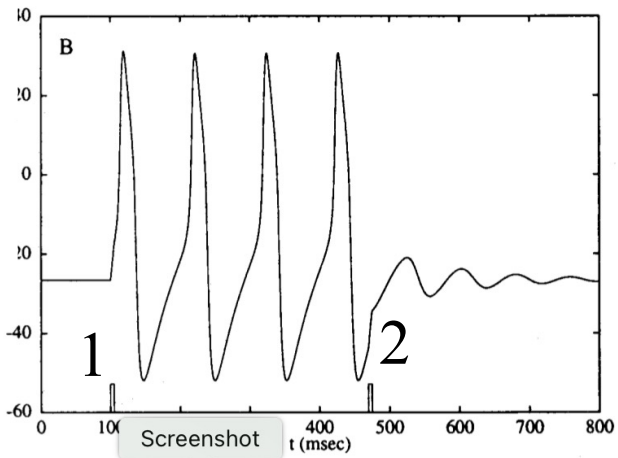
Hopf Bifurcations



Bistability







Unstable LC

Stable Limit Cycle

Screenshot

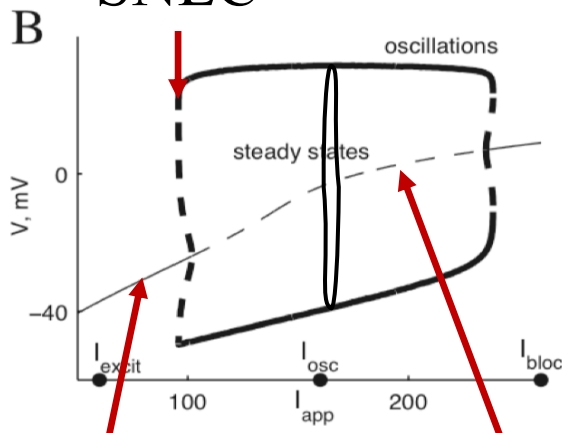
Morris-Lecar: another 2-d model

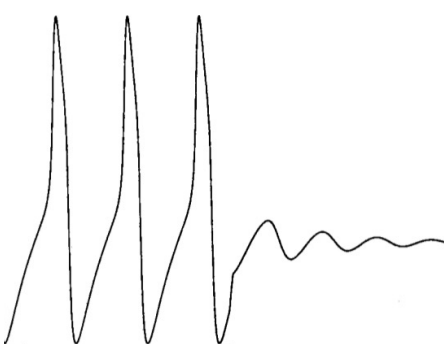
Im

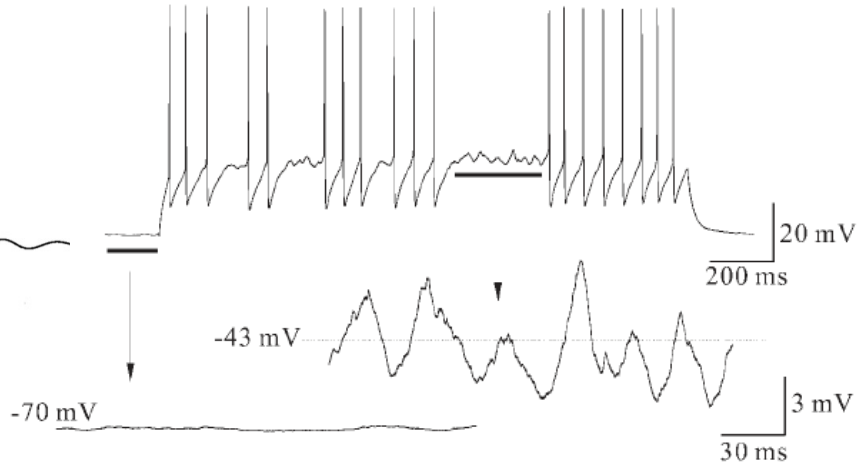
Hopf Bifurcations

Re

SNLC

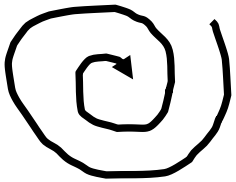


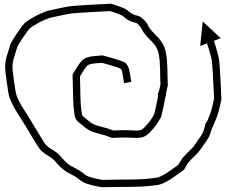




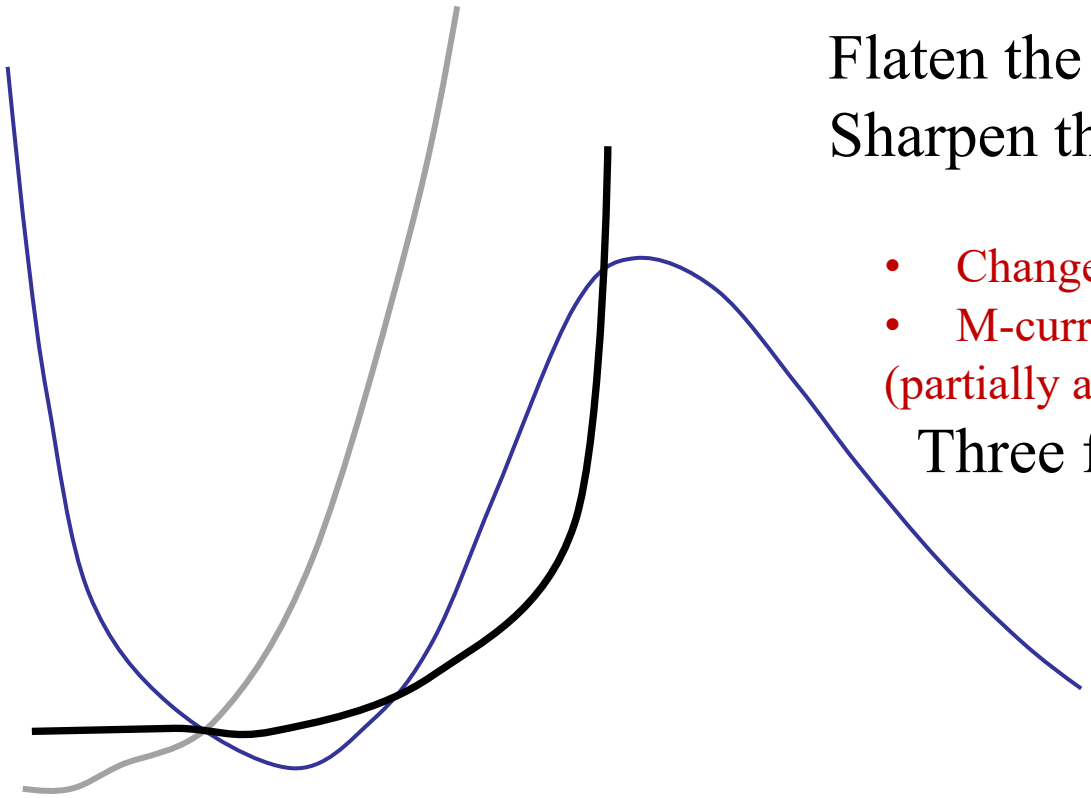
Stable Focus

Unstable Focus





Morris-Lecar

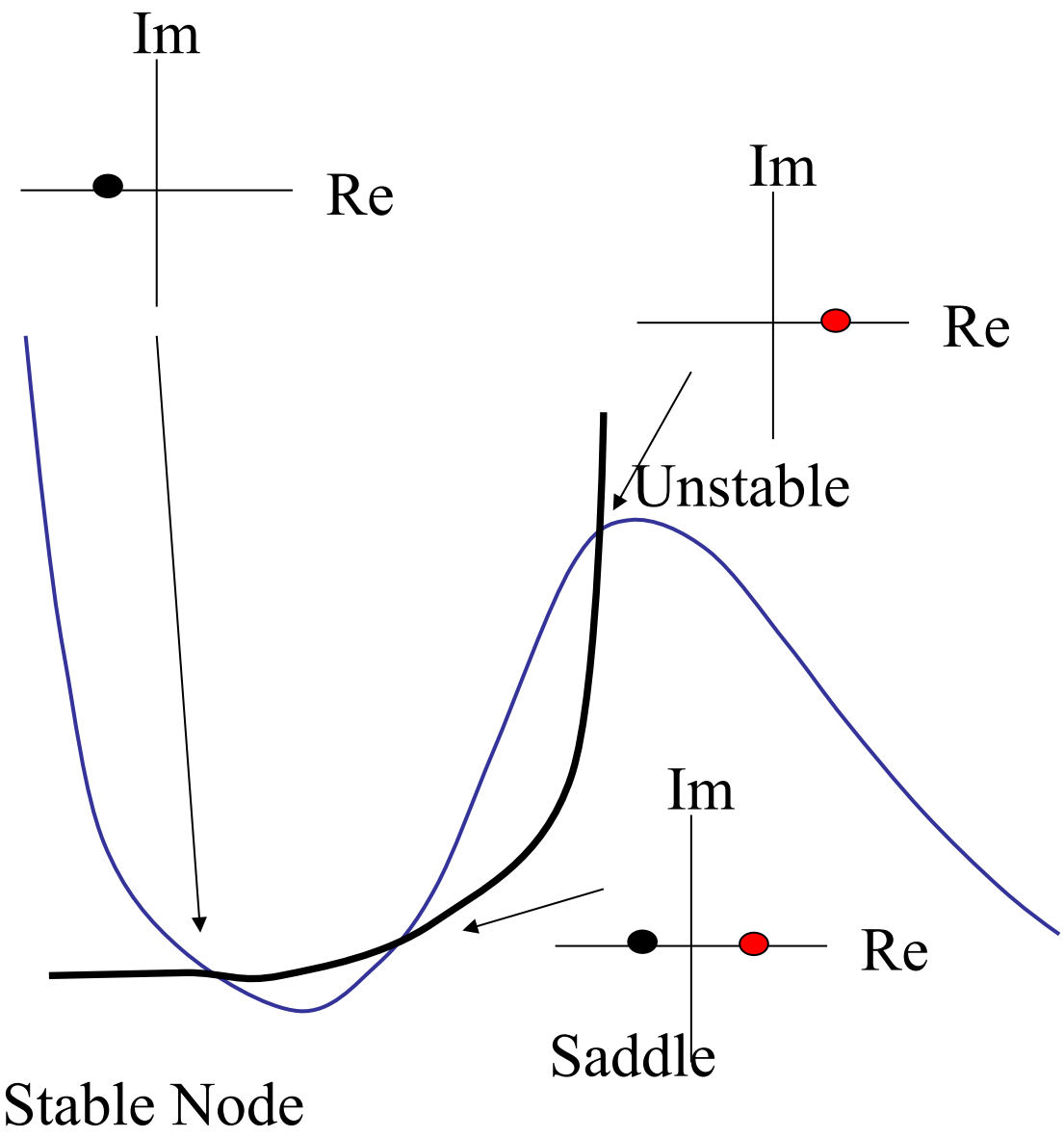


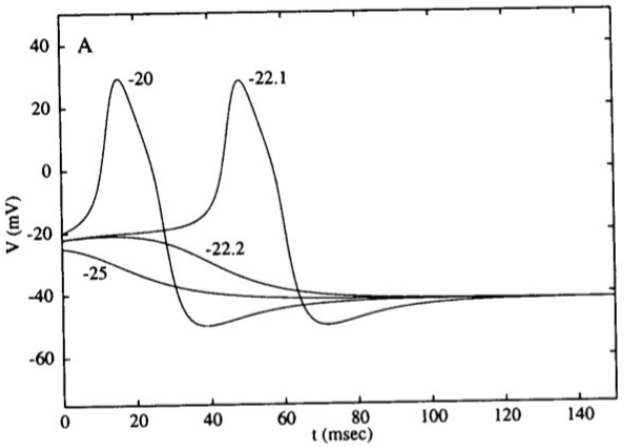
Flaten the w-activation at low V
Sharpen the w-activation at high V

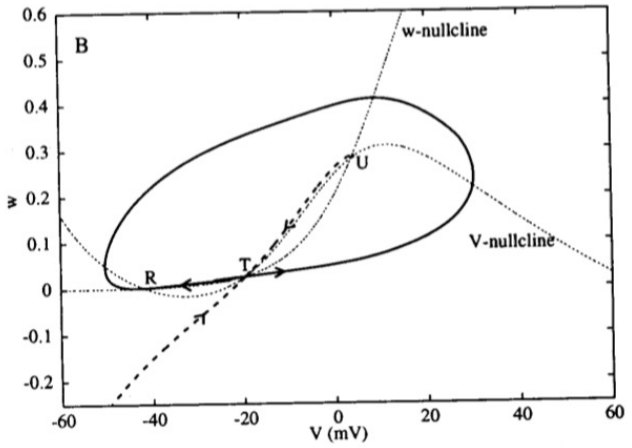
- Change in the K-voltage dependence
- M-current block

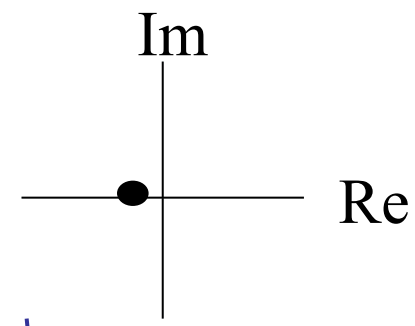
(partially activated at rest)

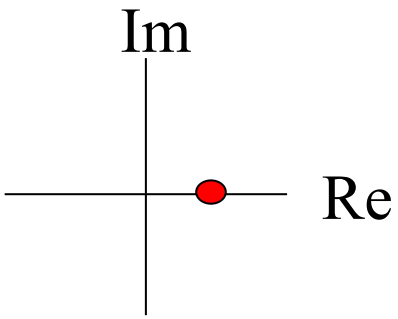
Three fixed points!

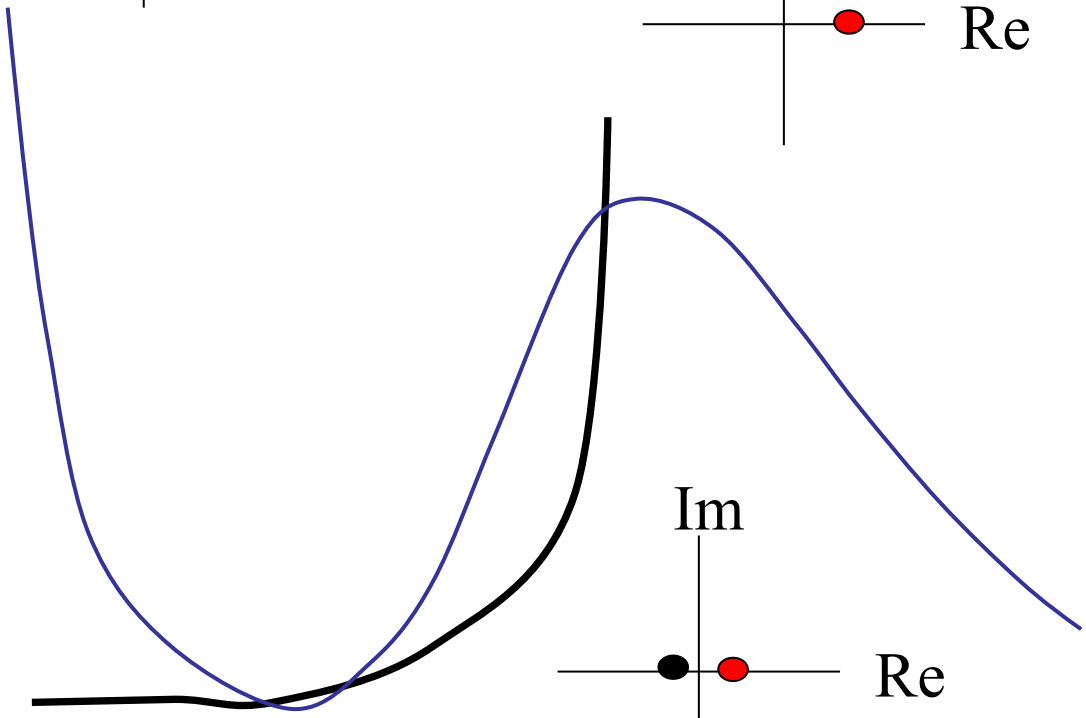

Im
Re
Im
Re
Unstable
Im
Re
Saddle
Stable Node


A
-20
-22.1
-22.2
-25
V (mV)
t (msec)


B
w-nullcline
V-nullcline
U
R
T
w
V (mV)

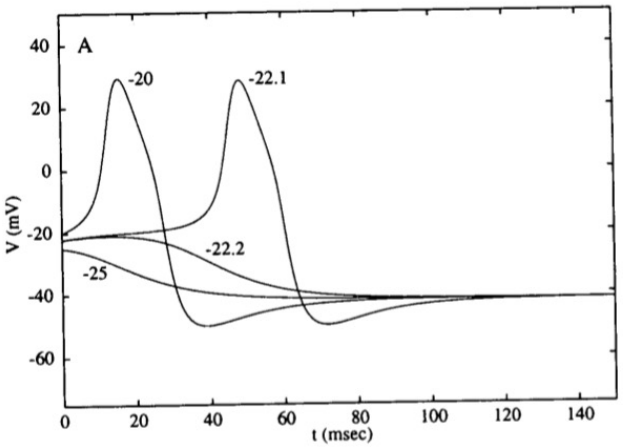


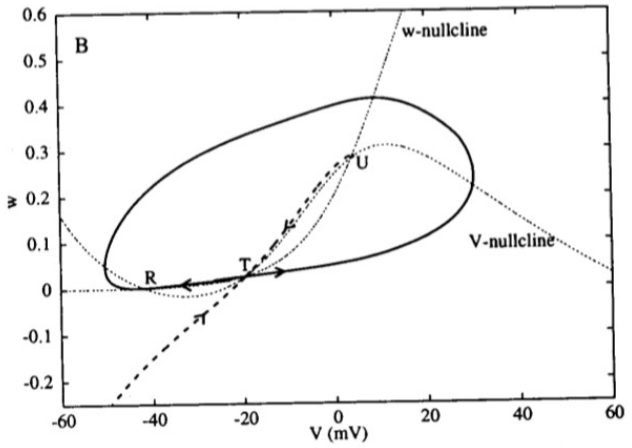




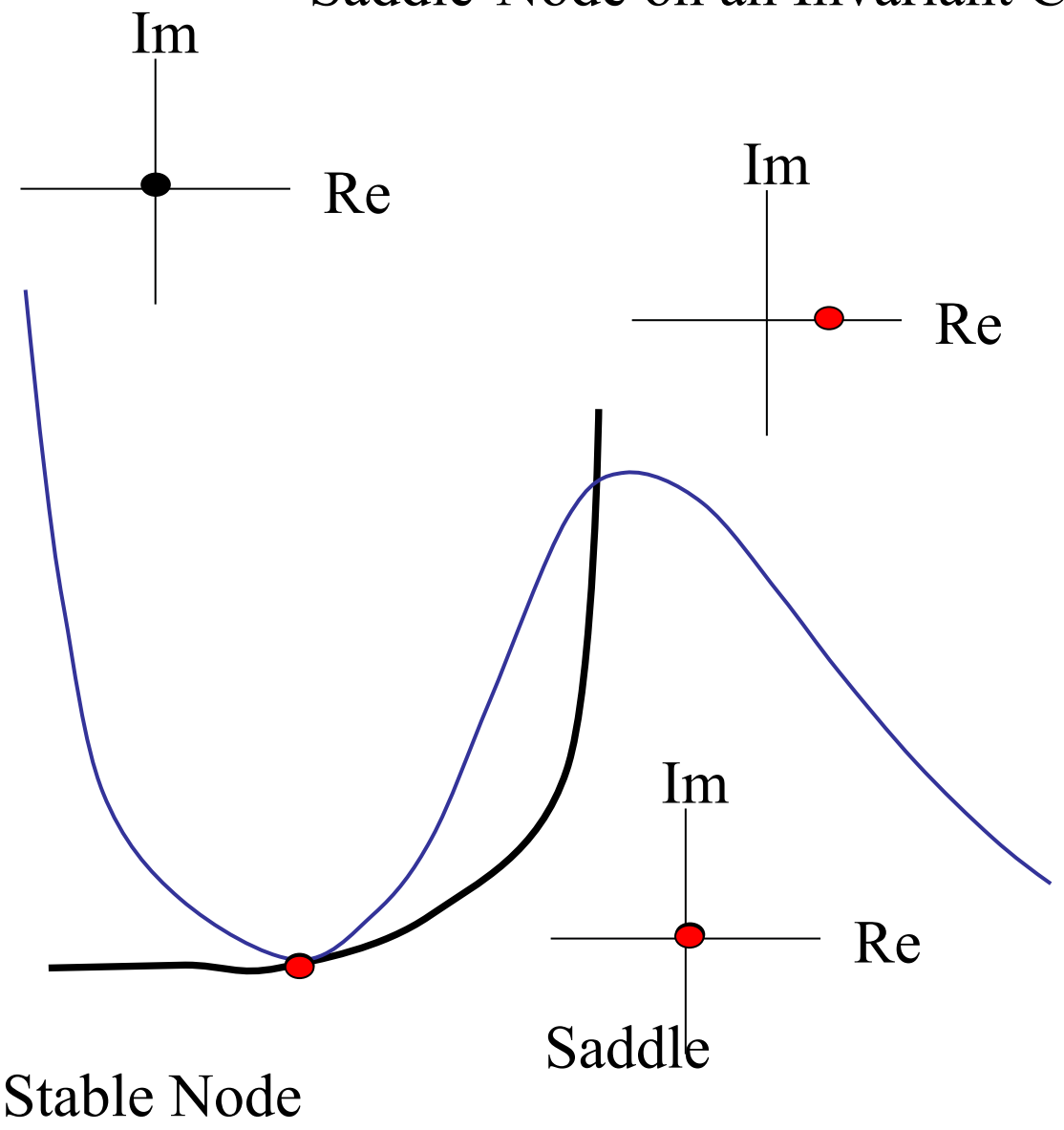
Saddle

Stable Node





Saddle-Node on an Invariant Cycle (SNIC)

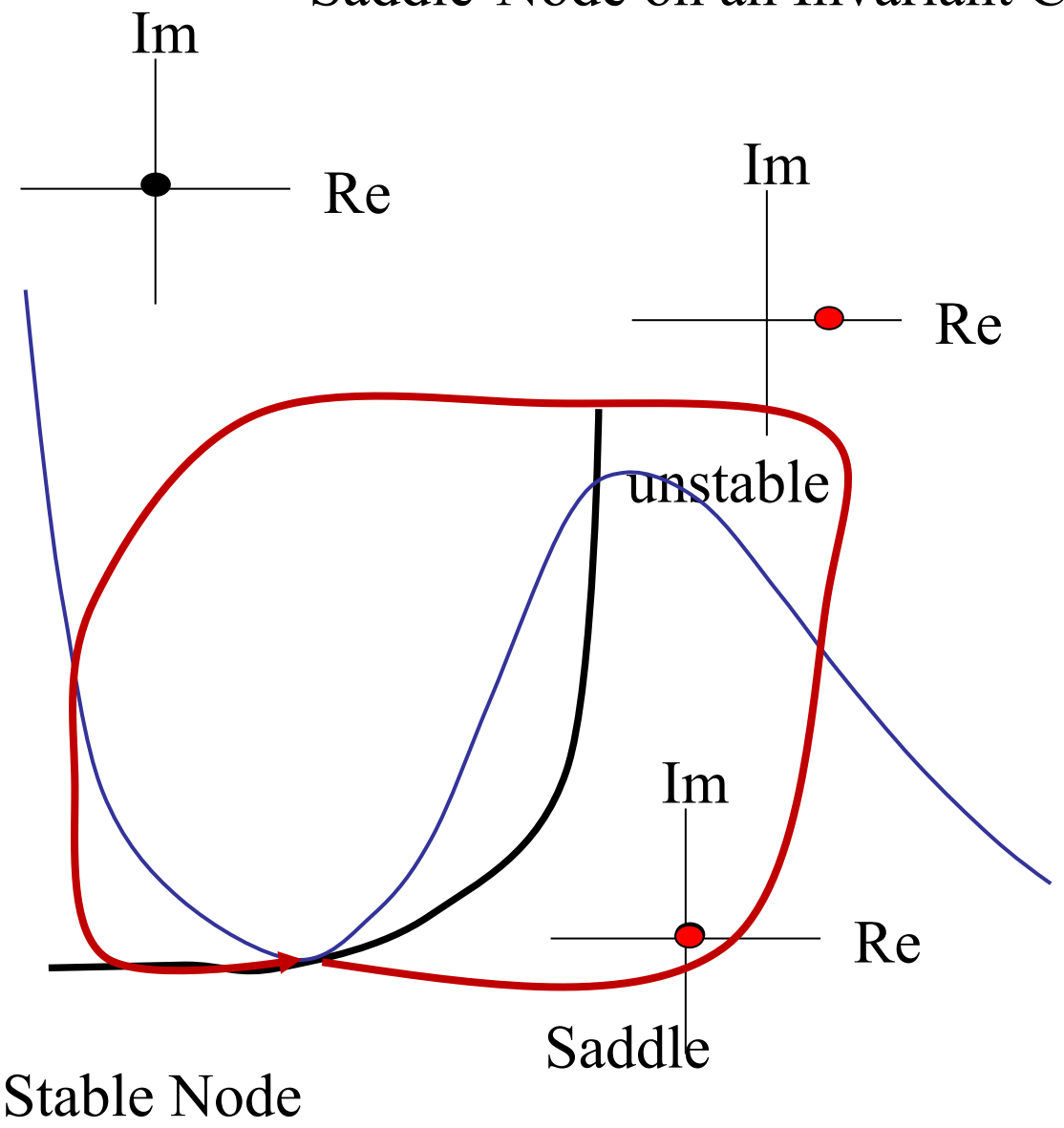


Homoclinic orbit!

Infinitely slow to leave
Infinitely slow to arrive

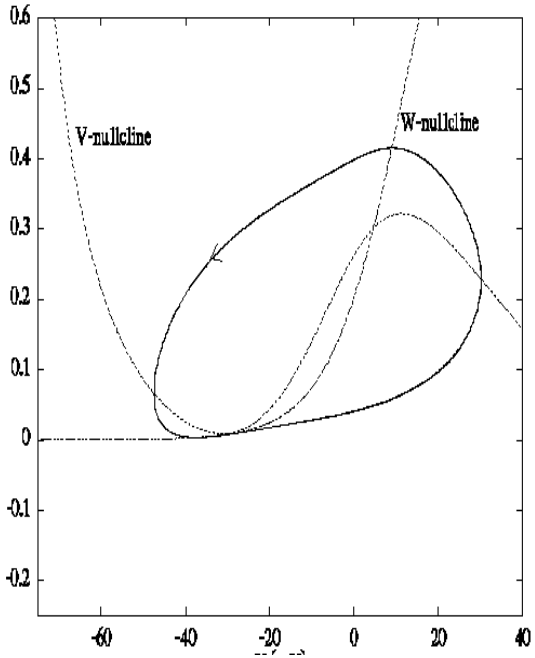
Period=∞

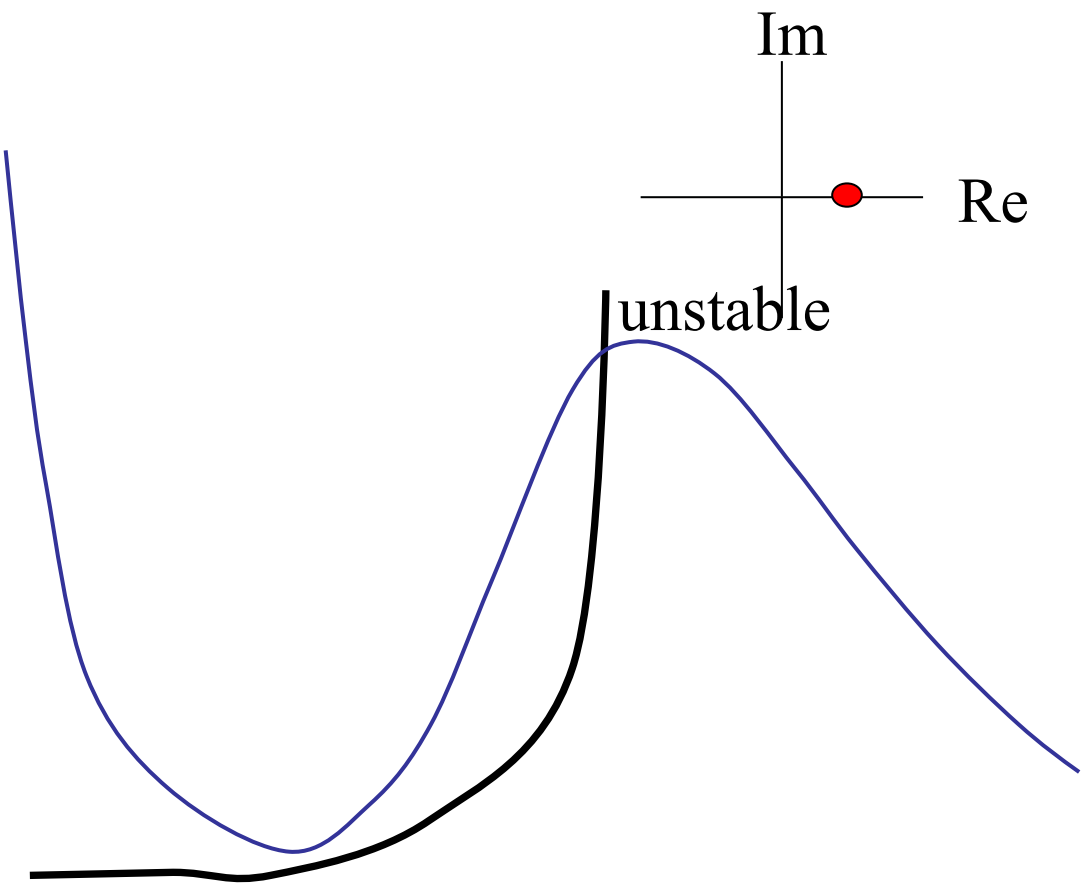
Saddle-Node on an Invariant Cycle (SNIC)



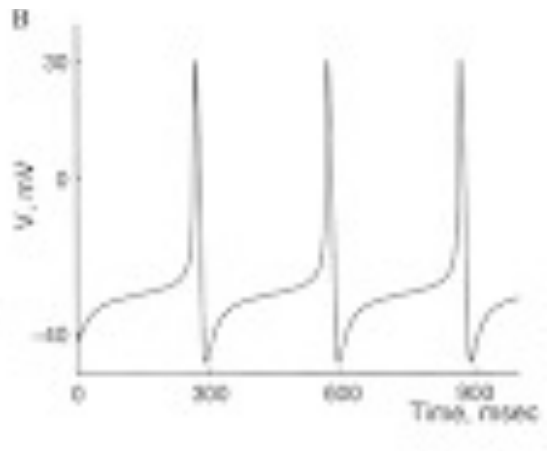
Homoclinic orbit!

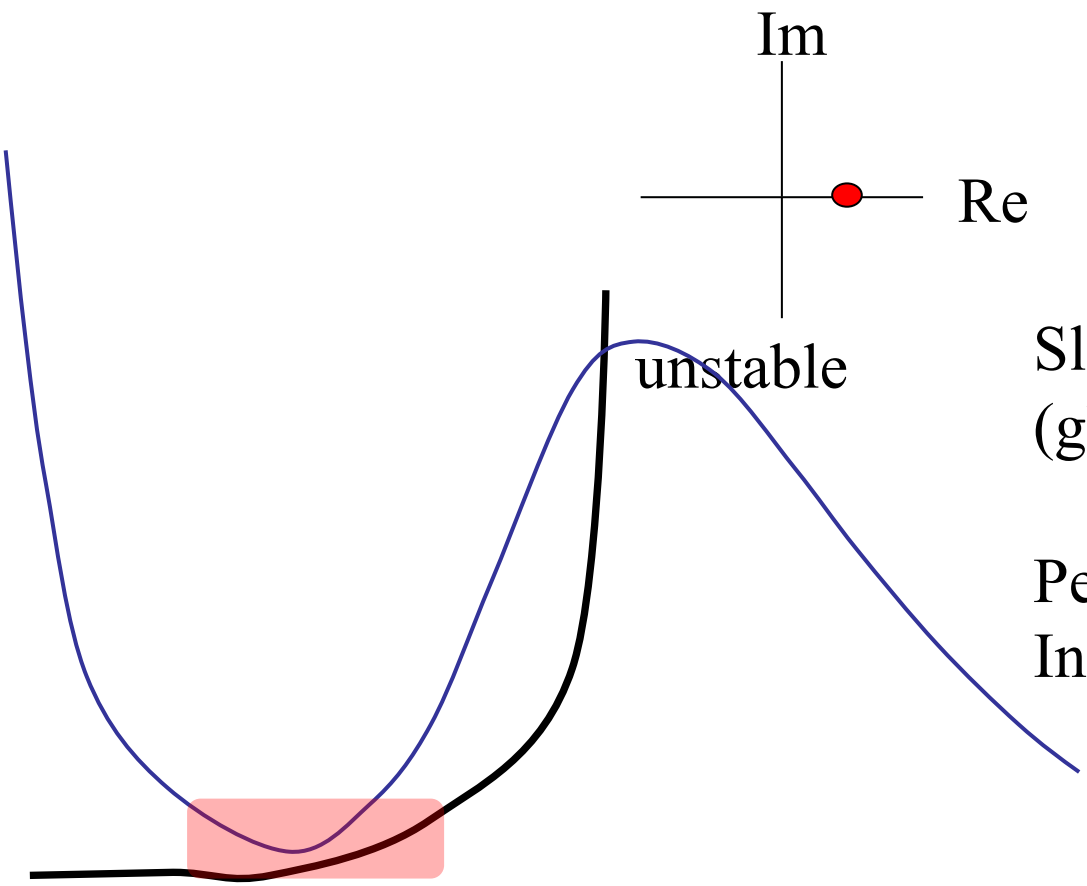
Infinitely slow to leave
Infinitely slow to arrive





Node and Saddle gone!





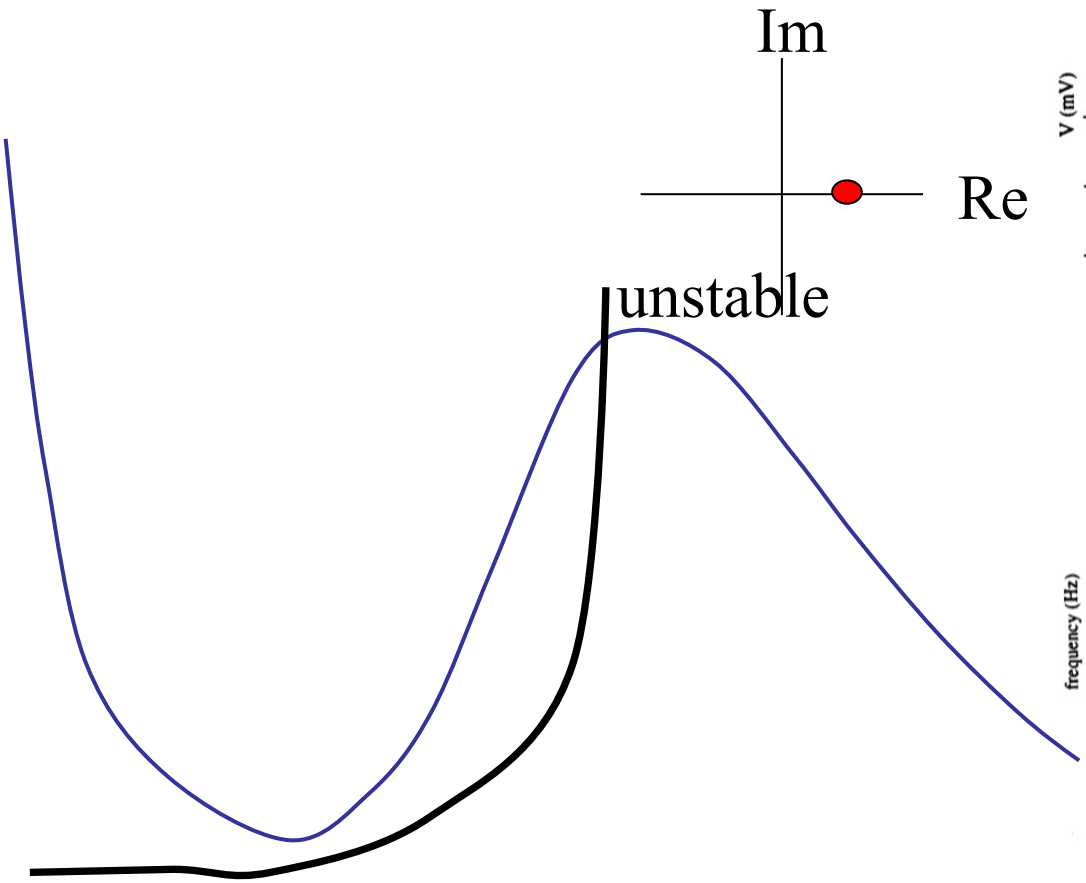
Node and Saddle gone!

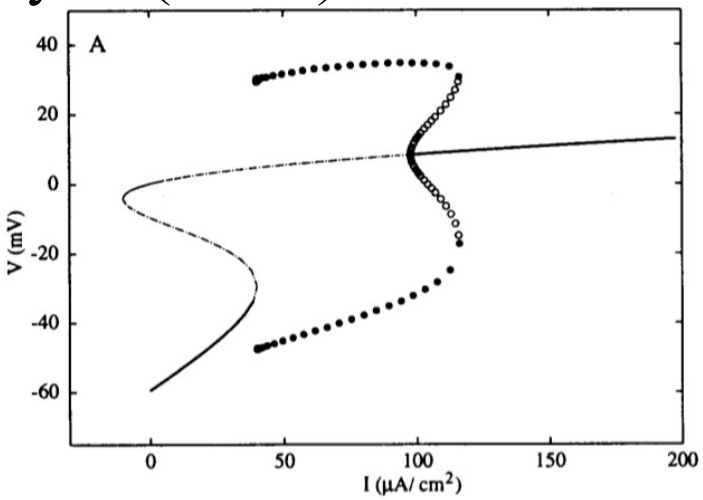
Slow channel
(ghost of the saddle-node)

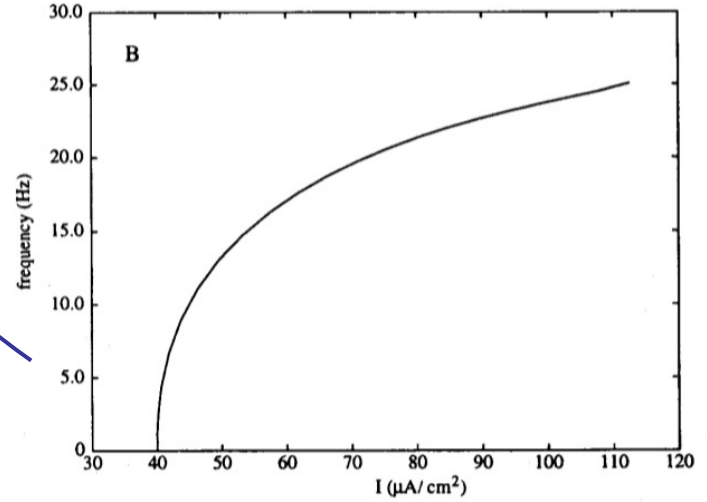
Period of Oscillation
Infinitely slow!

Stable Node

Saddle-Node on an Invarian Cycle (SNIC)

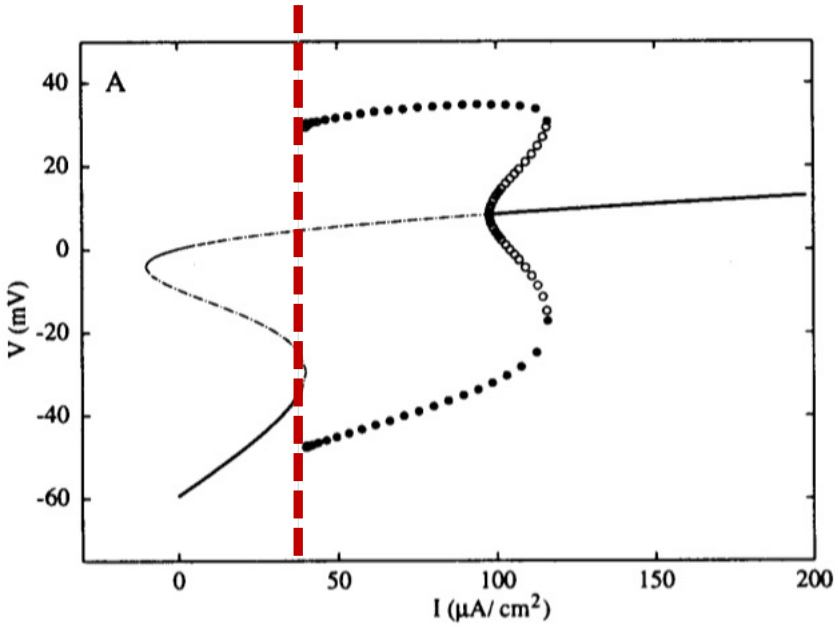






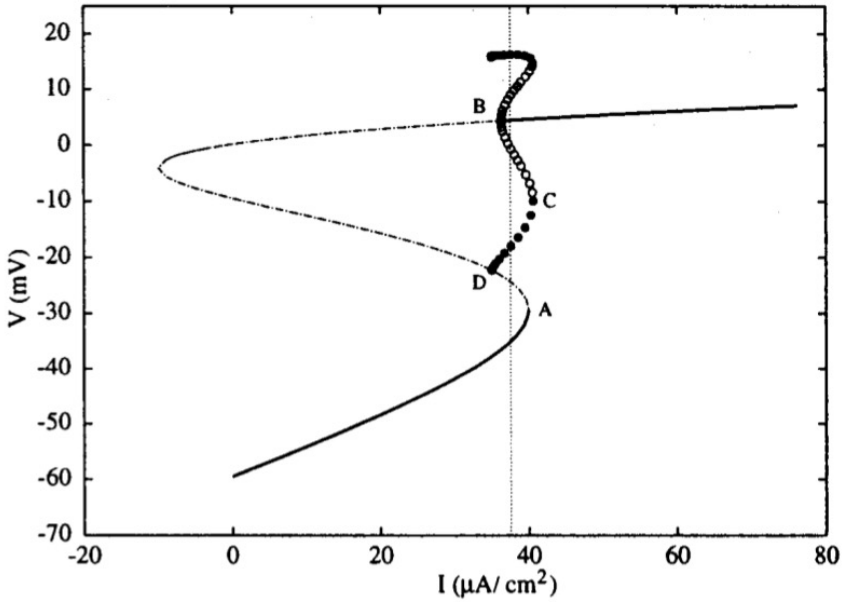
SNIC

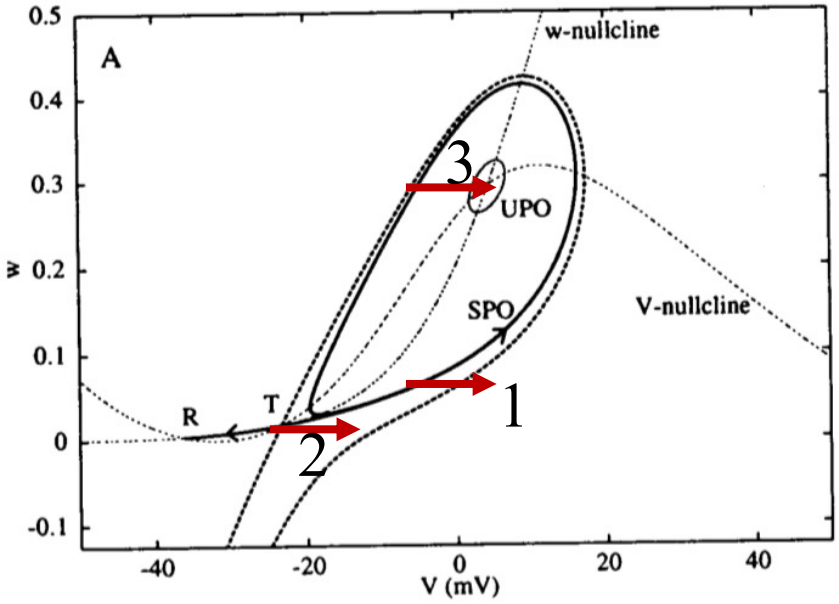
Limit Cycle dies right at SN

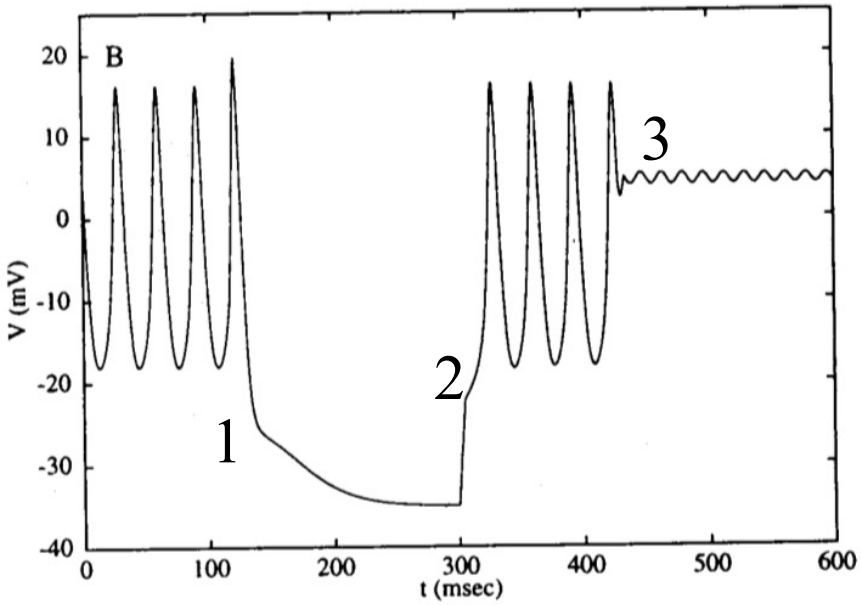


Homoclinic

Limit Cycle misses SN

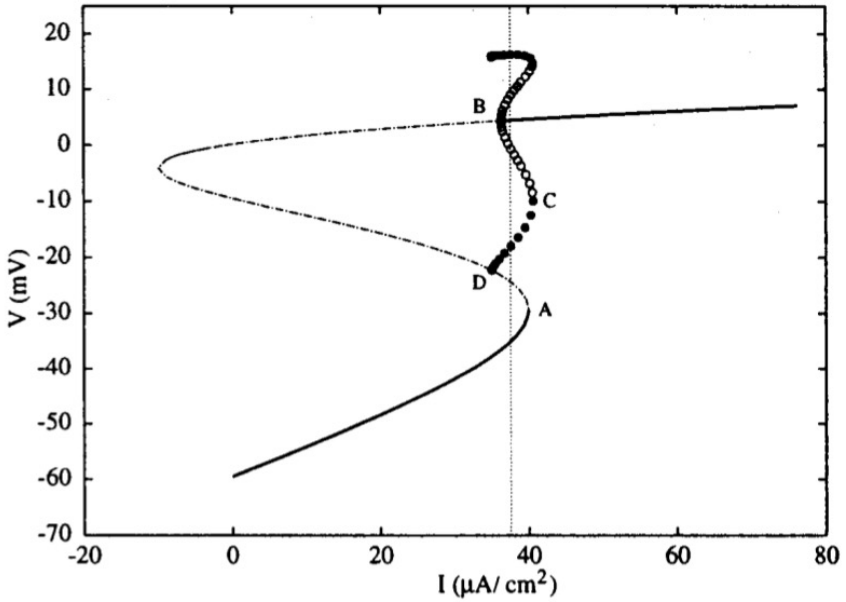






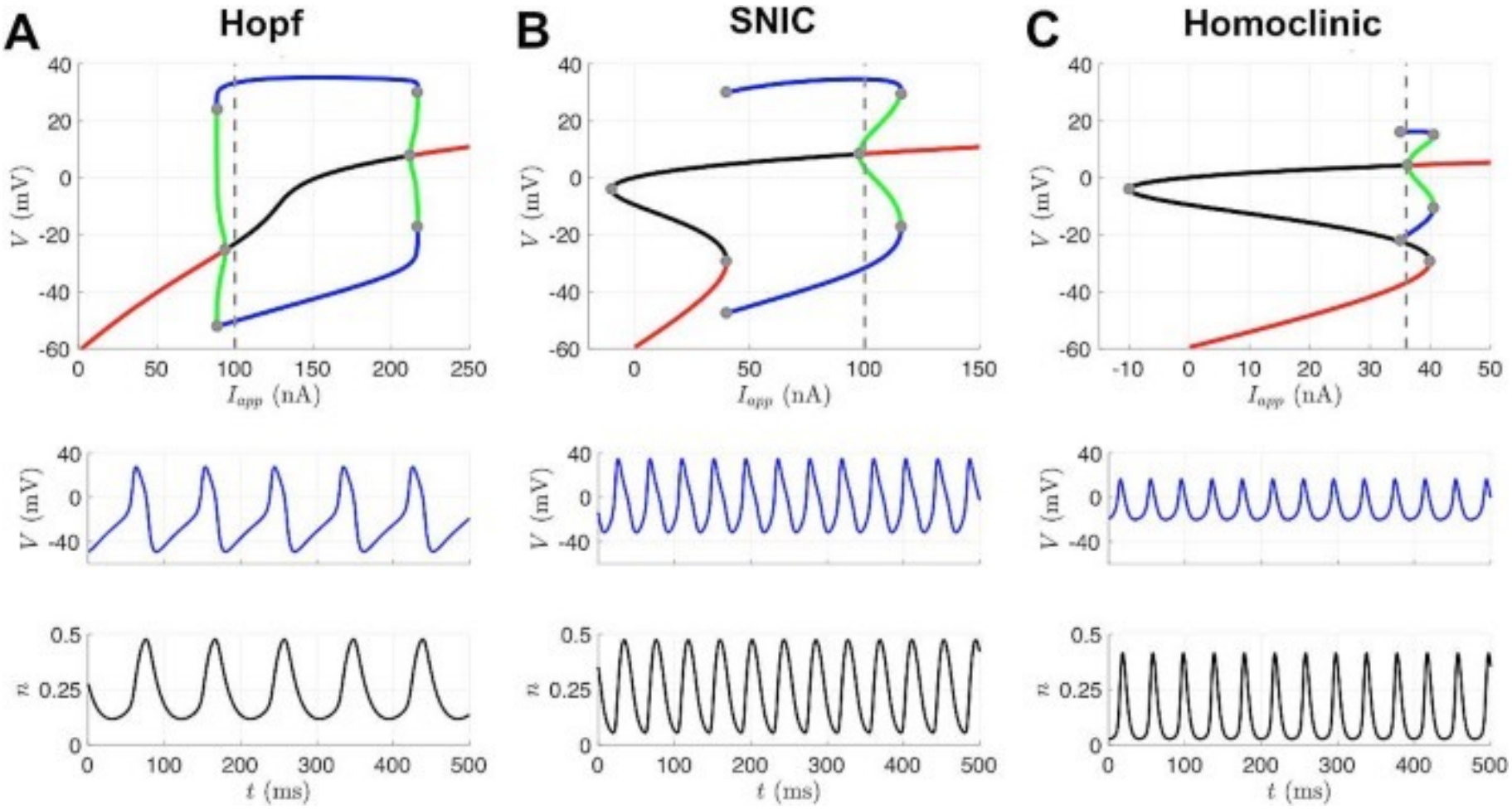
Homoclinic

Limit Cycle misses SN



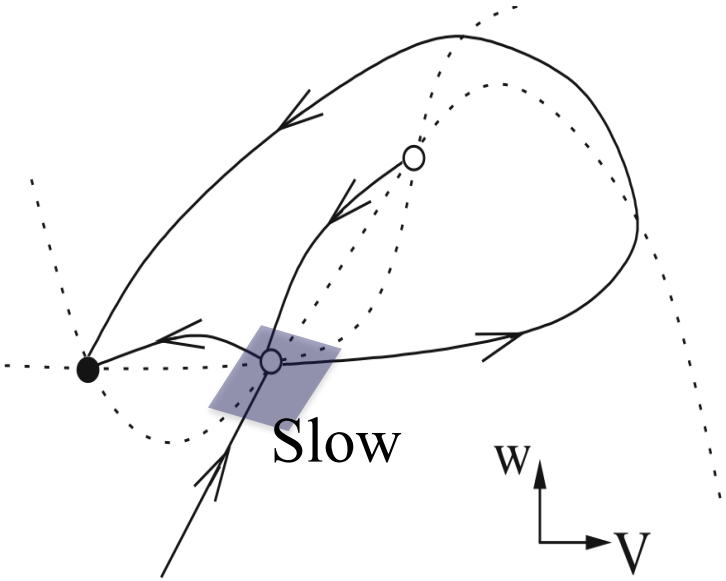
Bistability!

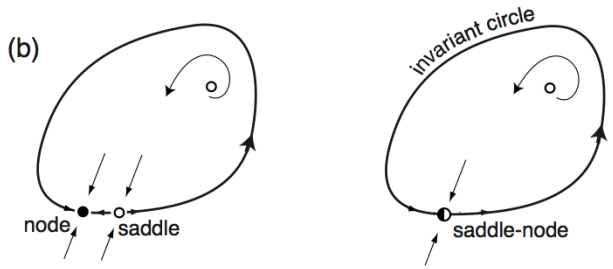
Morris-Lecar in all three cases



SNIC

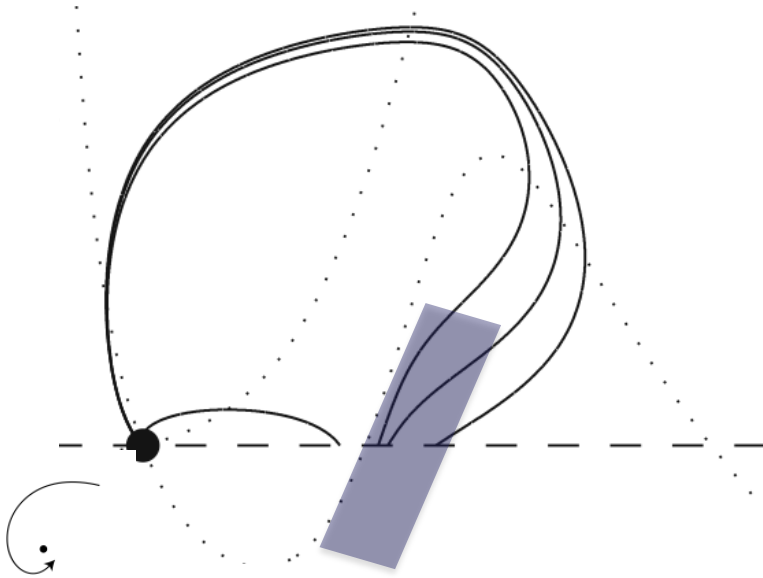
Saddle stable manifold: threshold – null speed motion

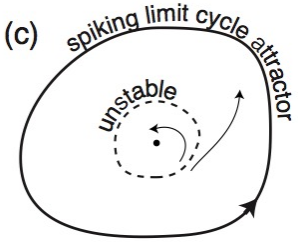




Hopf

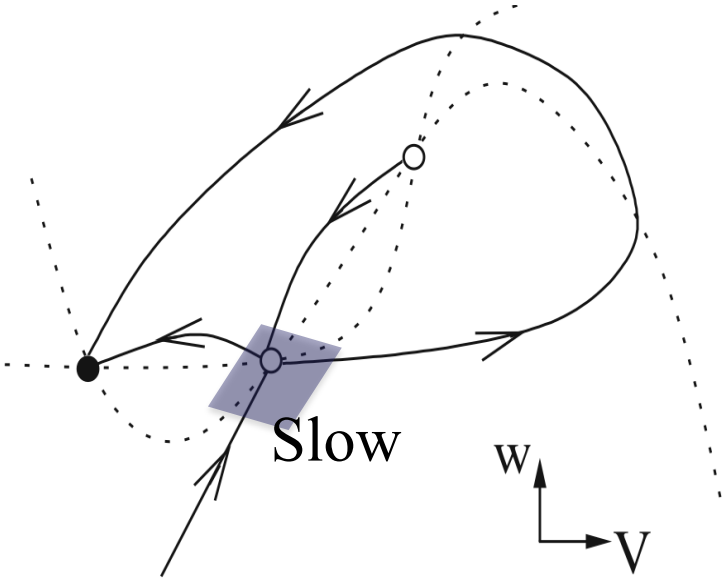
Threshold soft – finite speed motion

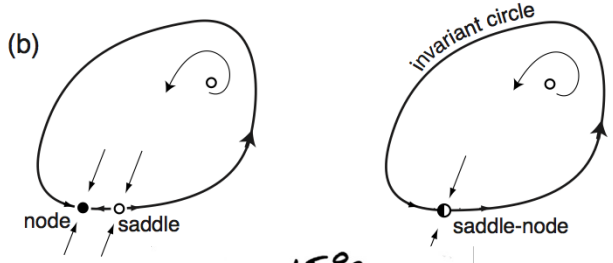


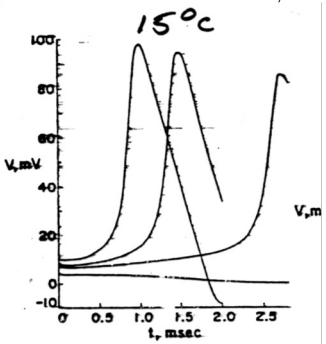


Calss1: SNIC (Nomoclinic)

Saddle stable manifold: threshold – null speed motion

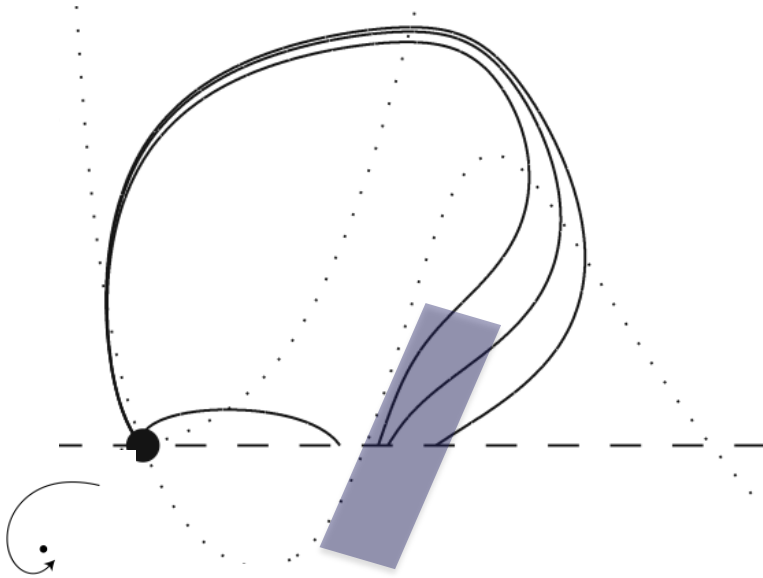


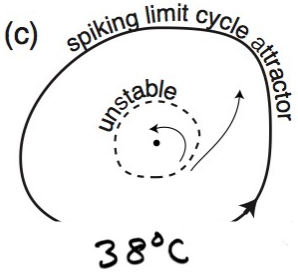


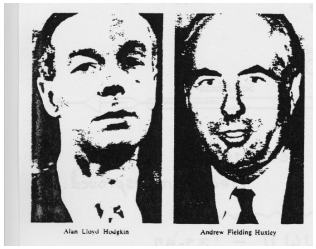


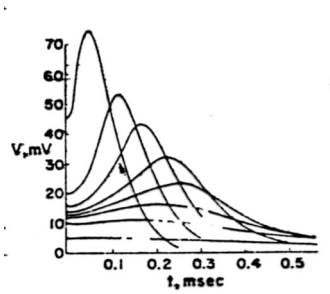
ClassII: Hopf

Threshold soft – finite speed motion



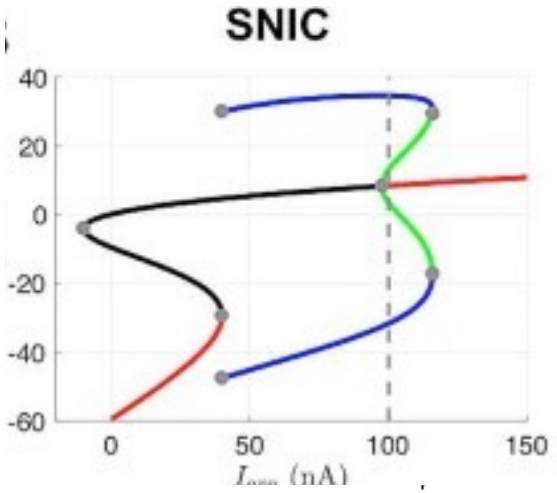


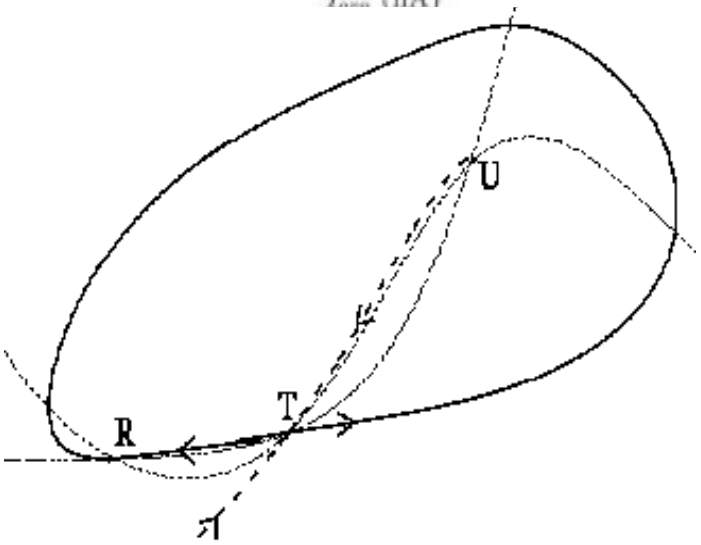




Using the "invariance" of the spike shape we can describe SNIC dynamics with a canonical normal form:

QIF or θ-neuron



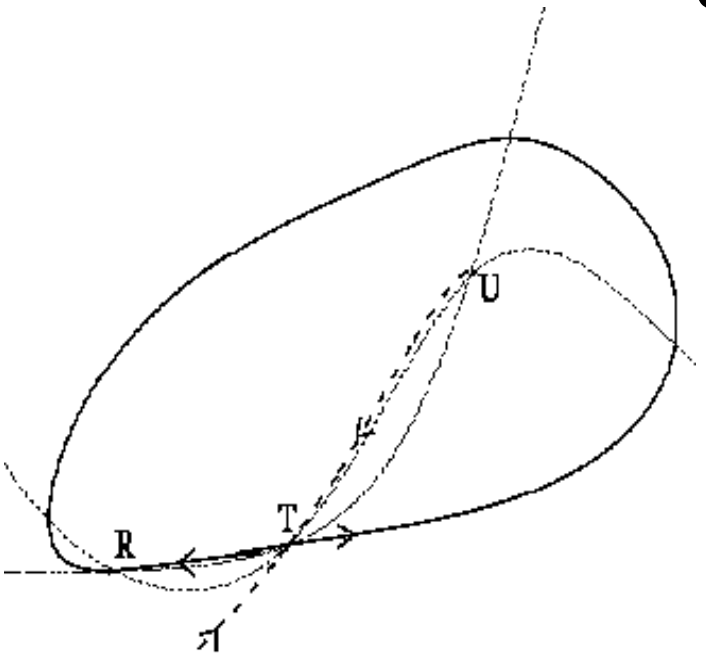


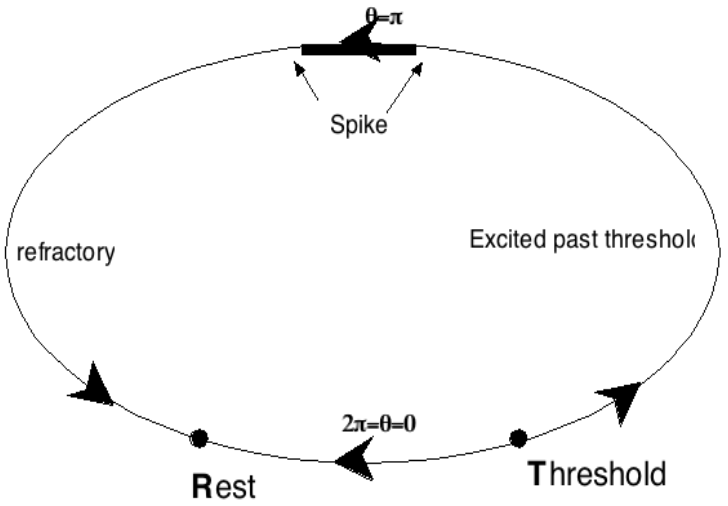
$$\frac{dx}{dt} = x^2 + \mathrm{I}$$

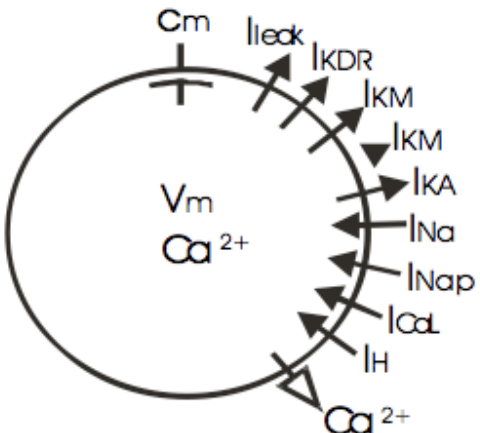
$$\tau_{\mathrm{m}} \frac{dV}{dt} = E - V + \frac{(V-E)^2}{\Delta_V} + R_{\mathrm{m}} I_{\mathrm{e}} ,$$

Using the "invariance" of the spike shape we can describe type I dynamics with a canonical model:

QIF or θ-neuron





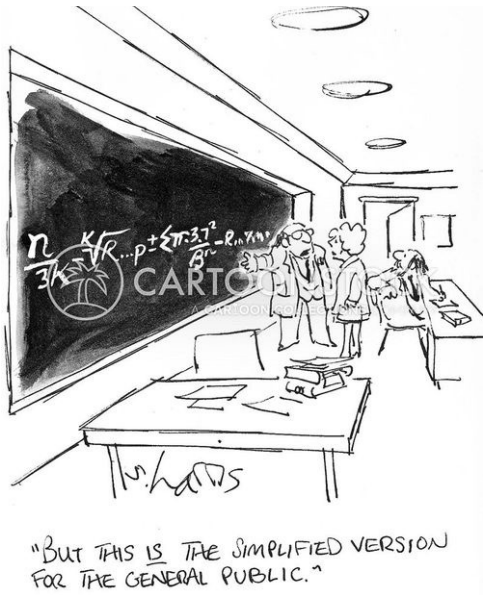


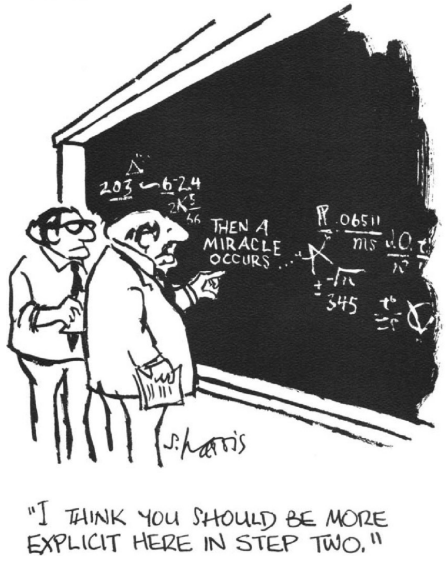
$$\dot{\theta} = (1-\cos\theta) + (1+\cos\theta)(\beta + I(t))$$

Mathematical Reduction is described in: Ermentrout, Neural Comp, 1996

Break!

- Characterizing Oscillating Neurons
- Synchrony/Rhythms in simple circuits
- GAMMA:ING/PING
- Reduced models of oscillations
- Macroscopic Phase Response for emergent oscillations





Oscillatory Rhythms 2

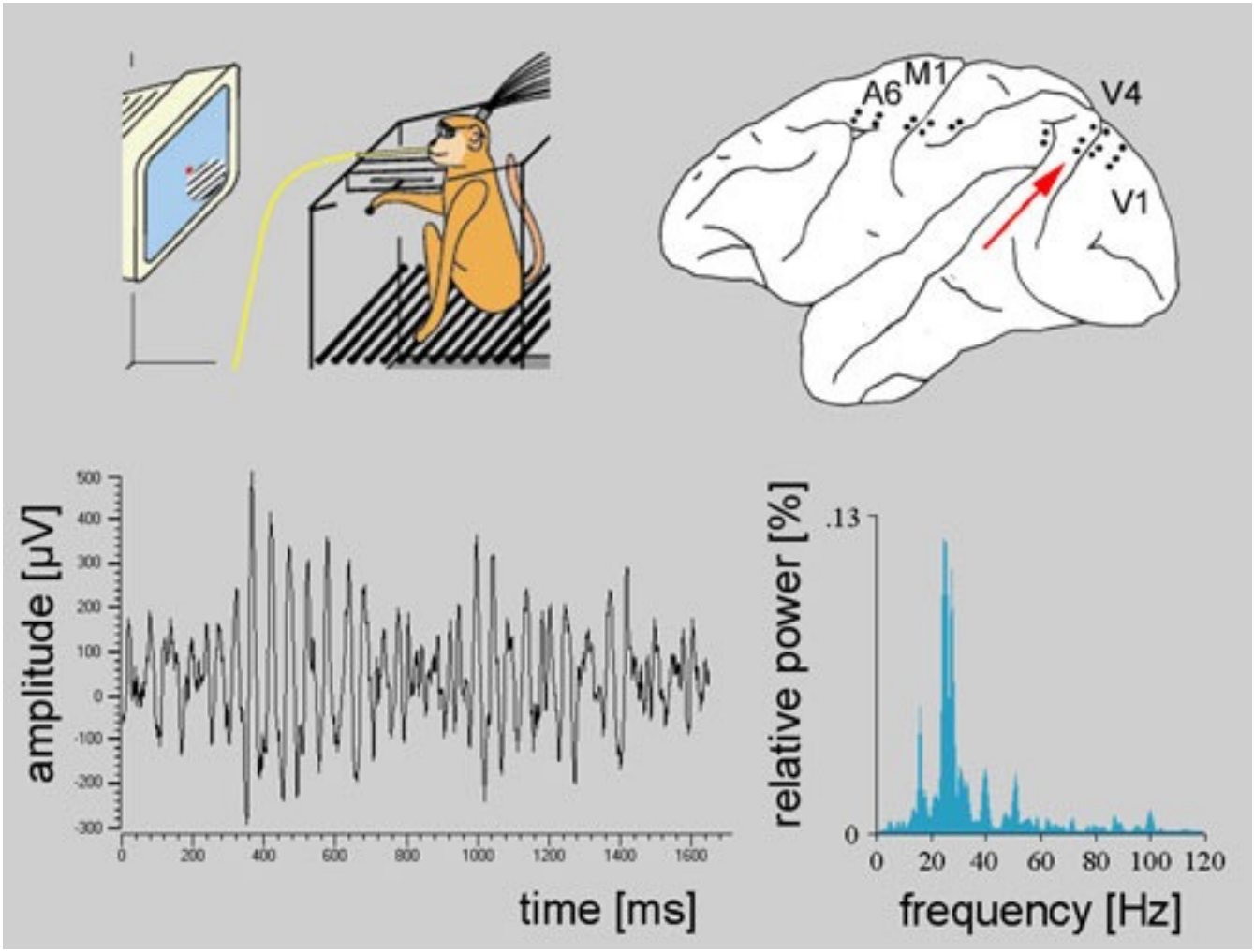
- Synchrony/Rhythms in simple circuits
- GAMMA:ING/PING
- Reduced models of oscillations
- Macroscopic Phase Response for emergent oscillations
- Phase Locking and Communication

Alphabet Soup

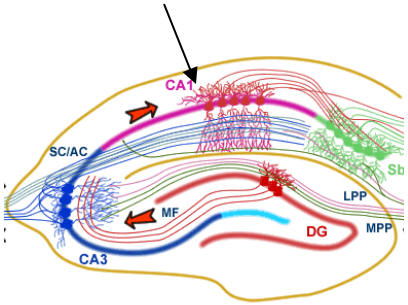


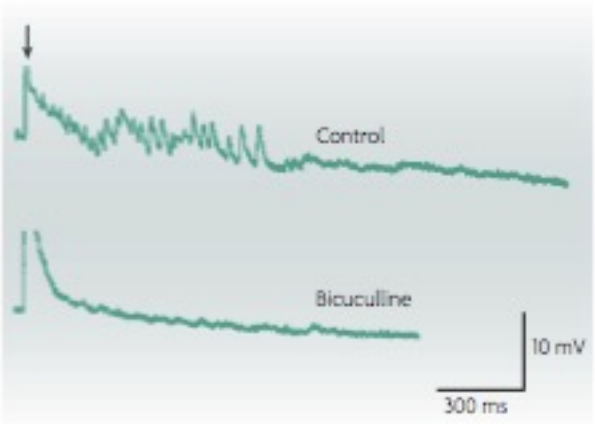
- delta (1-4 Hz)
- theta (4-12)
- alpha (9-11) Hz
- beta (12-30)
- **gamma (30-90)**
- spindling (11-15)
- ripples (100 -200)
- slow (< 1 Hz)

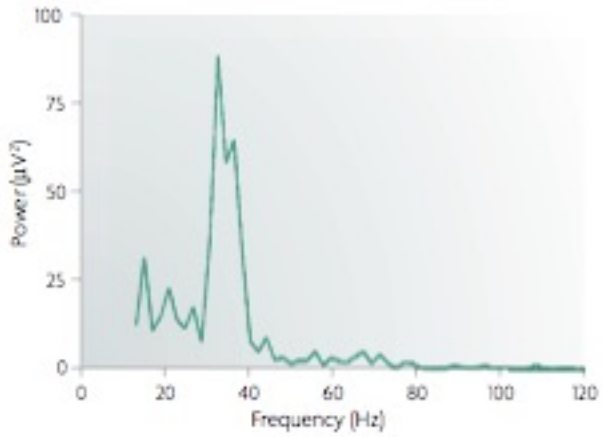
Gamma Frequency Oscillations



Sustained synchronous oscillations in slices!



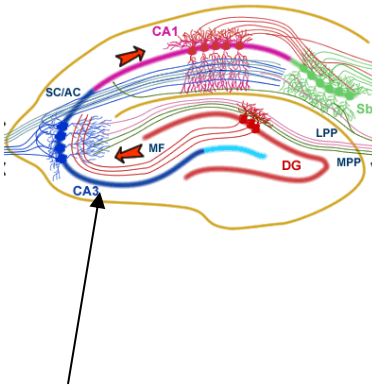


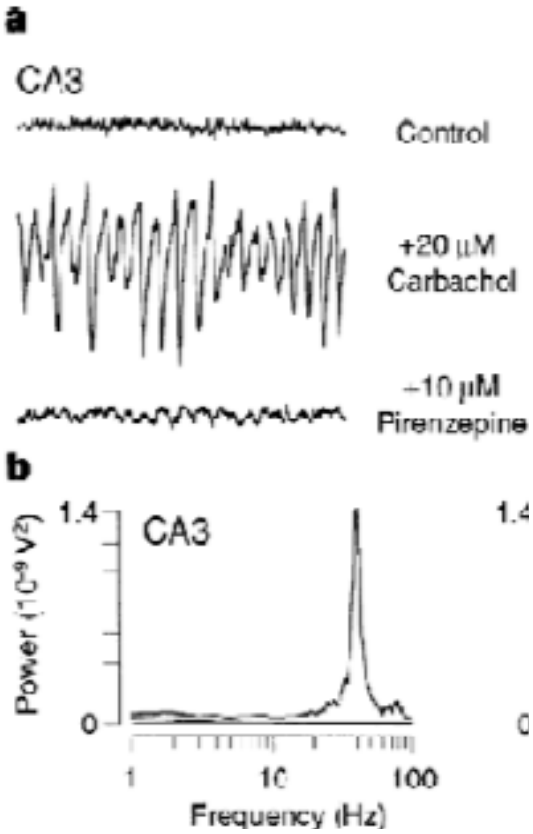


Bartos, Vida, Jonas 2006

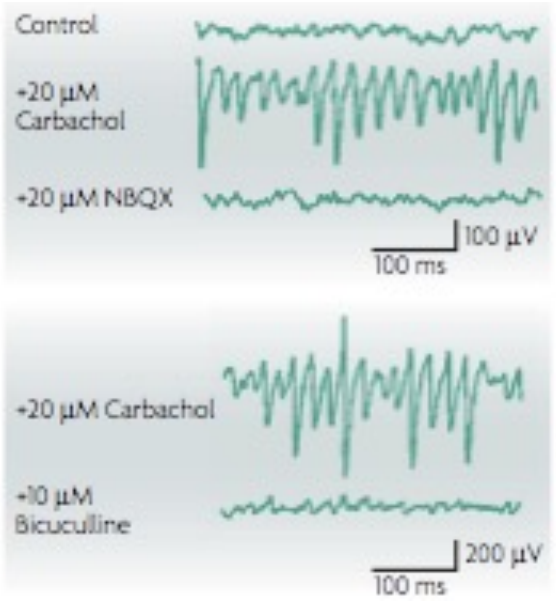
Cholinergic induction of 40 Hz oscillations in rat hippocampal slices

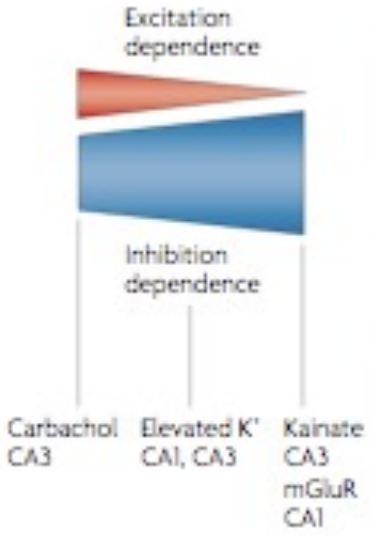
Fisahn et al, Nature 394, 186 (1998)

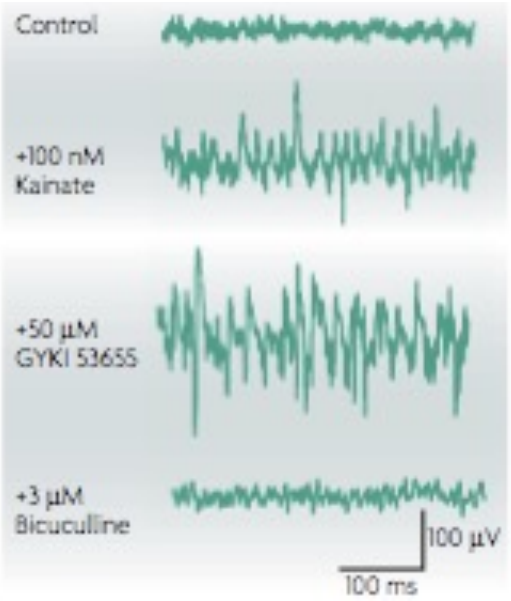


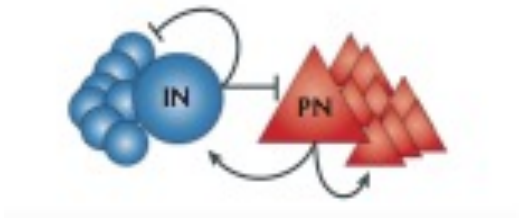


Them Gammas are not the same Gamma!











PING

ING

Gamma Oscillations: inhibition vs excitation?

How can we understand these results?

Math/Models to the rescue!

Gamma Oscillations: inhibition vs excitation?

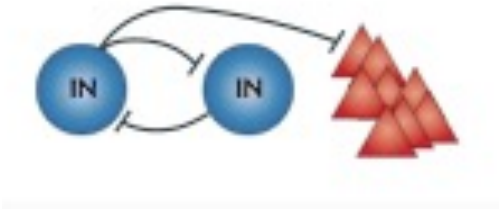
How can we understand these results?

Math/Models to the rescue!

- Consider that neurons are tonically firing even if disconnected

  Neurons = Oscillators
- Consider that synaptic coupling is relatively weak
- Develop a theory that allows to understand what are the important players:
  - Excitation
  - Inhibition
  - Intrinsic properties
  - Frequency dependence of synchrony

ING: Synchrony with inhibition



ING: Synchrony with inhibition



• Math analysis of a pair of weakly coupled oscillators shows that synchrony is stable with

- •Excitation if synapses are instantaneous
- •Inhibition if synapses have a finite time course
  - •Van Wreeswijk et al 1995
- •This depends on the intrinsic properties of the cell as defined by its

Phase Response Curve.

A mathematical Interlude: oscillators in general and weak coupling.

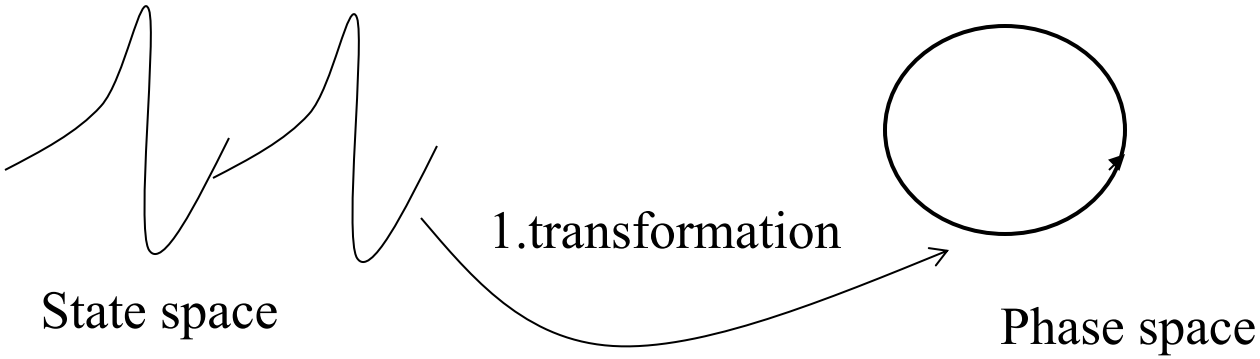
Definition: oscillator is a dynamical system that has a periodic solution

$$\dot{X} = f(X) \qquad X \in \mathbb{R}^N$$

there is a limit cycle attractor periodic orbit $u(t)$ for $X(t)$; $\boldsymbol{x}$ in R^N such that

$$u(t) = u(t+T) \quad \forall t$$

- To describe an oscillator we need
  1. How the state variable rotates in time
  2. How this rotation is changed by inputs



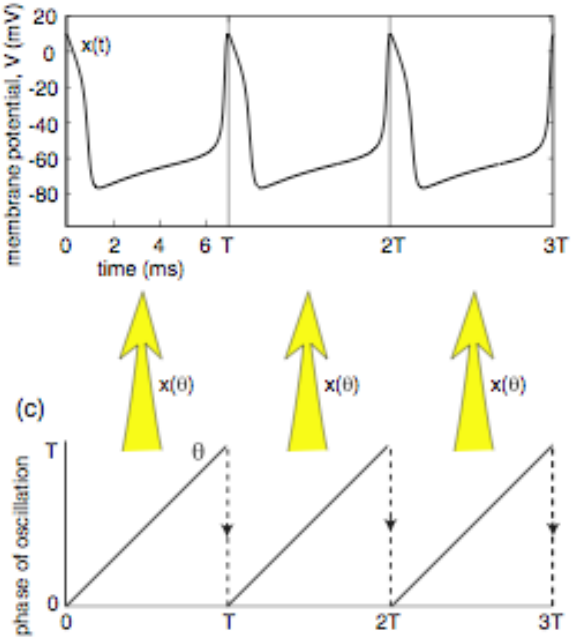
Define the phase for the oscillator

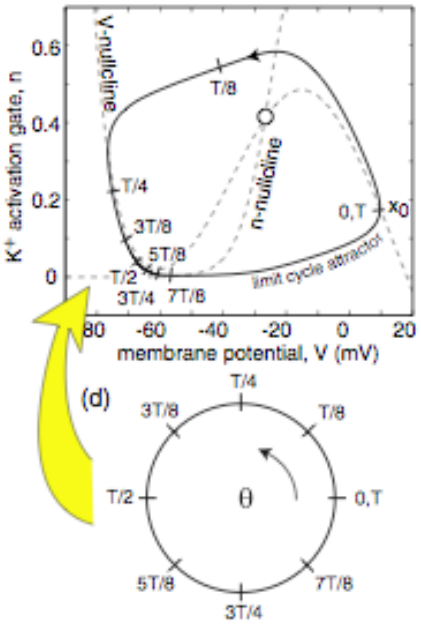
Let $x_0=x(0)$ be an arbitrary point on $\upsilon(t)$ then the state of the system can be uniquely determined by its phase θ in S^1 relative to x_0

Let us also assume that the LC $\upsilon(t)$ is exponentially stable, then there is a unique transformation between the state of the system and the phase.

$$\theta(t) = \Theta(x(t)) \qquad \dot{\theta}(t) = \Omega$$

(Or equivalently we can find a mapping from the phase back onto the state space)





Adapted from Izhikevich

How to define this mapping to make sure that the phase moves uniformly in time?

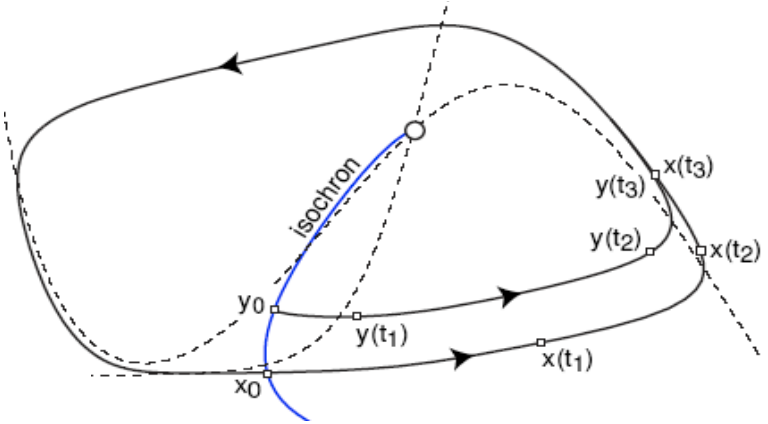
The notion of isochrons:

Note that the previously defined transformation $\theta(t) = \Theta(x(t))$

Is valid not only only on the limit cycle itself, but also in a neighborhood of it.

•Since the limit cycle solution is stable, if we start close to it (on some neighborhood *U*)

•Then a solution *y(t)* started at some *x(0)* in *U* will wind onto the limit cycle, joining as periodic solution u(t). So that eventually $y(t) \to u(t)$



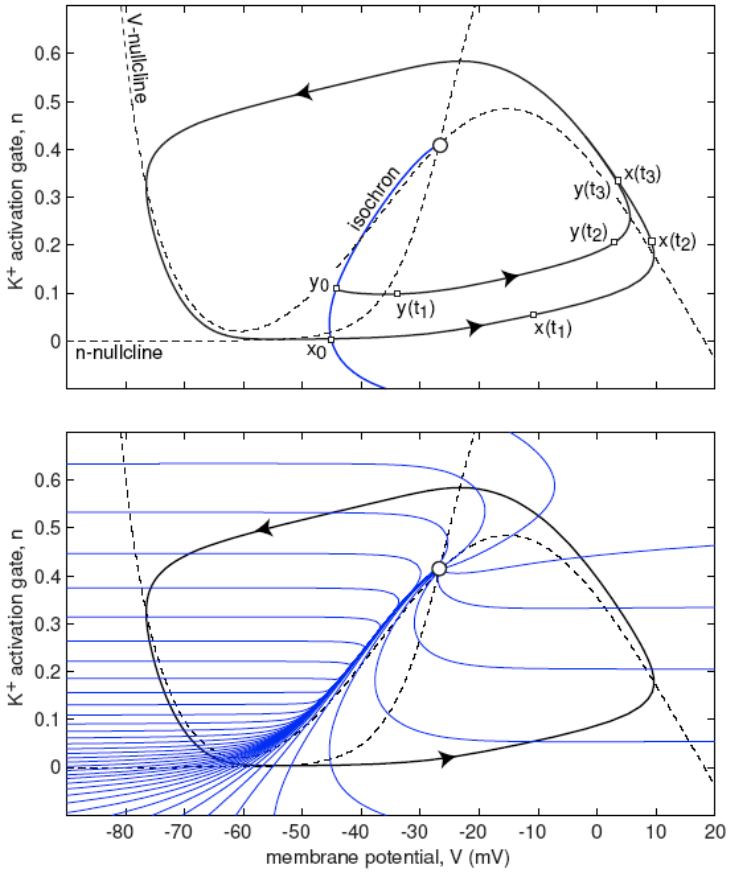
•We can define the phase of *y(t)* as the phase of its proxy the limit cycle: $\theta(y(t)) = \theta(u(t))$

•We can now define all the initial conditions *y(0)* s.t. *y(t)* started at *y(0)* that go to the same phase, or join the same proxy *u(t)*. This set now forms the stable manifold of a given phase $\theta(x(t))$ on the limit cycle. This manifold is called the **isochron**.

•Note that the function θ(x(t)) is constant along the isochrons. If we can compute the isochrons for the oscillator, we will know explicitly how to convert the state variables into the phase!

Isochrons for ML:

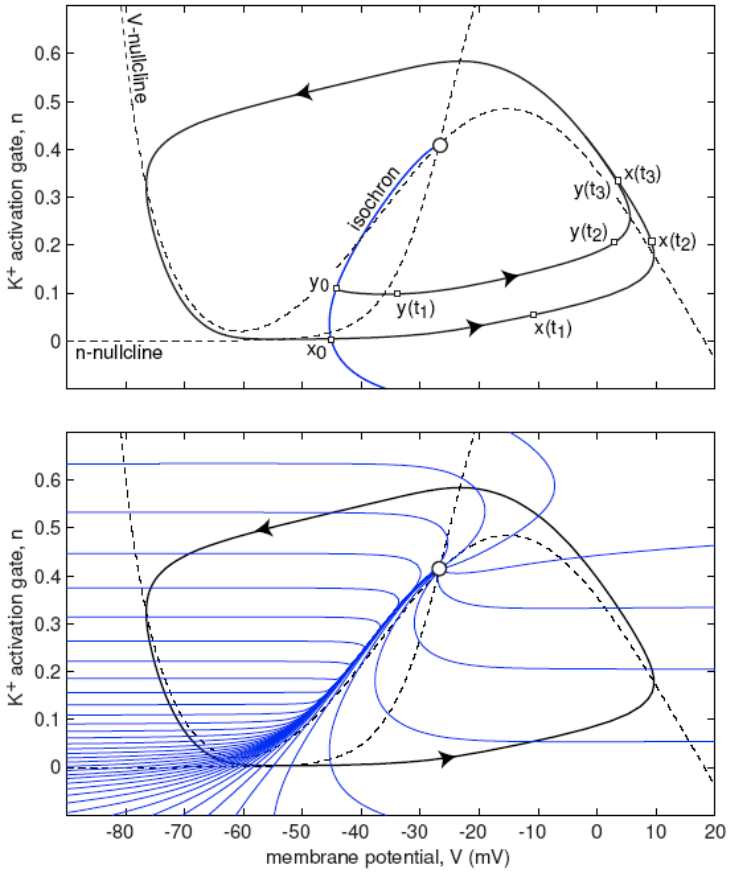
Type1: SNIC



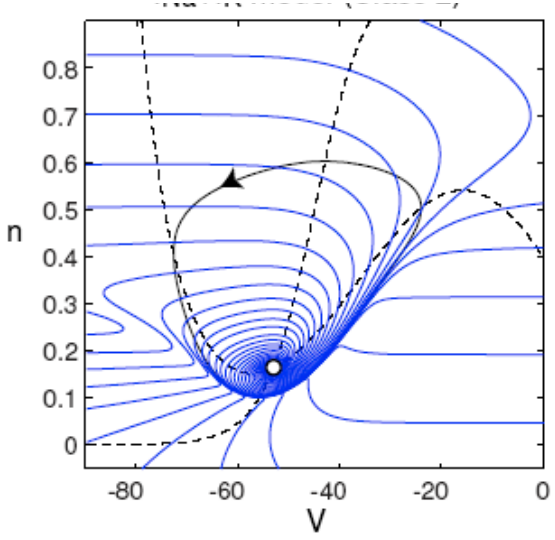
Reproduced from Izhikevich 2006

Isochrons for ML:

Class1: SNIC



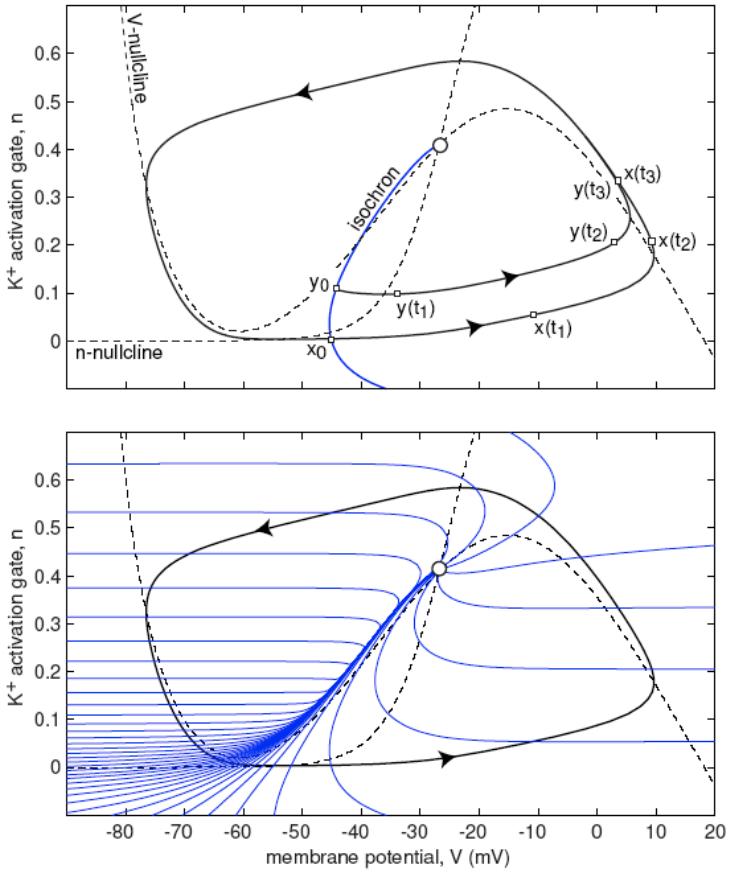
Class2: Hopf



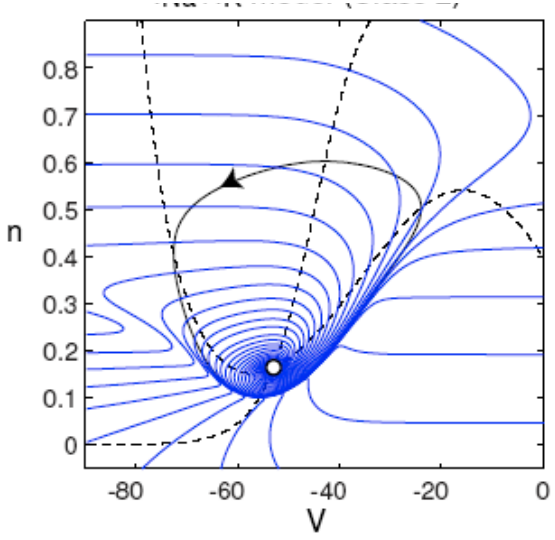
Reproduced from Izhikevich 2006

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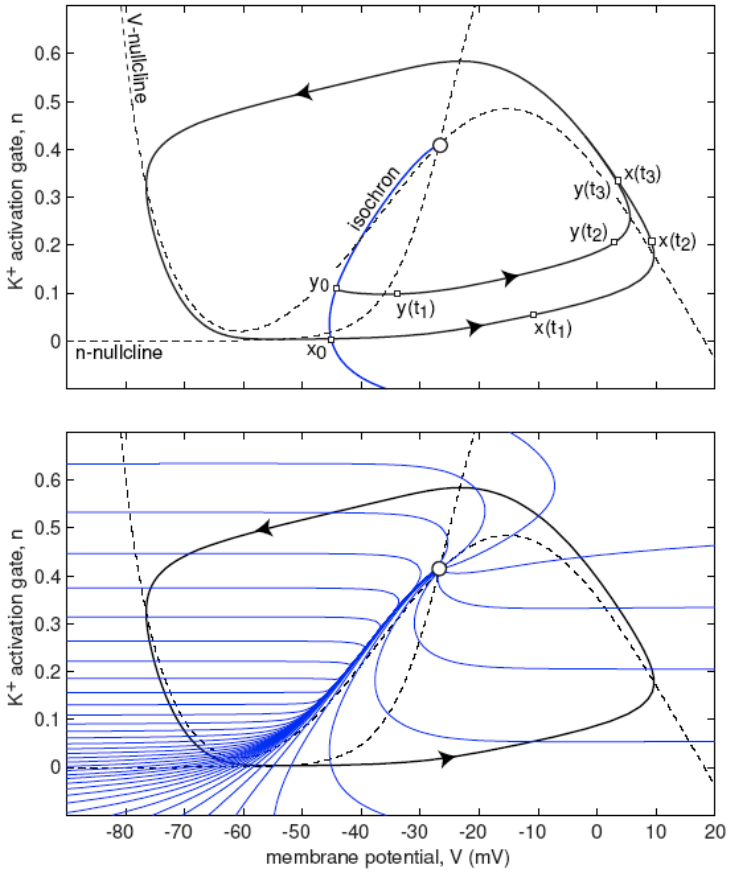
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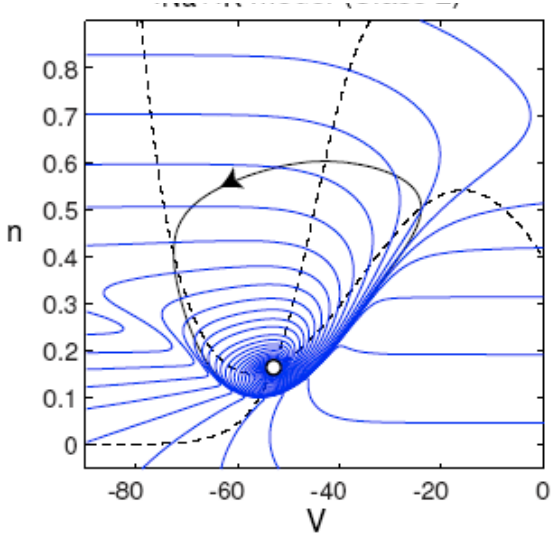
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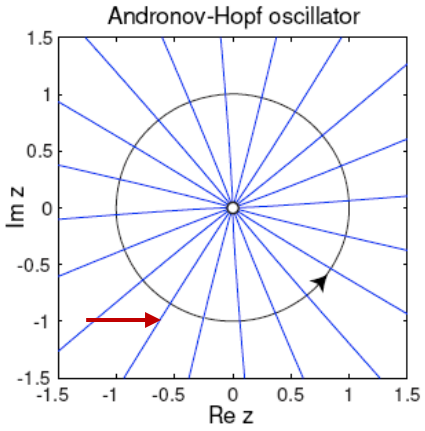
Isochrons for ML:

Class1: SNIC



Class2: Hopf



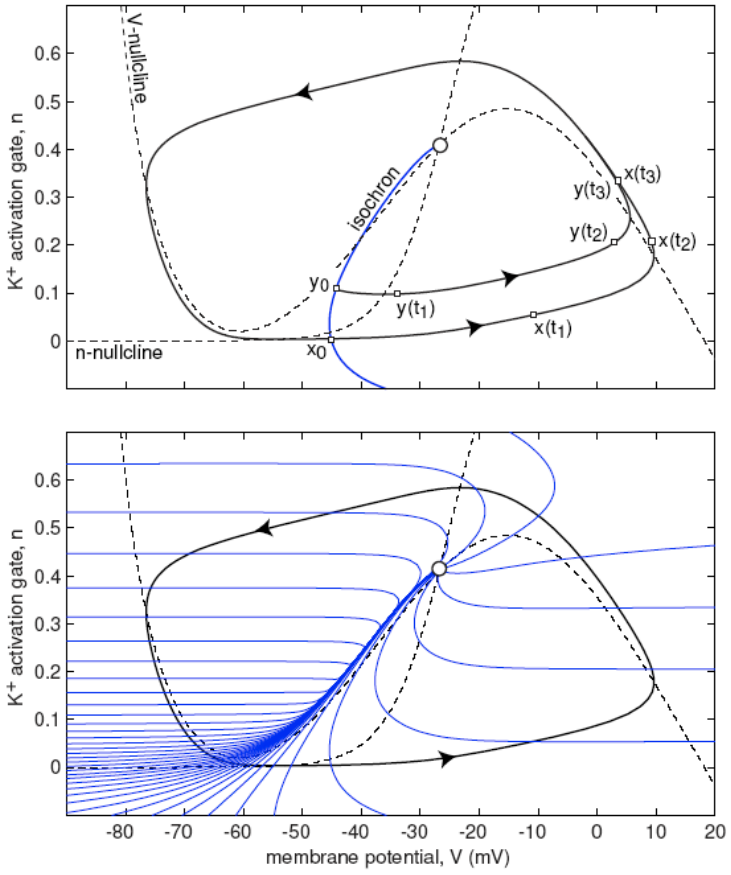


Phase advance

Reproduced from Izhikevich 2006

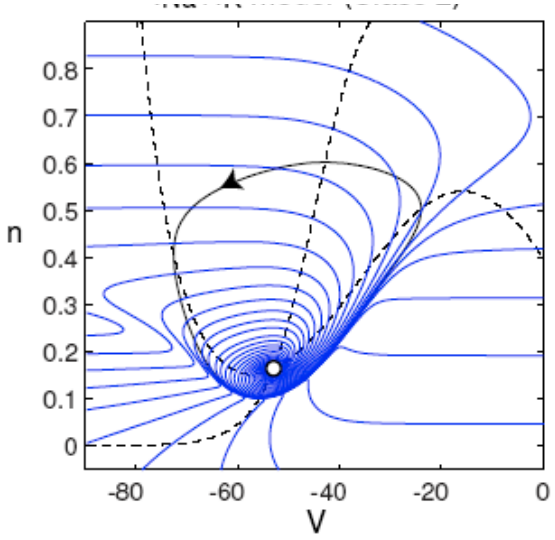
Isochrons for ML:

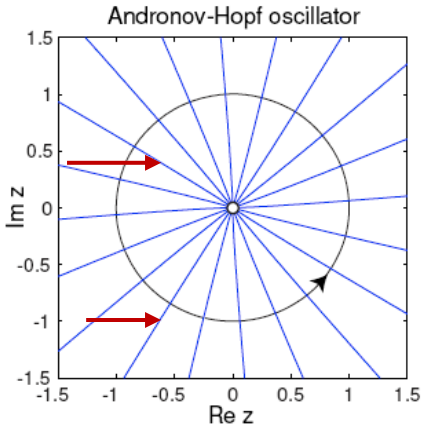
Class1: SNIC



Only Phase Advance

Class2: Hopf





Phase Delay!

Phase advance

Reproduced from Izhikevich 2006

Now we can describe the oscillator as a phase model.

We saw that the mapping defined by the isochrons can transform the oscillator into a phase model:

$$\dot{\theta}(t) = 1$$

What happens when the oscillator gets weak input s(t)?

$$\dot{x} = f(x) + \varepsilon s(t)$$

If ε is sufficiently small (!!) then the same transformation gives:

$$\dot{\theta} = 1 + \varepsilon Q(\theta) s(t)$$

Where $Q(\theta)$ can be computed from the oscillator equations:

$$Q(\theta) = \nabla_x \Theta(x(t))$$

- Note that computing this explicitly, and computing isochrons explicitly is tough.
- Gradient $Q(\theta)$ is the Infinitesimal Phase Reponse Function
- Can be computed from the dynamical system (Linearised adjoint)

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Aside: Kuramoto Model

Lets consider a network of N oscillators

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Let $\dot{\theta}_i = \omega_i$

$Q(\theta_i) = sin(\theta_i - \theta_j)$

$\varepsilon = K/N$

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Aside: Kuramoto Model

Lets consider a network of N oscillators

Let $\dot{\theta}_i = \omega_i$

$$\dot{\theta}_i = \omega_i + \frac{K}{N} \sum_{j=1}^{N} \sin(\theta_j - \theta_i)$$

$Q(\theta_i) = sin(\theta_i - \theta_j)$

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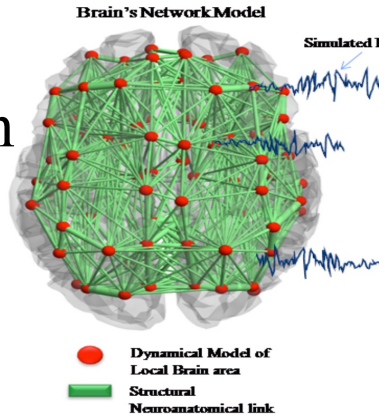
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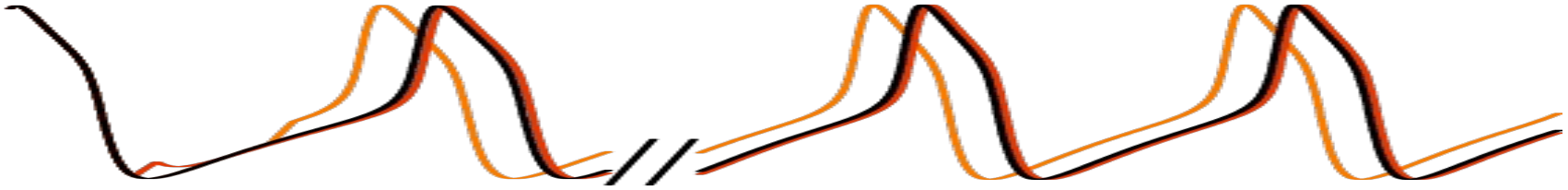
The Virtual Brain

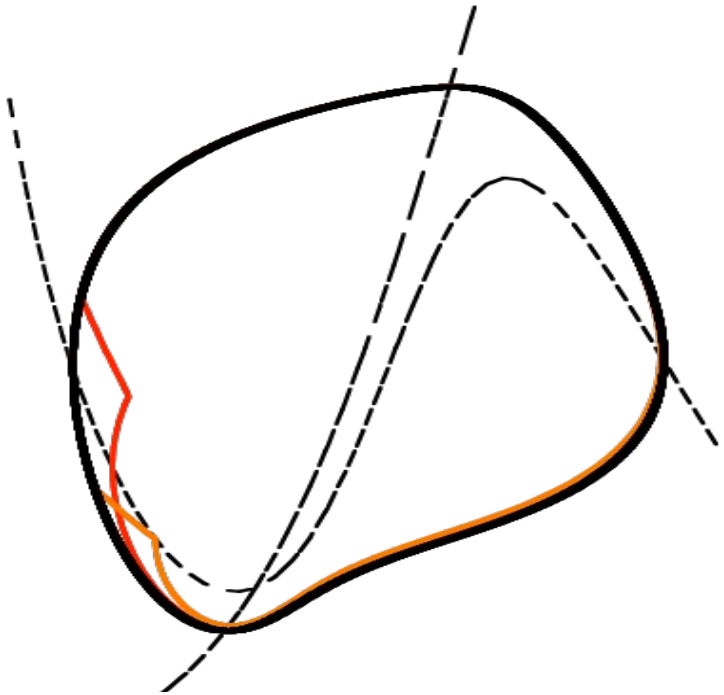
www.thevirtualbrain.org



$$\dot{\theta}_i = \omega_i + \frac{K}{N} \sum_{j=1}^{N} \sin(\theta_j - \theta_i)$$

Now we need to know how the oscillator will react to inputs?





For staters lets consider the input to be just a scaled delta function

$$\dot{x} = f(x(t)) + \varepsilon\delta(t - t')$$

$$\begin{aligned}
\frac{d\theta}{dt} &= \frac{\partial\theta}{\partial x}\frac{dx}{dt} = \frac{\partial\theta}{\partial x}\left(f(x) + \varepsilon\delta(t - t')\right) \\
&= Z(\theta)\left(f(x) + \varepsilon\delta(t - t')\right) \\
&= \varpi + Z(\theta)\varepsilon\delta(t - t') \\
&= \varpi + \varepsilon Z(\theta(t'))
\end{aligned}$$

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$Z(\theta)=PRC(\theta)$ is the **phase response curve**, or the phase resetting curve of the oscillator.

$$PRC(\theta) = \theta_{perturbed(new)} - \theta_{old}$$

Definition: **Phase Response Curve** tells how the phase is changed by an input timed at a particular phase (time)

Theoretical Methods to compute the infinitesimal PRC

Gradient: Kuramoto

$$PRC(\theta) = grad\Theta(x)$$

Adjoint: Malkin

$$\dot{PRC} = -\left\{Df(\gamma(t))\right\}^T PRC \qquad PRC(t)\cdot f(\gamma(t)) = \Omega$$

-this is the way XPP does it:

-Integrate for one cycle

-n**U**merics -> **A**veraging -> **N**ew adjoint -> replot

Direct method : Winfree

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

-This is the way you can do it for an arbitrary perturbation

-Measure in an experiment

Examples of PRC for simple oscillators and neural models

Perfect IF

$$\frac{dV}{dt} = I$$

$$\varpi = 2\pi I$$

$$PRC(\theta) = \frac{\varpi}{I} = 2\pi$$

Leaky IF

$$\frac{dV}{dt} = I - V \text{ with } V_r = 0 \text{ and } V_{threshold} = 1$$

$$\varpi = \frac{1}{T} = 2\pi\left[\ln\left(\frac{I}{I-1}\right)\right]^{-1}$$

$$PRC(\theta) = \frac{\varpi}{F(V(\theta))} = \varpi\left(1 - \exp\left(\frac{-2\pi}{\varpi}\right)\right)\exp\left(\frac{\theta}{\omega}\right)$$

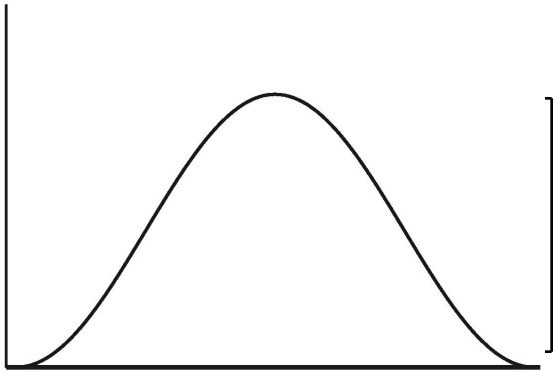
QIF

$$PRC(\theta) = \kappa(1 - \cos\theta)$$

For non-linear oscillators near the bifurcation

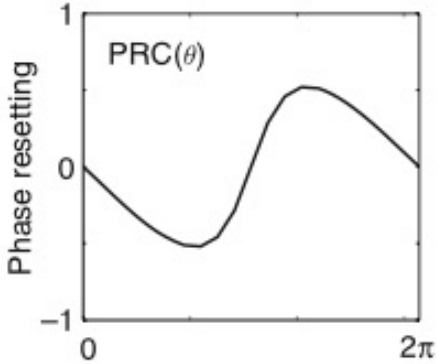
SNIC

$$PRC(\theta) = \kappa(1 - \cos\theta)$$



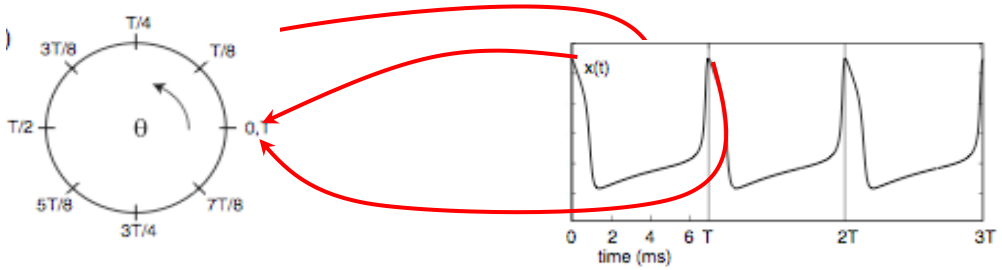
Hopf

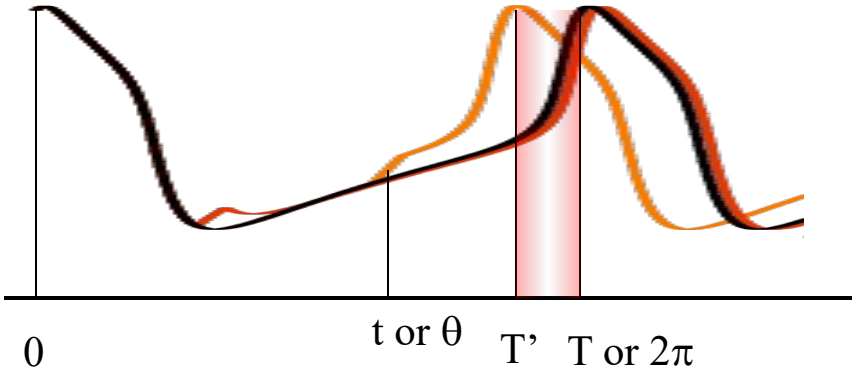
$$PRC(\theta) = \kappa\sin(\theta)$$



OK, what does this mean for neurons?

Natural position for x_0 -- the top of the spike, or the time of the spike





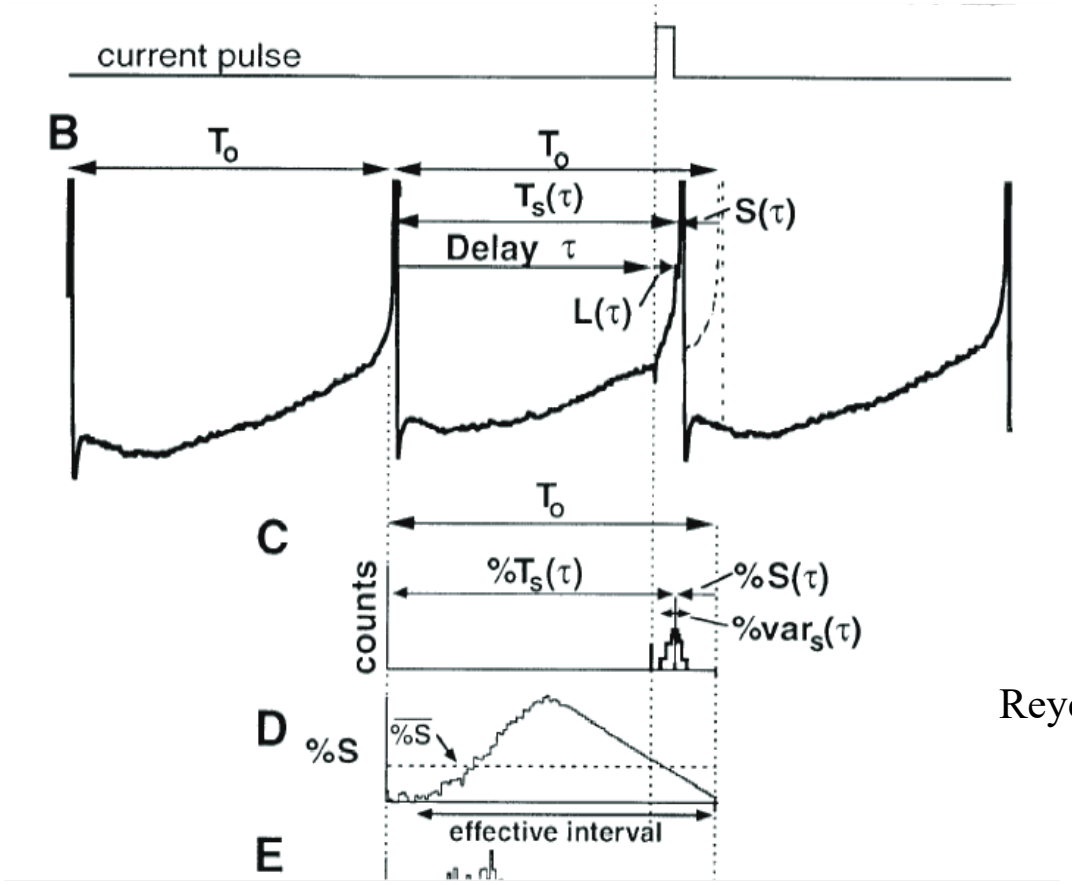
Now the PRC gives you the information about the timing of the spike

So another way we can define the resetting curve is

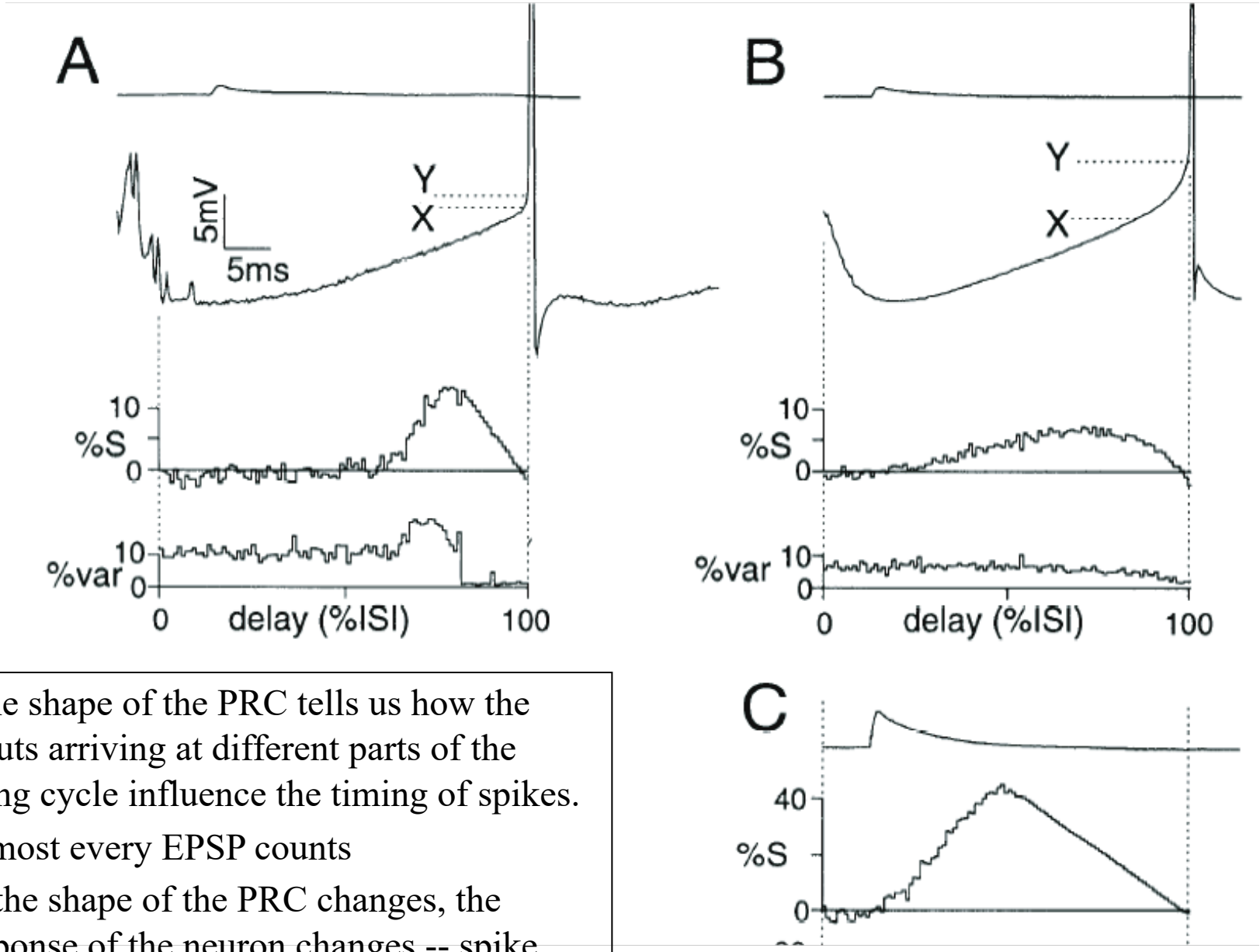
$$PRC=(ISI_{new}-ISI_{old})/ISI_{old}$$

PRC takes into account the internal biophysics of spike generation.

Experimental PRC Construction



Reyes and Fetz, 1993



- The shape of the PRC tells us how the inputs arriving at different parts of the firing cycle influence the timing of spikes.
- almost every EPSP counts
- if the shape of the PRC changes, the response of the neuron changes -- spike timing response.

Consider 2 weakly coupled oscillators

$$x_1' = F(x_1) + \varepsilon G_1(x_1, x_2)$$
$$x_2' = F(x_2) + \varepsilon G_2(x_1, x_2)$$

Let uncoupled oscillators have a stable limit cycle with period T, *U(t),* Then solution can be described by:

$$\dot{\theta}_1 = \nabla\Theta(x_1)\dot{x}_1 = 1 + \varepsilon\nabla\Theta(x_1)\bullet G_1(x_1, x_2)$$
$$\dot{\theta}_2 = \nabla\Theta(x_2)\dot{x}_2 = 1 + \varepsilon\nabla\Theta(x_2)\bullet G_2(x_1, x_2)$$

If ε is sufficiently small, $x_{1,2}(t)$ stays close to the uncoupled limit cycle $u_{1,2}(t)$

$$\dot{\theta}_1 = \nabla\Theta(x_1)\dot{x}_1 = 1 + \varepsilon\nabla\Theta(u(\theta_1)\bullet G_1(u(\theta_1), u(\theta_2))$$
$$\dot{\theta}_2 = \nabla\Theta(x_2)\dot{x}_2 = 1 + \varepsilon\nabla\Theta(\theta_2)\bullet G_2(u(\theta_1), u(\theta_2))$$

Apply the averaging trick (see Ermentrout's book)

$$\dot{\theta}_1 = \nabla\Theta(x_1)\dot{x}_1 = 1 + \varepsilon H_1(\theta_1 - \theta_2)$$
$$\dot{\theta}_2 = \nabla\Theta(x_2)\dot{x}_2 = 1 + \varepsilon H_2(\theta_2 - \theta_1)$$

$$\phi = \theta_2 - \theta_1$$

$$H_j(\phi) = \frac{1}{T}\int_0^t \nabla\Theta\bullet G_1(u(t+\phi), u(t))dt$$

$$\dot{\theta}_1 = \nabla\Theta(x_1)\dot{x}_1 = 1 + \varepsilon H_1(\theta_1 - \theta_2)$$

$$\dot{\theta}_2 = \nabla\Theta(x_2)\dot{x}_2 = 1 + \varepsilon H_2(\theta_2 - \theta_1)$$

Letting $\phi = \theta_2 - \theta_1$ We get:

$$\phi' = \varepsilon\{H_2(-\phi) - H_1(\phi)\} = \varepsilon d(\phi)$$

Phase locked solutions when $d(\phi) = 0$

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If $H_1 = H_2$ $\boxed{d(\phi) = -H_{odd}(\phi)}$

So the phase locked solution ϕ is stable when

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Phase locked solutions when $d(\phi) = 0$

If $H_1 = H_2$ $\boxed{d(\phi) = -H_{odd}(\phi)}$

So the phase locked solution ϕ is stable when

$\boxed{\dot{H}(\bar{\phi}) > 0}$

$$H_j(\phi) = \frac{1}{T}\int_0^t \nabla\Theta \bullet G_1(u(t+\phi), u(t))dt$$

$$H_j(\phi) = \frac{1}{T}\int_0^t X^*(t)G_j(X_0(t+\phi), X_0(t))dt$$

Where X^* is the solution to the linearized adjoint

$$\frac{dX^*}{dt} = -\left[D_x F(X_0(t))\right]^T X^*(t)$$

$$X^*(t)\frac{dX_0(t)}{dt} = 1$$

A pair of neural oscillators:

For two coupled neurons, the coupling is synaptic: $G(V_{pre},V)=s_{pre}(t)(E-V(t))$

$$H(\phi)=\frac{1}{T}\int_0^t V^*(t)s_{pre}(t-\phi)(E_s-V(t))dt$$

Where

$$V^*(t)=\lim_{a\to 0}\frac{PRC(t,a)}{a}T$$

(Hansel et al. 1995)

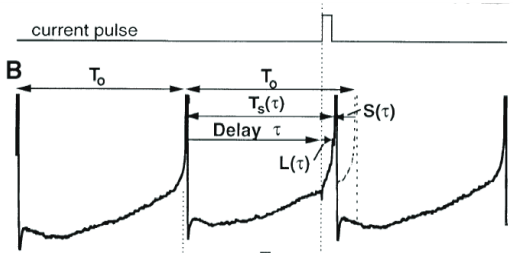
$$PRC(t,a)=(T(t,a)-T(t,0))/T(t,0))$$

So by looking at the slope of $H(\phi)$ when $\phi'=0$, we can determine the stability of the phase locked solutions

Back to Circuits: Synchrony

$$H = \frac{1}{T}\int_0^T \boxed{v^*(t)} \cdot (E_{syn} - v(t)) \cdot s(t+\phi)dt$$

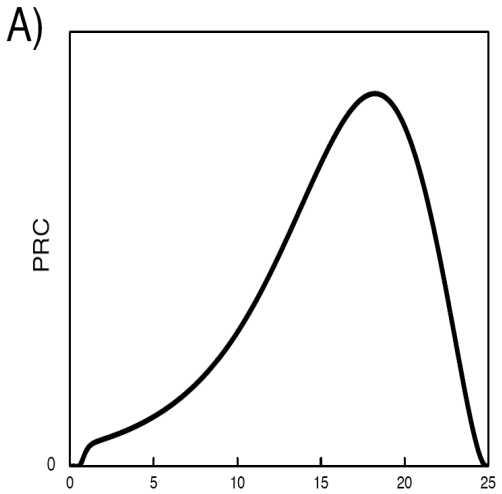
Phase Response Curve

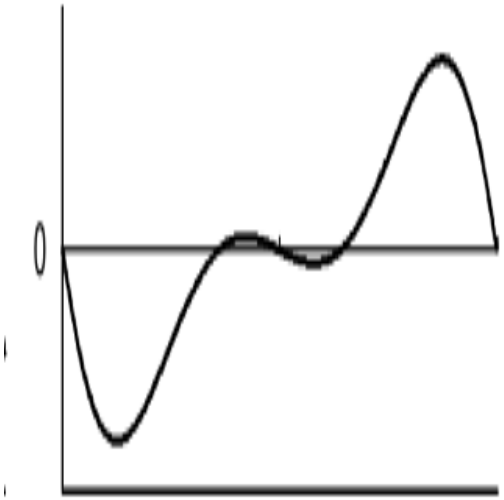


$$PRC(t) = \frac{T - \hat{T}(t)}{T}$$

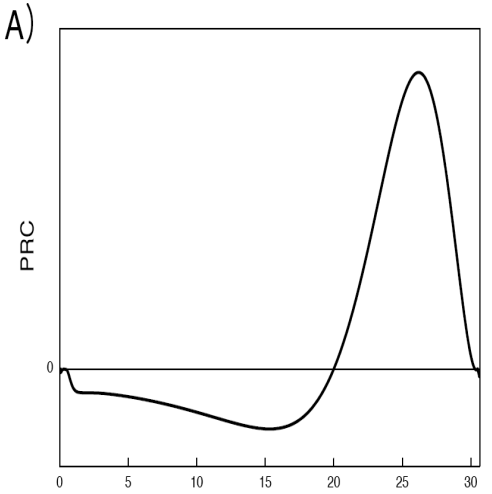
Hodd

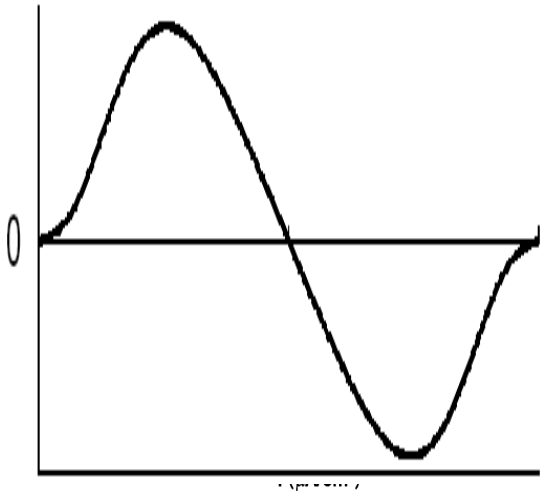
Type I





Type II

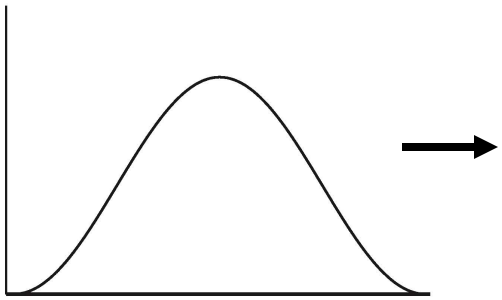




Spike to spike synchrony: 2 cell analysis

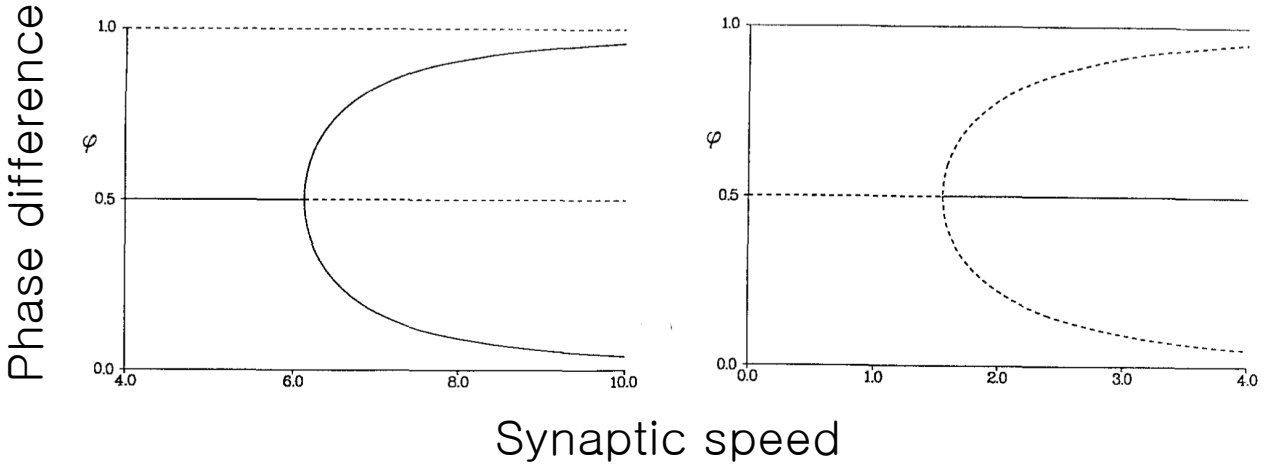
- Consider two coupled oscillating neurons
- Find phase configurations where neither neuron changes its neighbor phase: fixed phase difference solutions (phase-locked)
- By symmetry: in-phase and out of phase solutions are always possible
- Which is stable?

Type I



Excitation

Inhibition

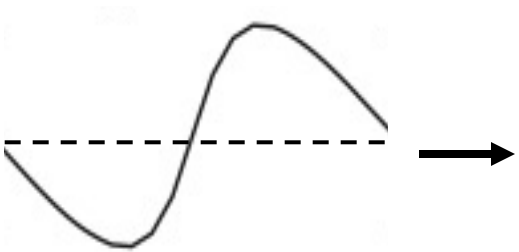


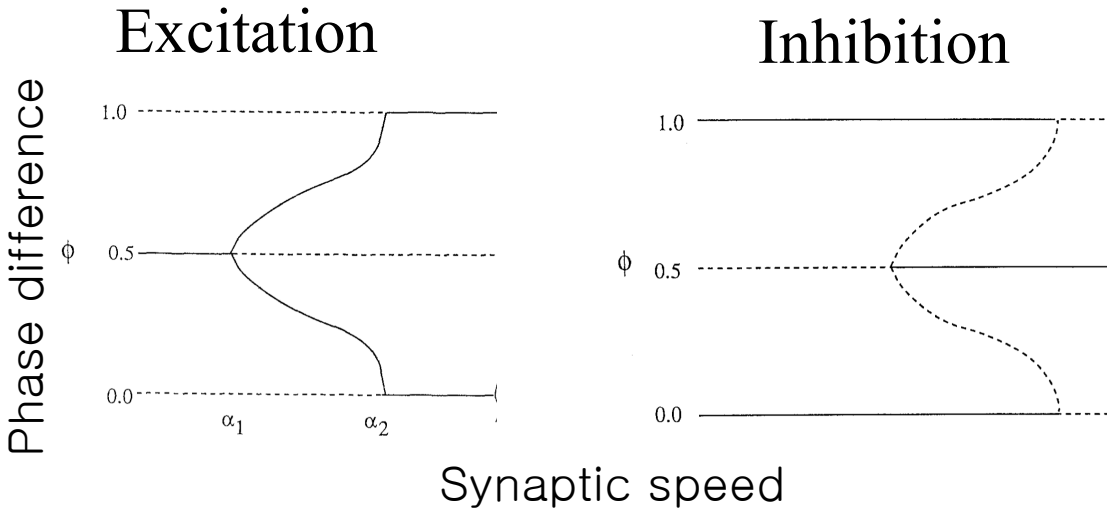
Van Vreeswijk et al (1995) :

Spike to spike synchrony: 2 cell analysis

- Consider two coupled oscillating neurons
- Find phase configurations where neither neuron changes its neighbor phase: fixed phase difference solutions (phase-locked)
- By symmetry: in-phase and out of phase solutions are always possible
- Which is stable?

Type II



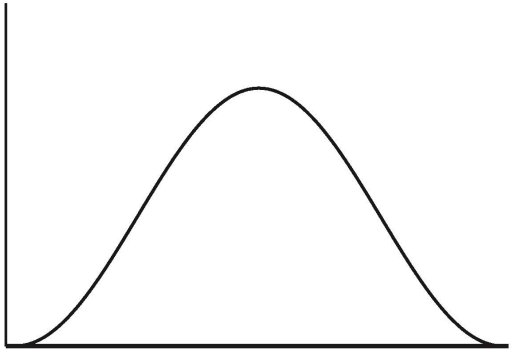


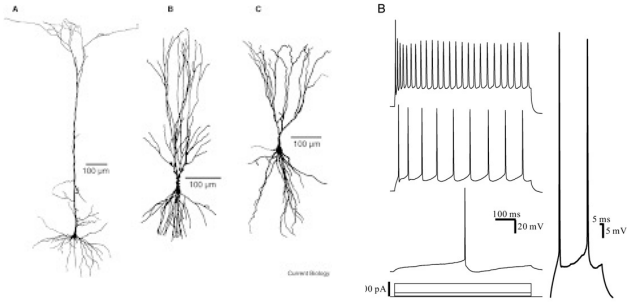
Van Vreeswijk et al (1995) :

Synchrony with inhibition or excitation depending on intrinsic properties

Type I

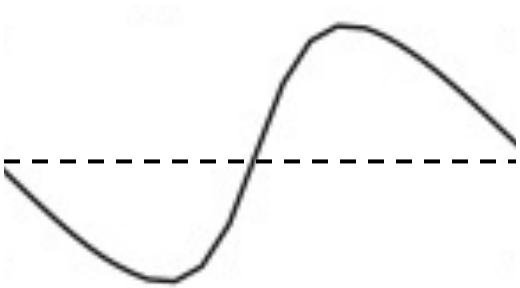
Fast excitation, slow inhibition

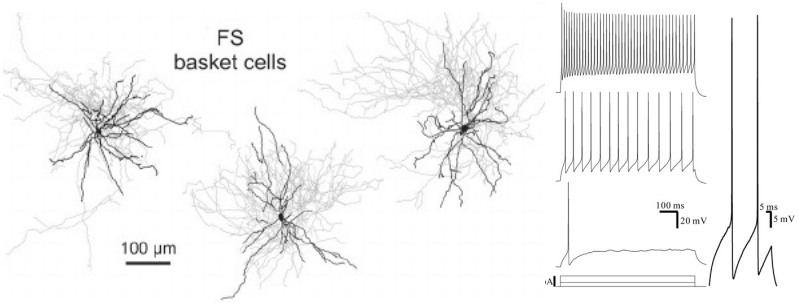




Type II

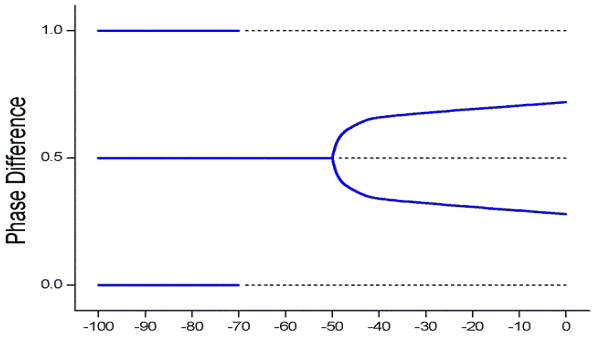
Fast inhibition, excitation



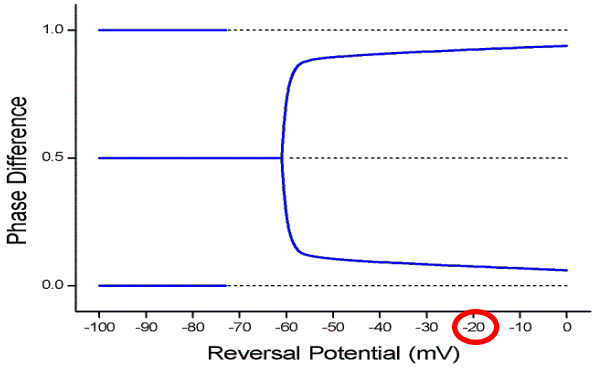


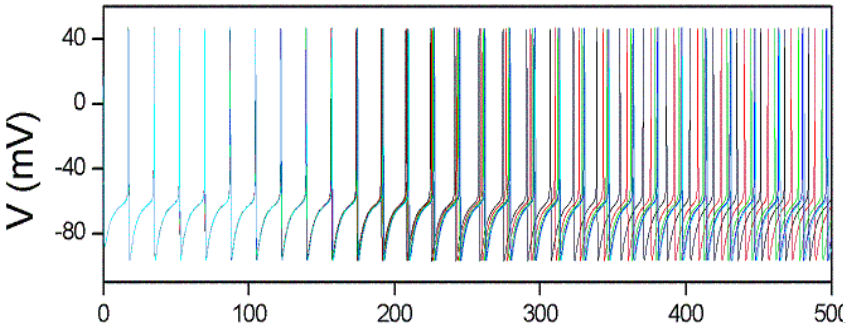
Synchronization depends on firing frequency

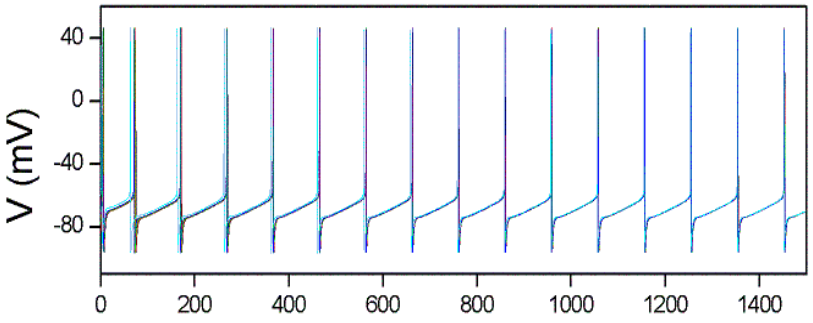
40Hz



10Hz

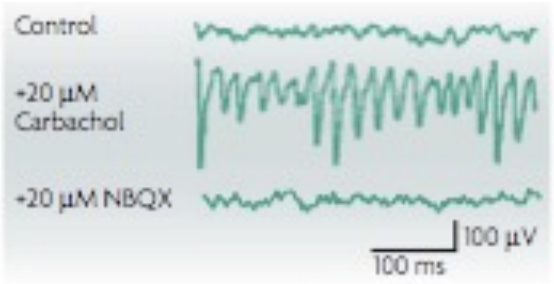


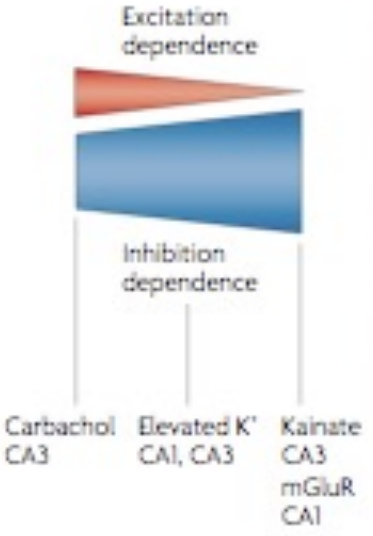


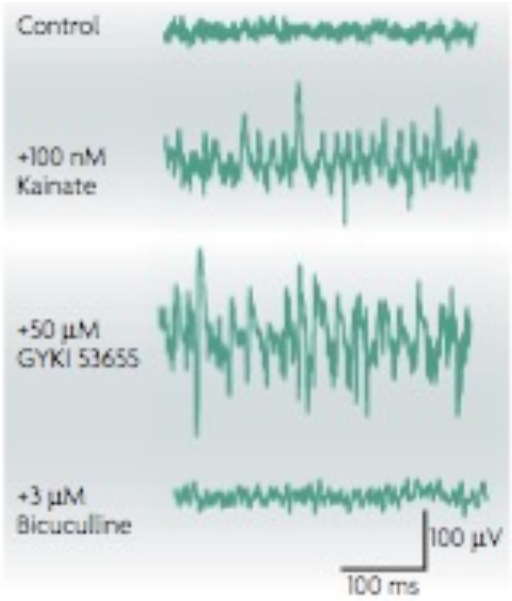


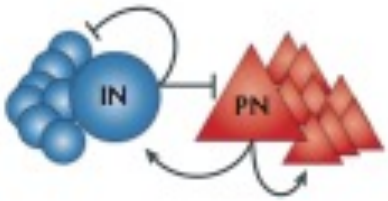
- Speed of synapse is relative to the firing frequency
- PRC shape changes with firing frequency

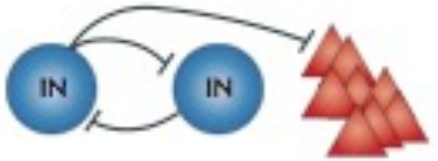
But what about ING and PING and networks?





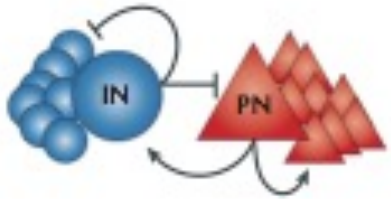


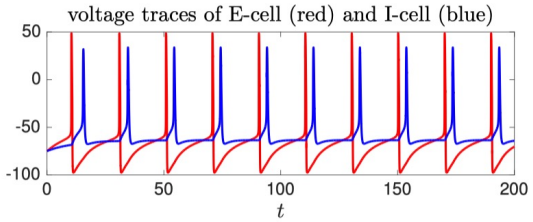


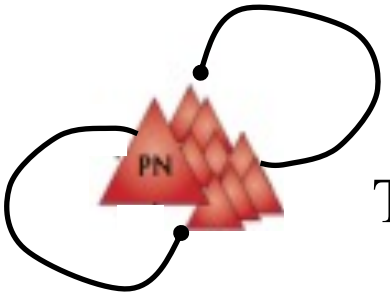


Them Gammas are not the same Gamma!

PING

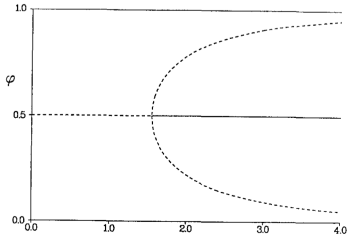






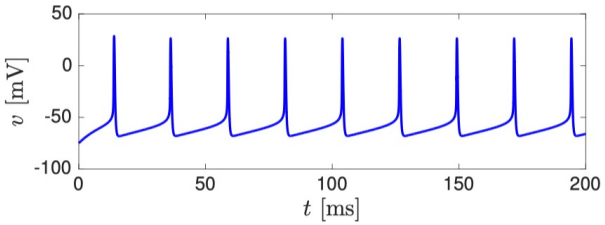
Type I

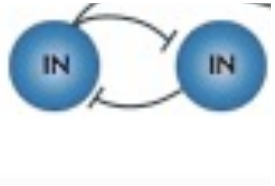
Net inhibitory coupling



ING

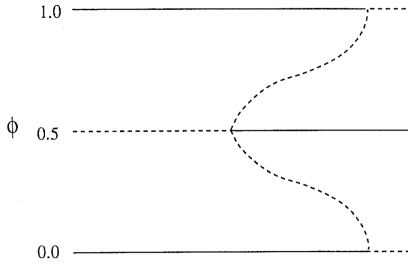






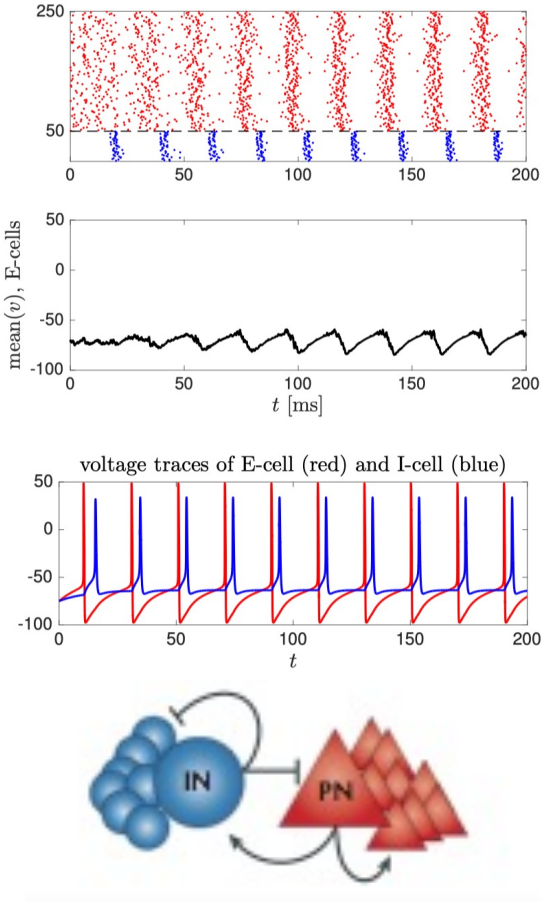
Type II

Inhibitory Coupling

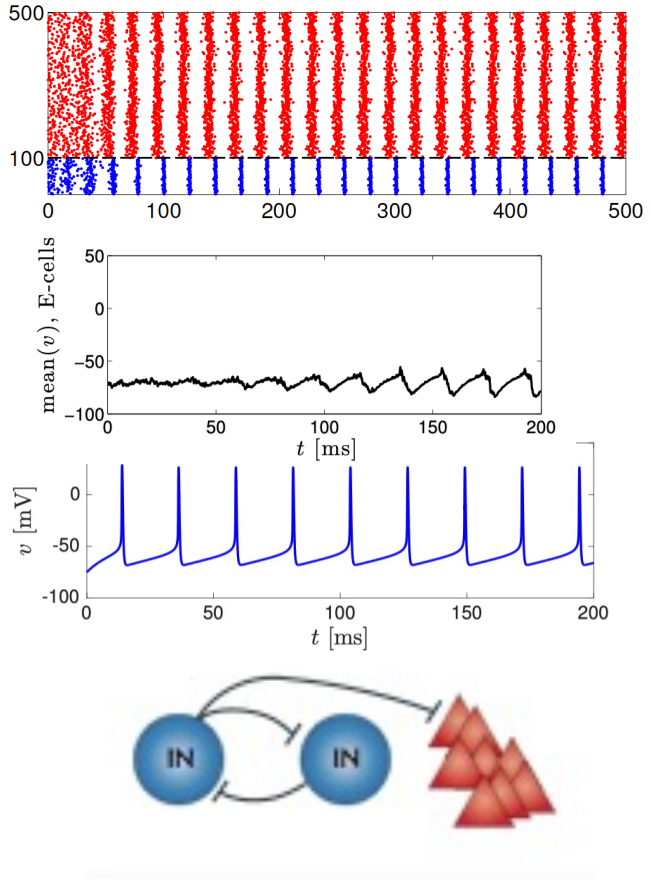


Them Gammas are not the same Gamma!

PING



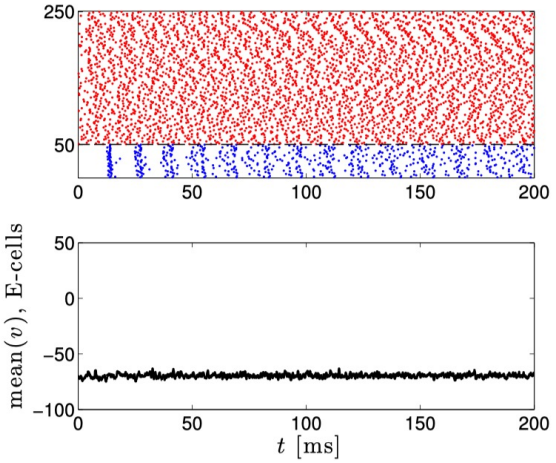
ING

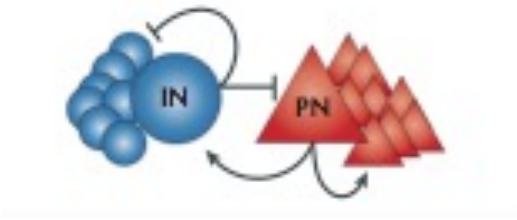


Them Gammas are not the same Gamma!

Strong E-E destroys PING

ING

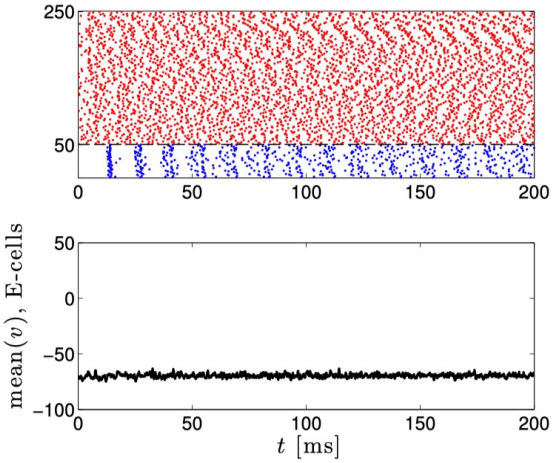


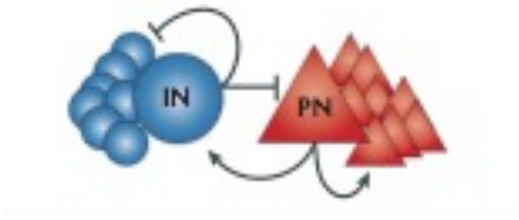




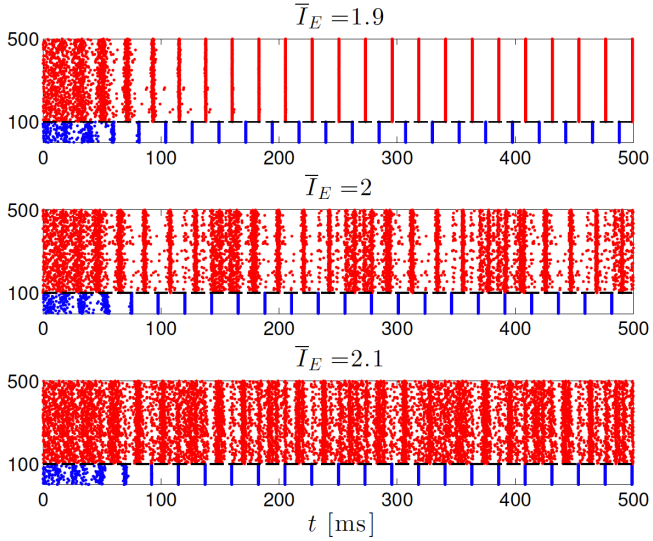
Them Gammas are not the same Gamma!

Strong E-E destroys PING



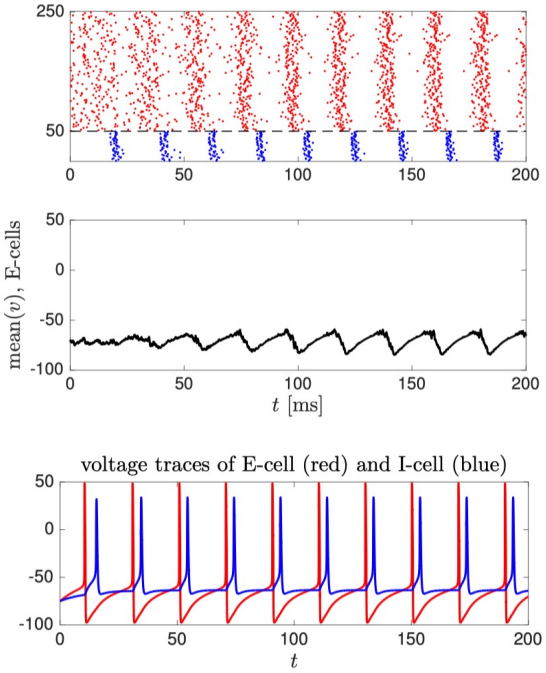


Strong Input to E-cells destroys ING





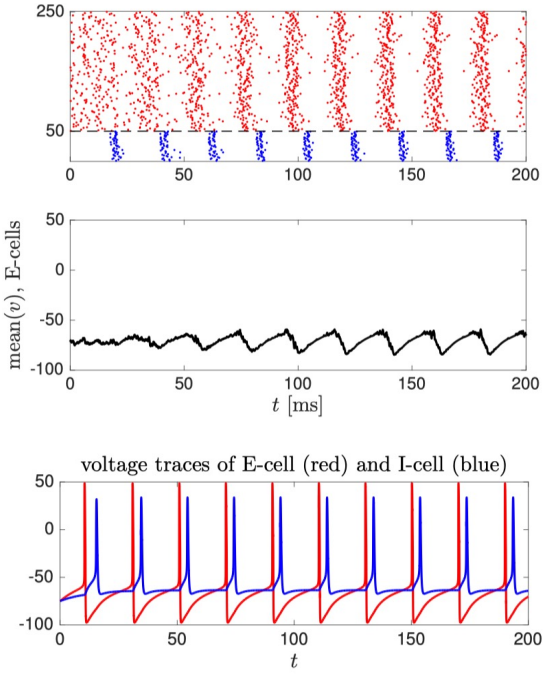
Gamma in large populations
Reduction to neural mass



$$C\dot{V} = I_{\text{app}} - \bar{g}_{\text{Na}} m_\infty^3(V)(1 - W) \times (V - V_{\text{Na}}) - \bar{g}_{\text{K}}(W/S)^4(V - V_{\text{K}}) - g_L(V - V_L) \quad (1)$$

$$\dot{W} = \phi[W_\infty(V) - W]/\tau(V) \quad (2)$$

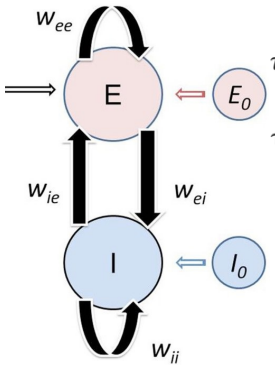
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Wilson-Cowan

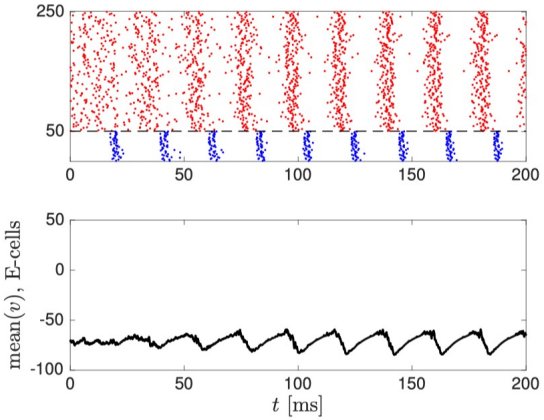


$$\tau_e \frac{dr_e}{dt} = -r_e + f(w_{ee}r_e - w_{ei}r_i + I_e)$$

$$\tau_i \frac{dr_i}{dt} = -r_i + f(w_{ie}r_e - w_{ii}r_i + I_i)$$

F(...) is a sigmoid

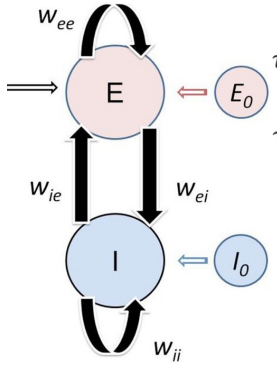
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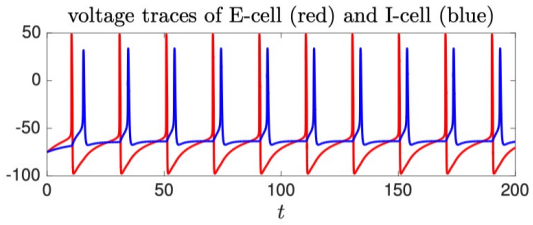
Wilson-Cowan

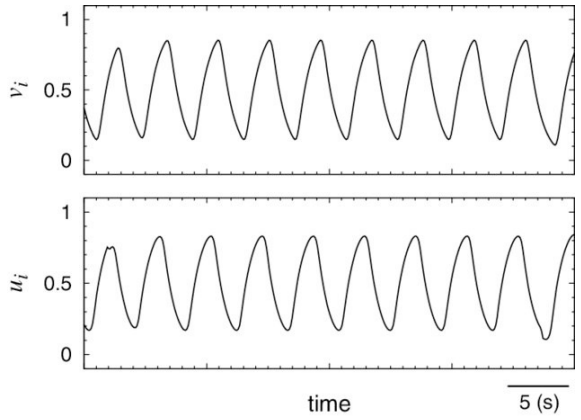


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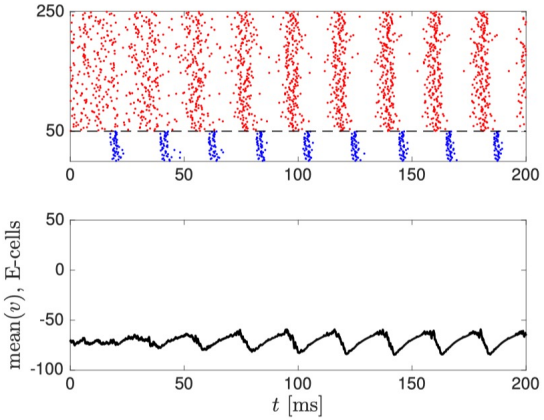
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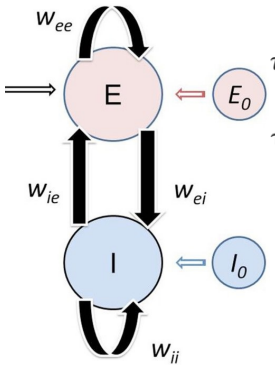
Gamma in large populations
Reduction to neural mass



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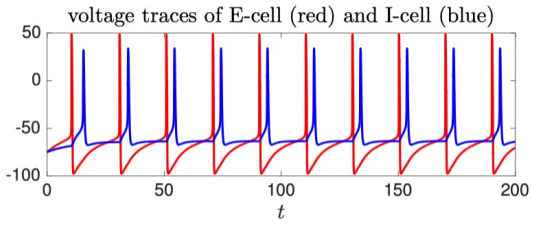
Wilson-Cowan

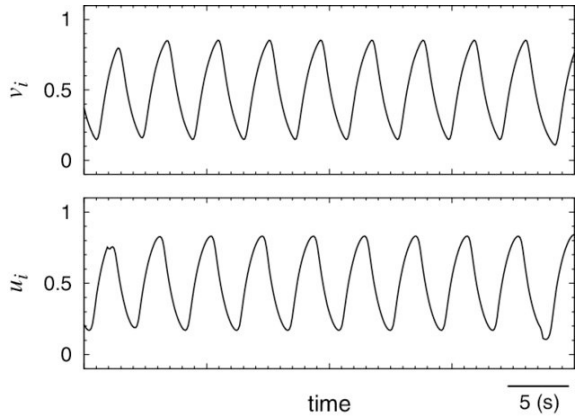


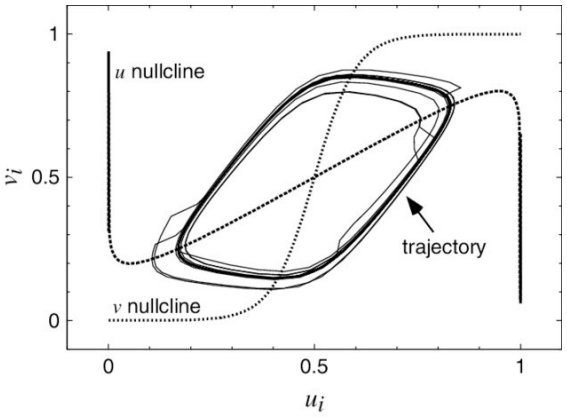
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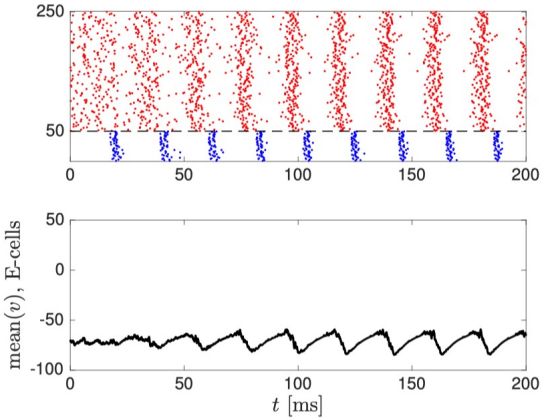
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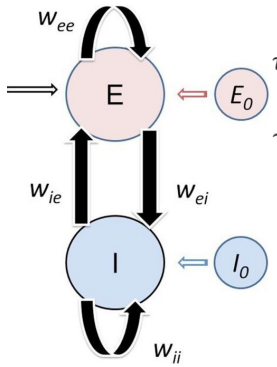
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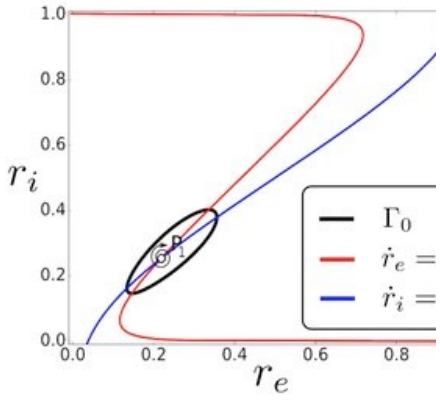
Wilson-Cowan

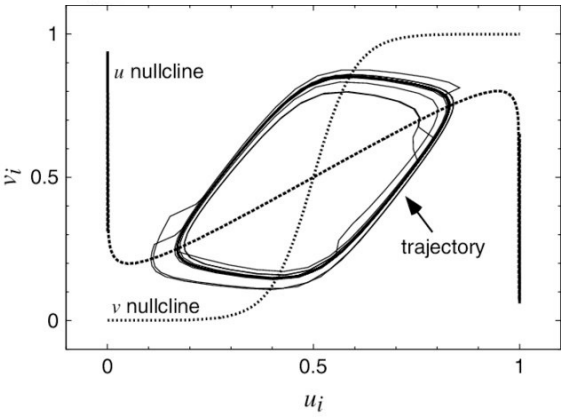


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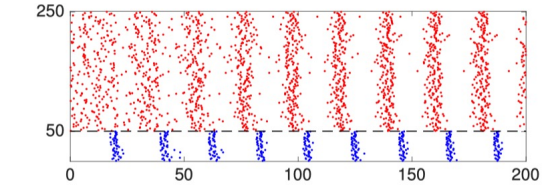
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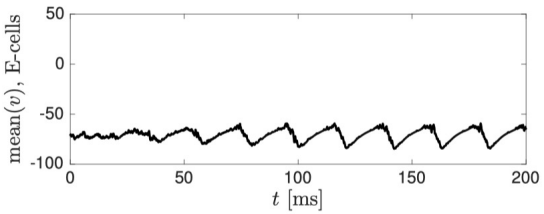
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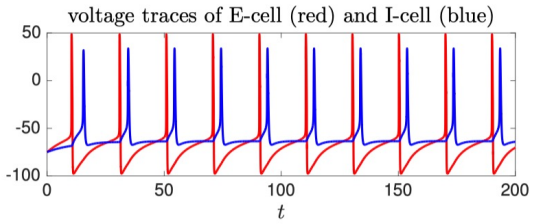




Gamma in large populations
Reduction to neural mass



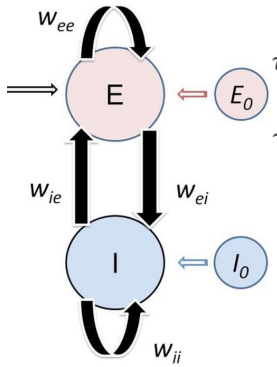




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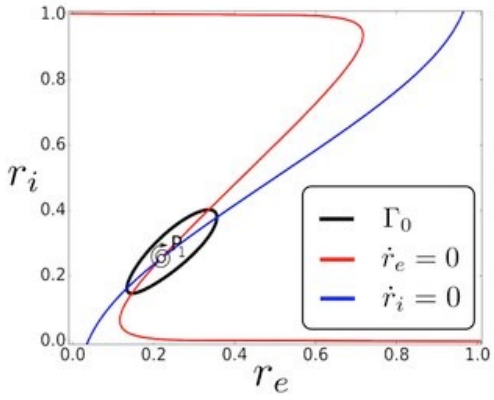
Wilson-Cowan

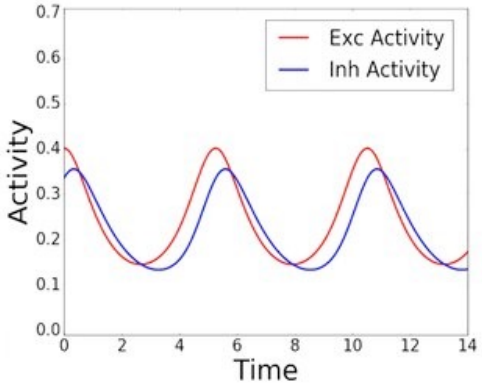


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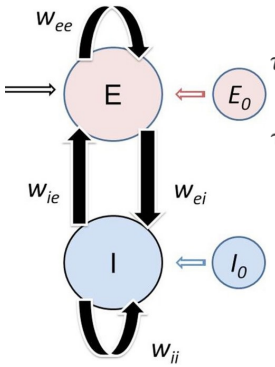




Gamma in large populations
Reduction to neural mass

- Ad hoc description
- Can do better!
- EXACT REDUCTION

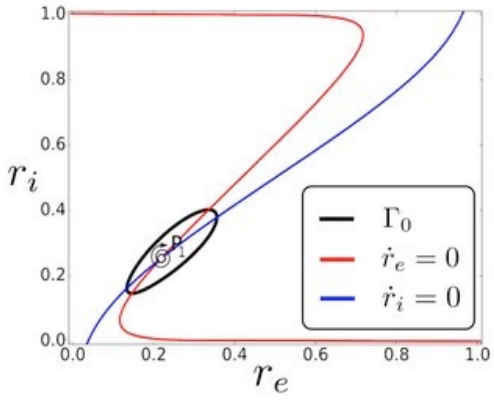
Wilson-Cowan

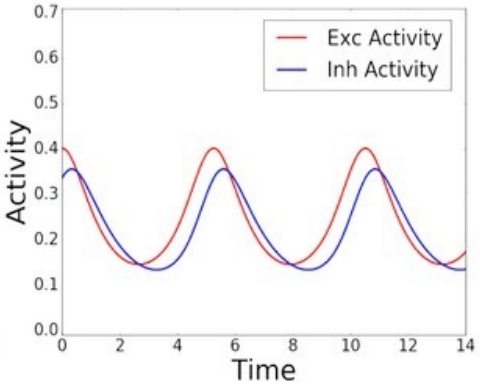


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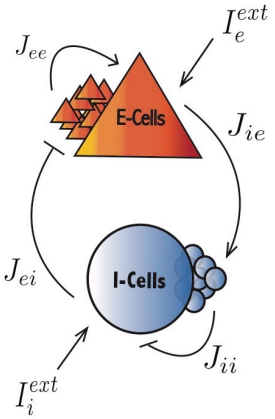
Exact reduction of E-I Networks

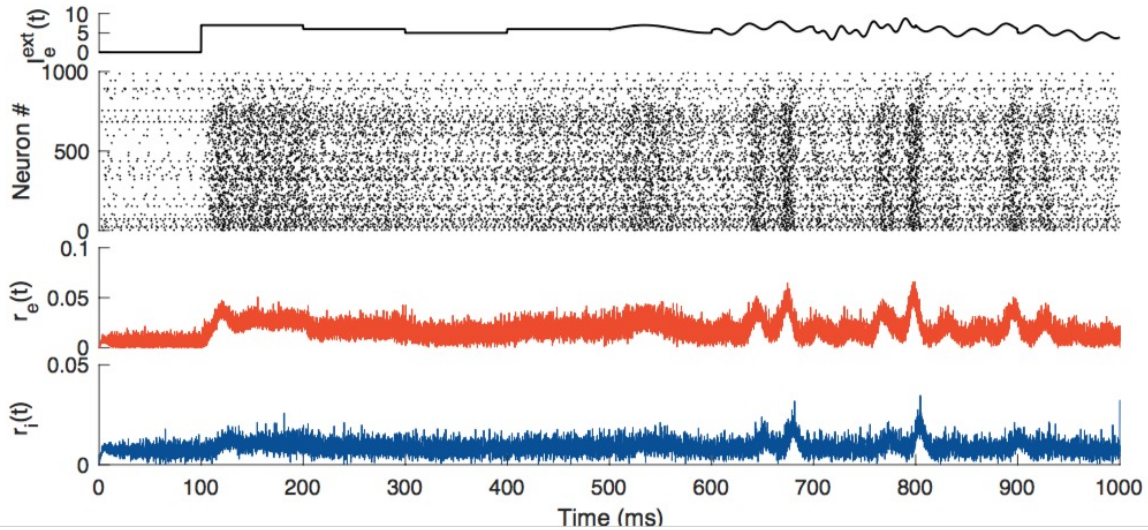
Simulation of a **cortical network** made up of 1000 neurons (80% E-cells, 20% I-cells)

$$\tau \frac{d}{dt} v(t) = \eta + v^2(t) + I(t)$$ QIF neurons

$$I(t) = I_e^{ext}(t) + s_{ee}(t) - s_{ei}(t), \quad I(t) = I_i^{ext}(t) + s_{ie}(t) - s_{ii}(t),$$

$$\tau_s \frac{d}{dt} s(t) = -s + Jr(t) \qquad r(t) = \frac{1}{N} \sum_j \sum_f \delta(t - t_j^f)$$

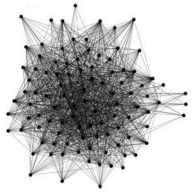




The Exact Reduction Method

Reduction method

$$\tau \frac{d}{dt} v(t) = \eta + v^2(t) + I(t)$$



Mean field

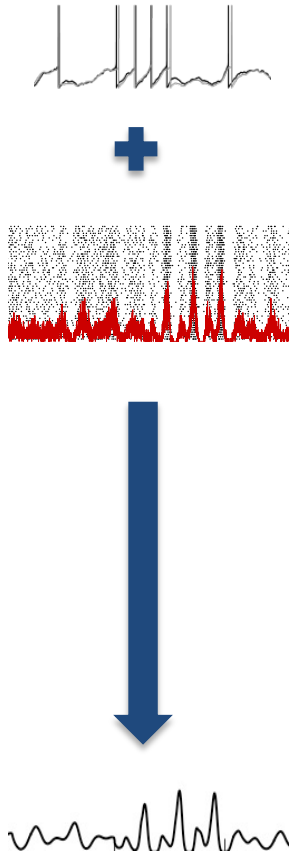
$$\tau \frac{\partial}{\partial t} p(t, v \mid \eta) + \frac{\partial}{\partial v}\left[\left(\eta + v^2 + I(t)\right) p(t, v \mid \eta)\right] = 0$$

MPR Reduction

$$\tau \frac{d}{dt} V = \bar{\eta} + V^2 + I(t) - \pi^2 \tau^2 r^2$$

$$\tau \frac{d}{dt} r = \frac{\Delta}{\pi\tau} + 2\pi V r$$

Neural activity



Montbrio, E., Pazo, D., Roxin, A. Phys.Rev. X 5, 021,028 (2015)

Mean-field description

Probability Density Function $\rho(V^X|\eta,t)$

- In a population of neurons, each neuron may be in a different internal state.
- Track the distribution of neurons over state space instead of tracking individual neurons.

- Define flux through the firing boundary as $J(V^X|\eta,t) = \frac{dV^X}{dt}\rho(V^X|\eta,t)$:

Continuity Equation

$$\frac{\partial}{\partial t}\rho(V^X|\eta,t) = -\frac{\partial}{\partial V^X}J(V^X|\eta,t)$$

Continuity Equation

$$\frac{\partial}{\partial t}\rho(V^X|\eta,t) = -\frac{\partial}{\partial V^X}J(V^X|\eta,t)$$

Solve continuity equation

$\rho(V^X|\eta,t) = ?$

Solve continuity equation

$$\rho(V^X|\eta,t) = ?$$

$$\frac{\partial}{\partial t}\rho(V^X|\eta,t) = -\frac{\partial}{\partial V^X}J(V^X|\eta,t)$$

Lorentzian ansatz:

For QIF neuron models, the variables V of the microscopic system always follow a Lorentzian distribution of the form

$$\rho(V^X|\eta,t) = \frac{1}{\pi}\frac{x(\eta,t)}{[V^X - y(\eta,t)]^2 + x(\eta,t)^2}$$

[Montbrío et al. 2015]

Solutions:

$$C_m\frac{\partial x(\eta,t)}{\partial t} = 2axy + (b - \sum_Y g_{XY})x$$

$$C_m\frac{\partial y(\eta,t)}{\partial t} = -ax^2 + ay^2 + (b - \sum_Y g_{XY})y + c - u^X + I_{ext} + \sum_Y g_{XY}E_r^Y + \eta$$

Firing rate and mean voltage

$$r_X = lim_{V^X \to V_{peak}} \int J(V^X|\eta,t) g(\eta) d\eta$$

$$v_X = \int \int \rho(V^X|\eta,t) g(\eta) dV d\eta$$

$$r_X = \frac{a}{C_m} \int x(\eta,t)(g(\eta)) d\eta$$

$$v_X = \int \int \frac{x(\eta,t)}{\pi} \frac{V^X}{[V^X - y(\eta,t)]^2 + x(\eta,t)^2} g(\eta) dV d\eta$$

If $g(\eta) = \frac{1}{\pi} \frac{\Delta}{(\eta - \overline{\eta})^2 + \Delta^2}$, we can use Cauchy principal value and the residue theorem to solve the above equations:

$$r_X = \frac{a}{C_m \pi} x(\overline{\eta} - i\Delta, t)$$

$$v_X = y(\overline{\eta} - i\Delta, t)$$

$$\tau \frac{d}{dt} V = \overline{\eta} + V^2 + I(t) - \pi^2 \tau^2 r^2$$

$$\tau \frac{d}{dt} r = \frac{\Delta}{\pi\tau} + 2\pi V r$$

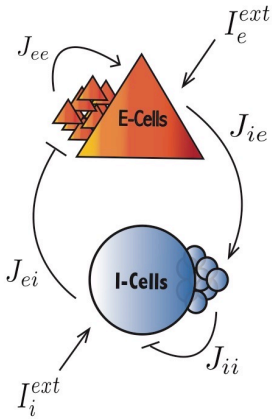
Exact Reduced Gamma Models

$$
\begin{cases}
\tau_e \dfrac{d}{dt} r_e = \dfrac{\Delta_e}{\pi \tau_e} + 2 r_e V_e \\
\tau_e \dfrac{d}{dt} V_e = V_e^2 + \bar{\eta}_e + I_e - \tau_e^2 \pi^2 r_e^2, \\
\tau_s \dfrac{d}{dt} s_{ee} = -s_{ee} + J_{ee} r_e, \\
\tau_s \dfrac{d}{dt} s_{ei} = -s_{ei} + J_{ei} r_i,
\end{cases}
$$

$$
\begin{cases}
\tau_i \dfrac{d}{dt} r_i = \dfrac{\Delta_i}{\pi \tau_i} + 2 r_i V_i \\
\tau_i \dfrac{d}{dt} V_i = V_i^2 + \bar{\eta}_i + I_i - \tau_i^2 \pi^2 r_i^2. \\
\tau_s \dfrac{d}{dt} s_{ie} = -s_{ie} + J_{ie} r_e, \\
\tau_s \dfrac{d}{dt} s_{ii} = -s_{ii} + J_{ii} r_i,
\end{cases}
$$

$$I_e = I_e^{ext} + \tau_e s_{ee} - \tau_e s_{ei},$$

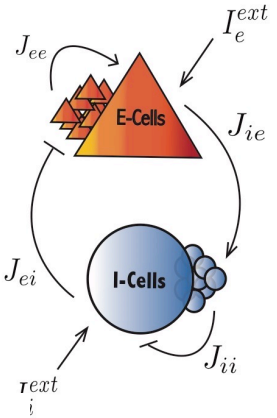
$$I_i = I_i^{ext} + \tau_i s_{ie} - \tau_i s_{ii}.$$

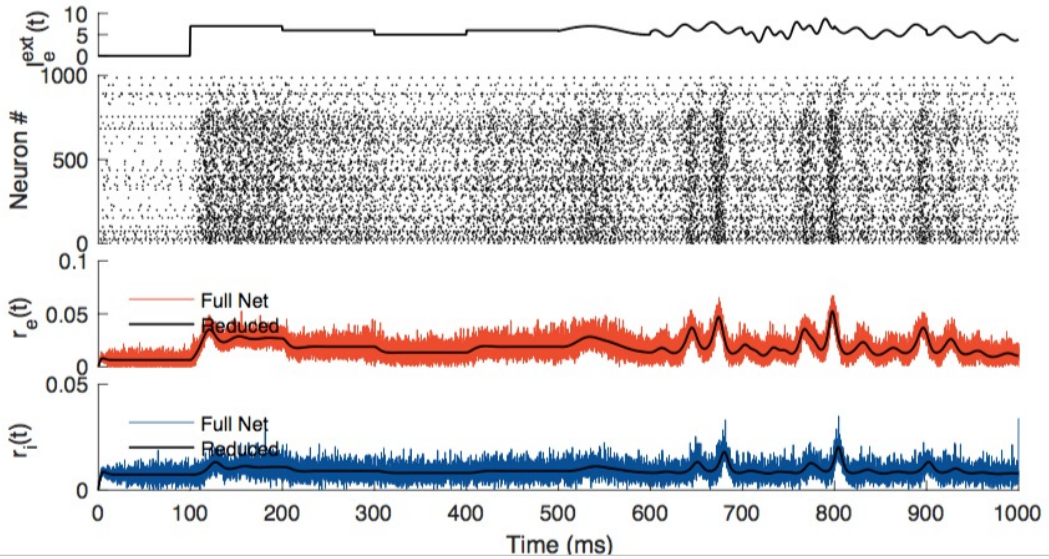


Dumont and Gutkin PRE (2018); PLOS CB 2019.

Comparison with the Full Network

Comparison between a simulation of the full spiking neural using the QIF model and the activity generated by the mean-field equation

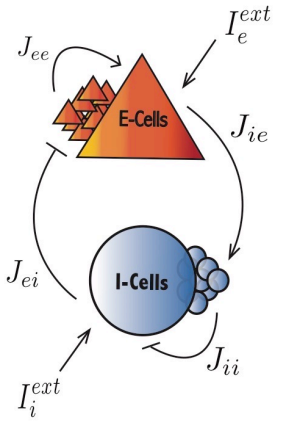


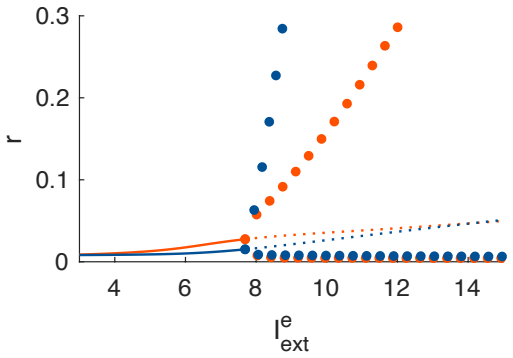


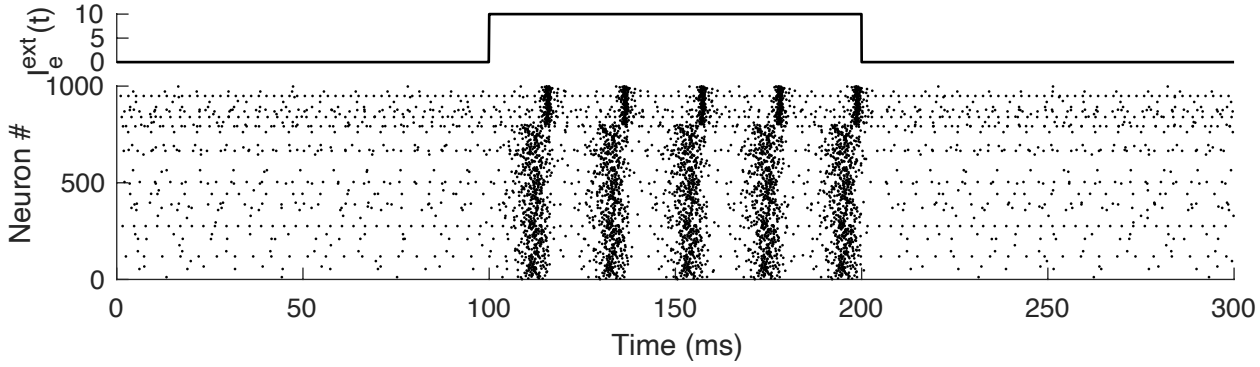
Emergence of Oscillations

We can study the **stability** of the dynamical system:

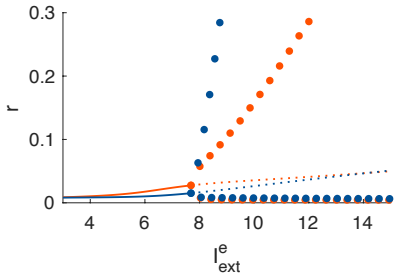
- Bifurcation diagrams made with XPPAUT
- Emergence of the PING oscillations due to intercoupling
- Note that here most neurons are NOT oscillators!

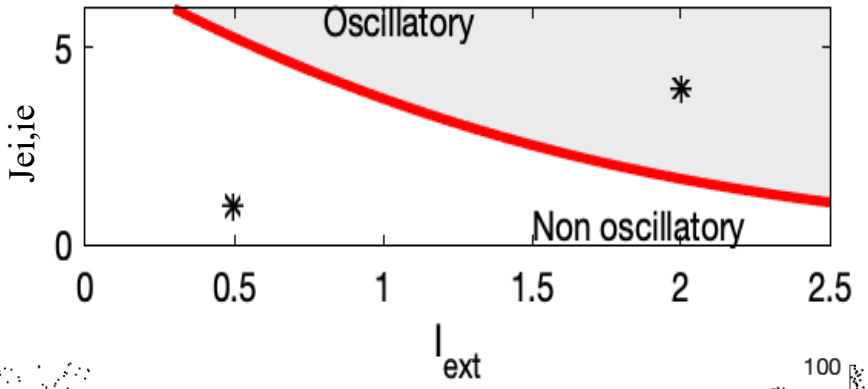


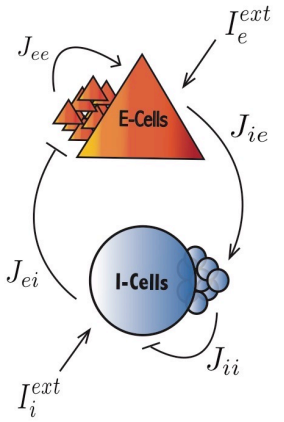


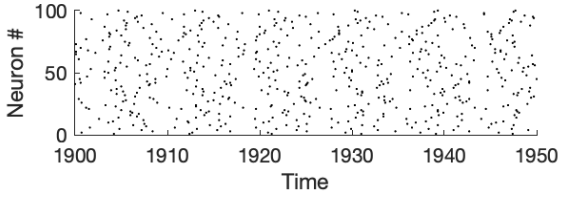


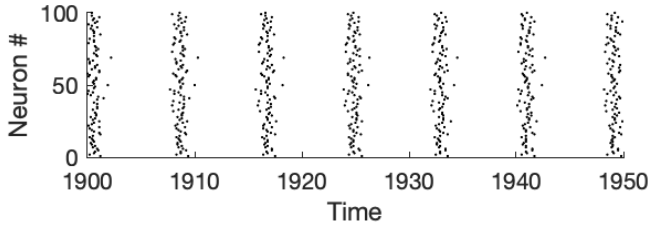
Emergent PING

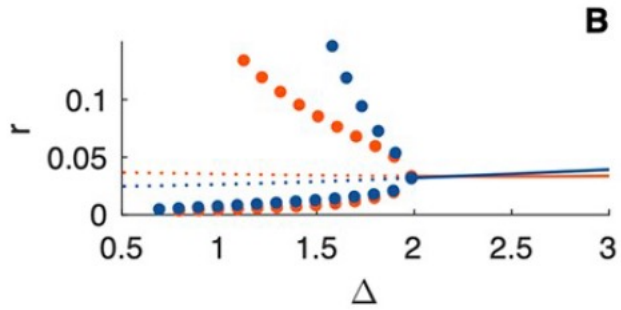


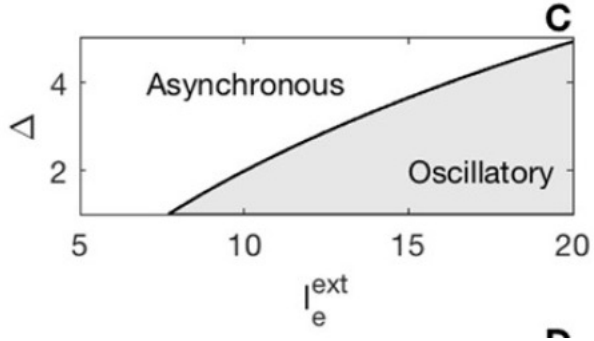








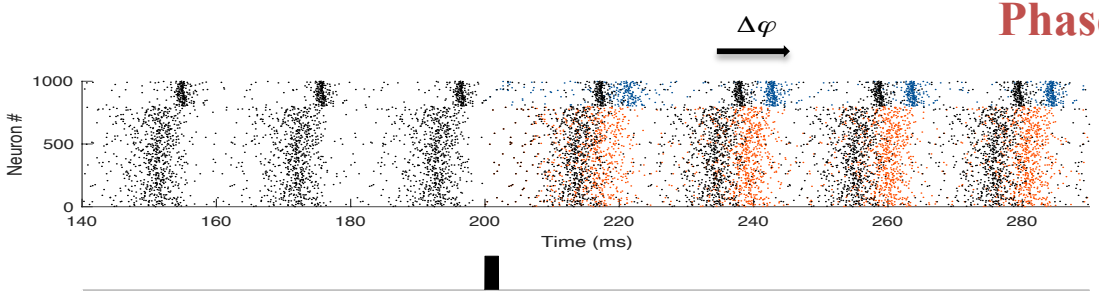


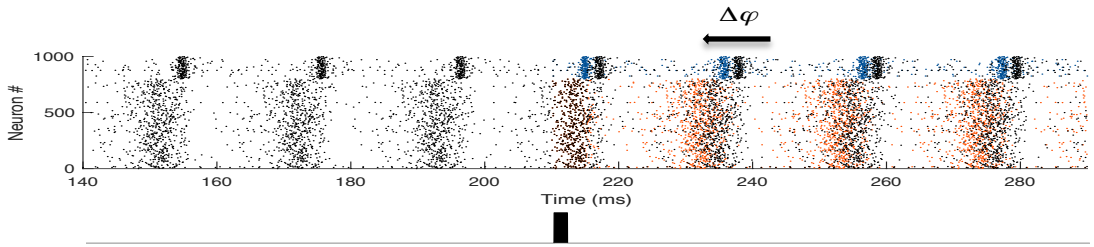


Phase Resetting Curve

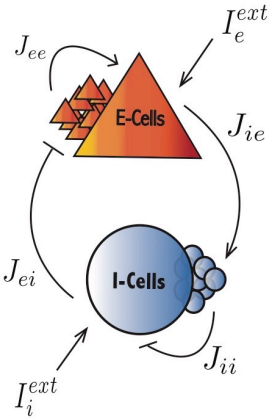
PRC obtained by **direct** stimulation

Phase delay





Phase advance

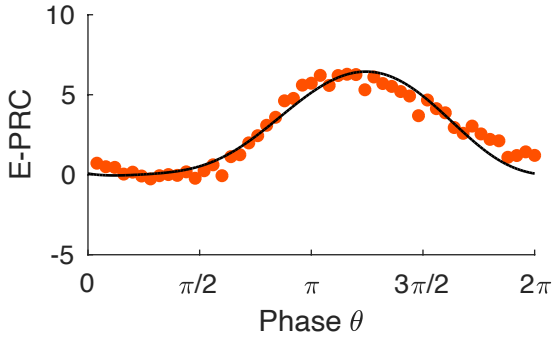


G. Dumont, B. Ermentrout, B. Gutkin (2017), PRE

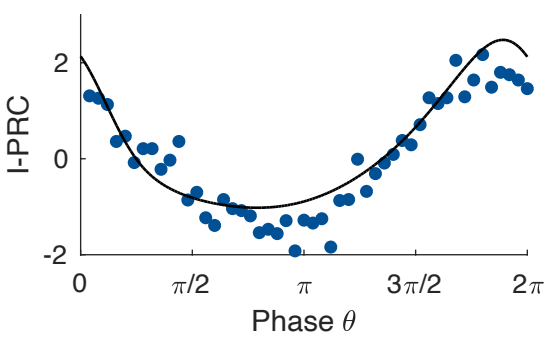
Phase Resetting Curve

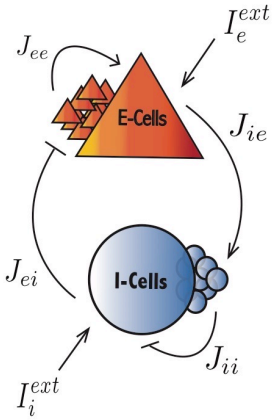
PRC obtained by **direct** stimulation **compared** with the **theory**

Type I PRC



Type II PRC

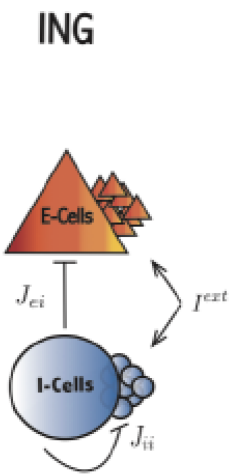


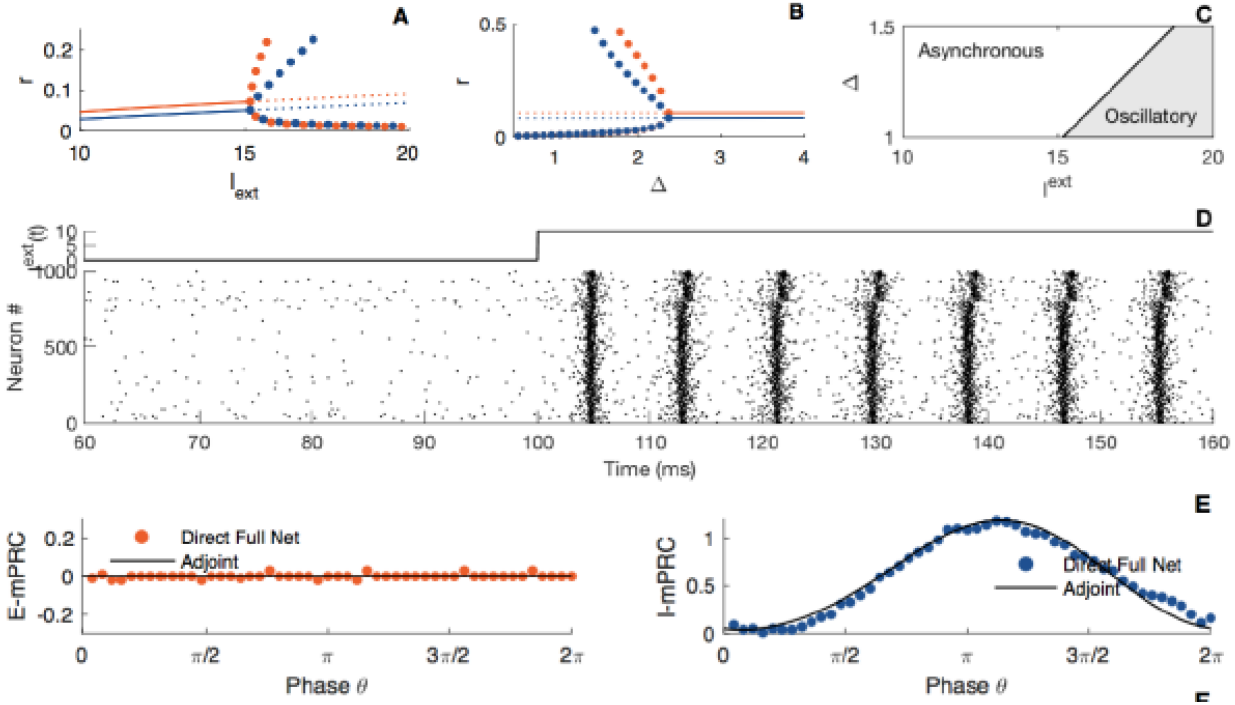


Only the **I-cells** can explain the **type II PRC**

G. Dumont, B. Ermentrout, B. Gutkin (2017), PRE

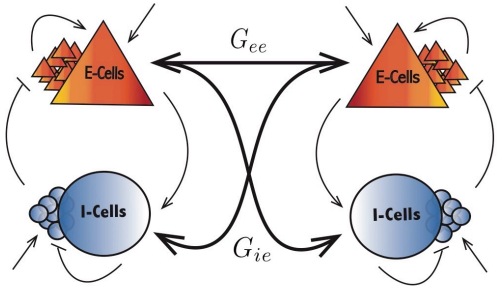
ING emergence



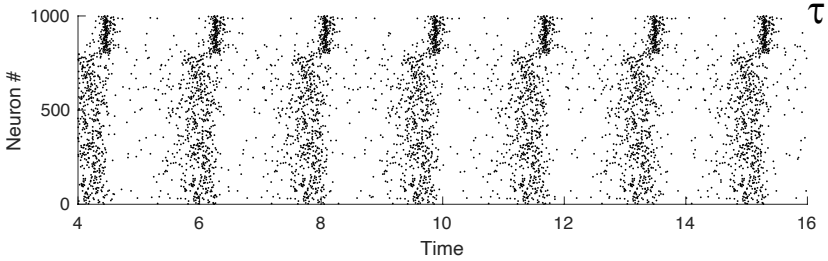


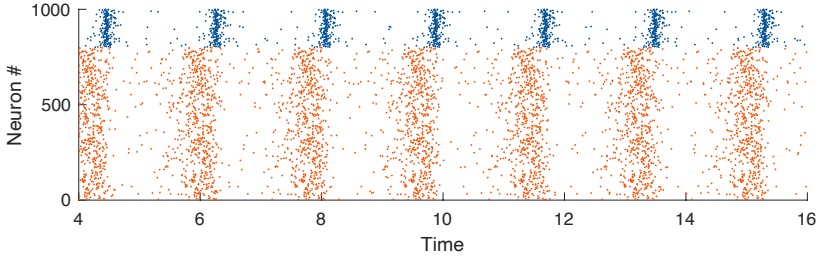
G. Dumont, B. Ermentrout, B. Gutkin (2017), PRE

Two Neural Networks



τ – delay



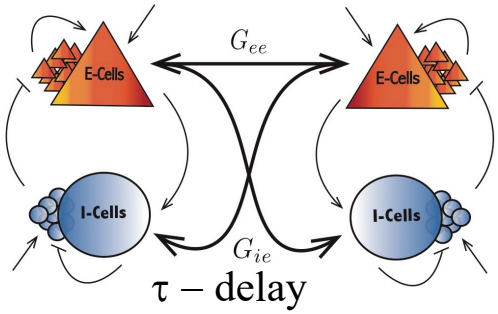


Let us define $\varphi_1(t)$ the phase of the first network and $\varphi_2(t)$ of the second network.

Can we *predict* the dynamics of the **phase difference**?

$$\theta(t) = \varphi_1(t) - \varphi_2(t)$$

Weakly Coupled Oscillators



Within the framework of **weakly coupled oscillators**, we can reduce the full system to a simple **phase equation**

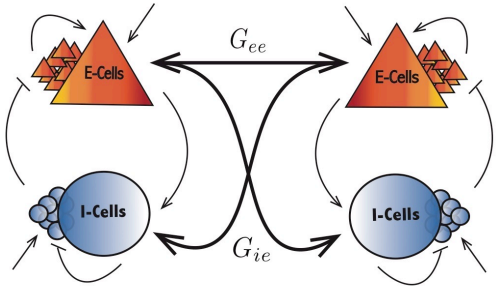
$$\frac{d}{dt}\theta(t) = G(\theta(t))$$

Where G is the odd part of the **interaction function** H:

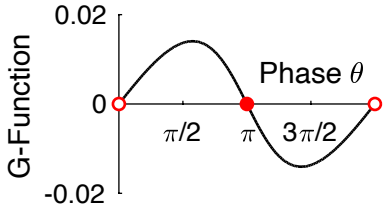
$$H(\theta) = \frac{G_{ee}}{T}\int_0^T PRC_e(s)r_e(\theta - s)\,ds + \frac{G_{ie}}{T}\int_0^T PRC_i(s)r_e(\theta - s)\,ds$$

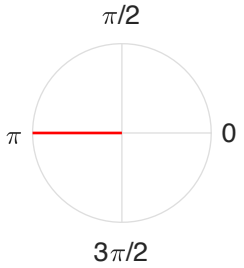
Anti-phase Locking

Reduction of the system to a **phase equation**

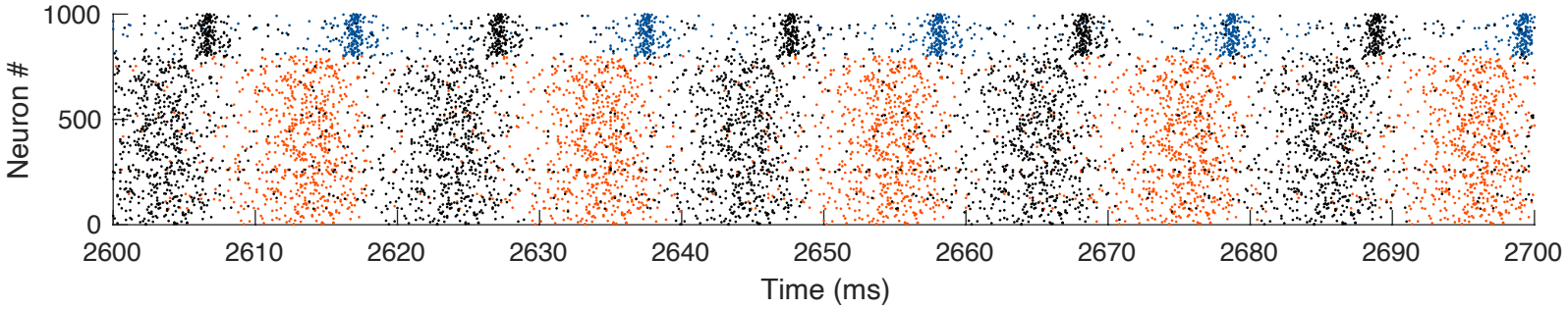


$$\frac{d}{dt}\theta(t) = G(\theta(t))$$



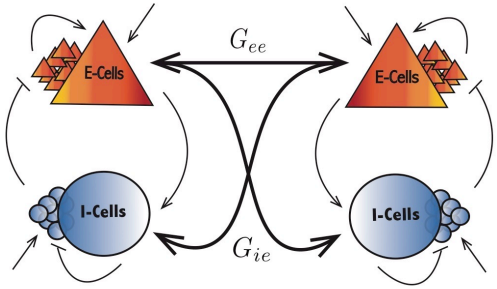


The **zeros** of the G-function determine the **locking modes**

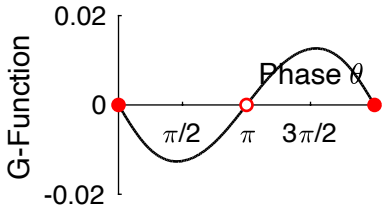


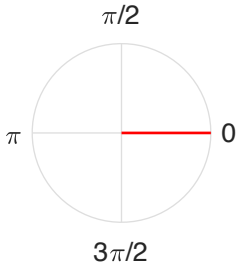
In-Phase Locking

Reduction of the system to a **phase equation**

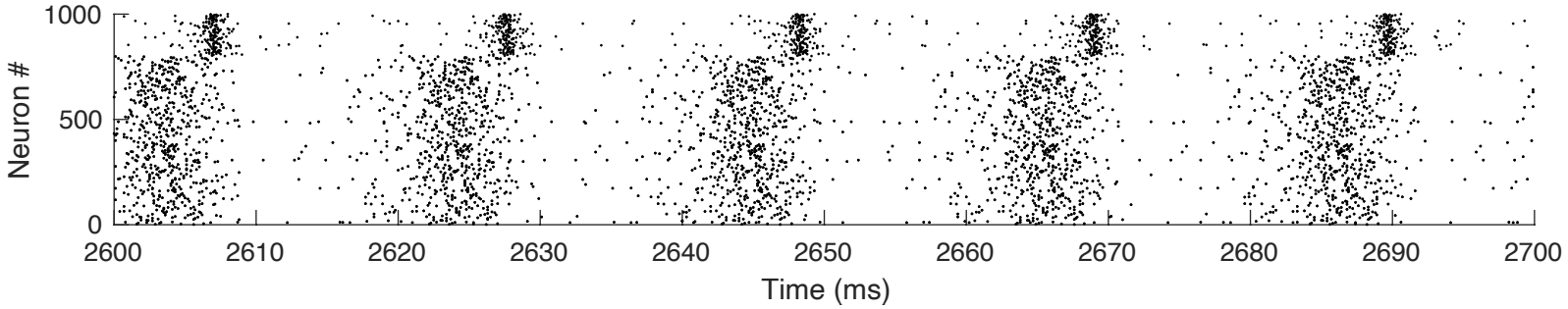


$$\frac{d}{dt}\theta(t) = G(\theta(t))$$



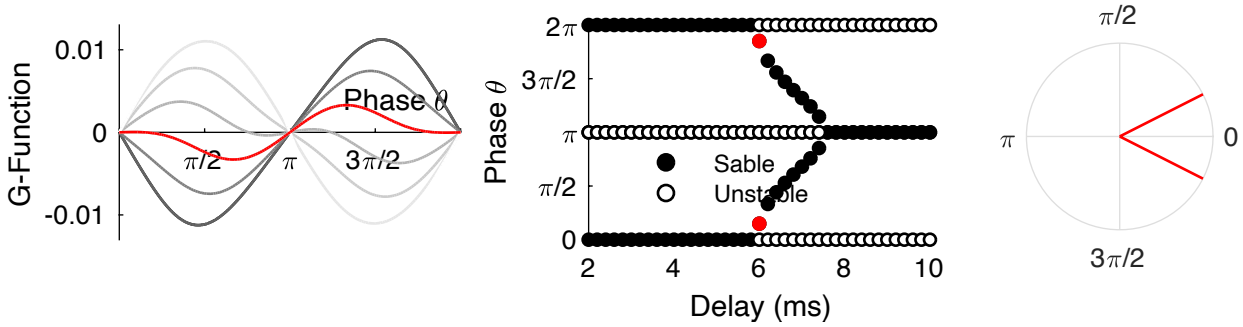


The **zeros** of the G-function determine the **locking modes**

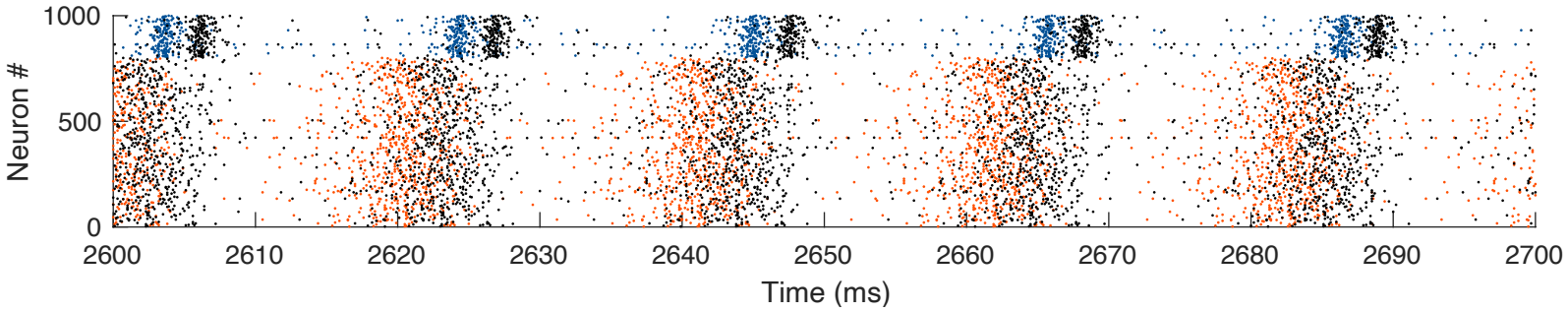


Symmetry Breaking

Effect of delay on the interaction function

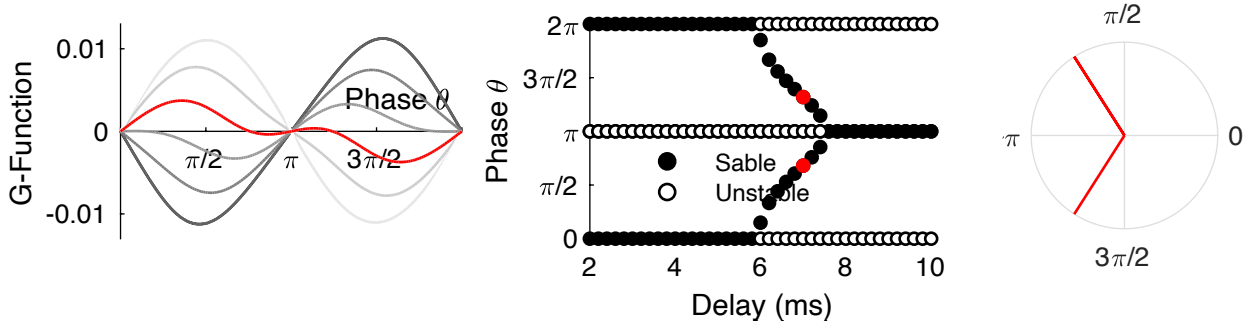


The **breaking of symmetry** induces a **variety of phase lags**

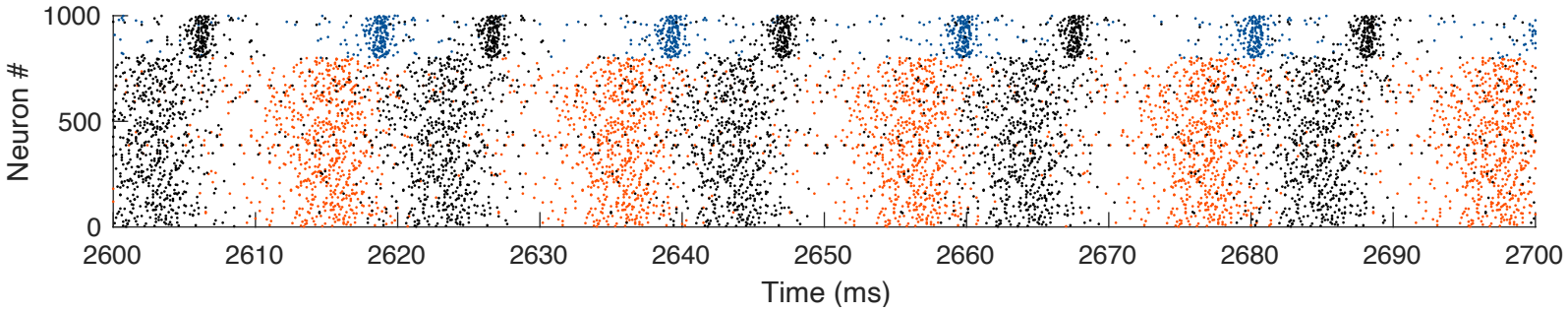


Symmetry Breaking

Effect of delay on the interaction function

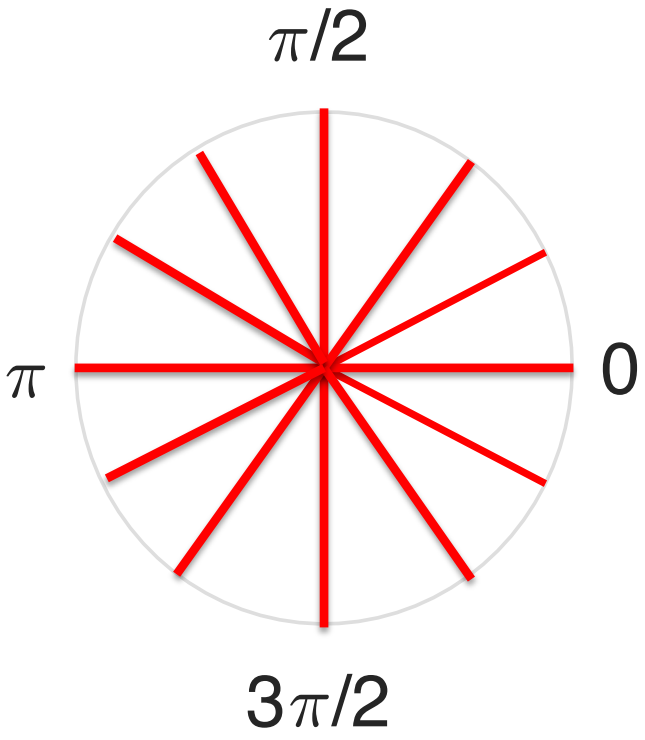


The **breaking of symmetry** induces a **variety of phase lags**



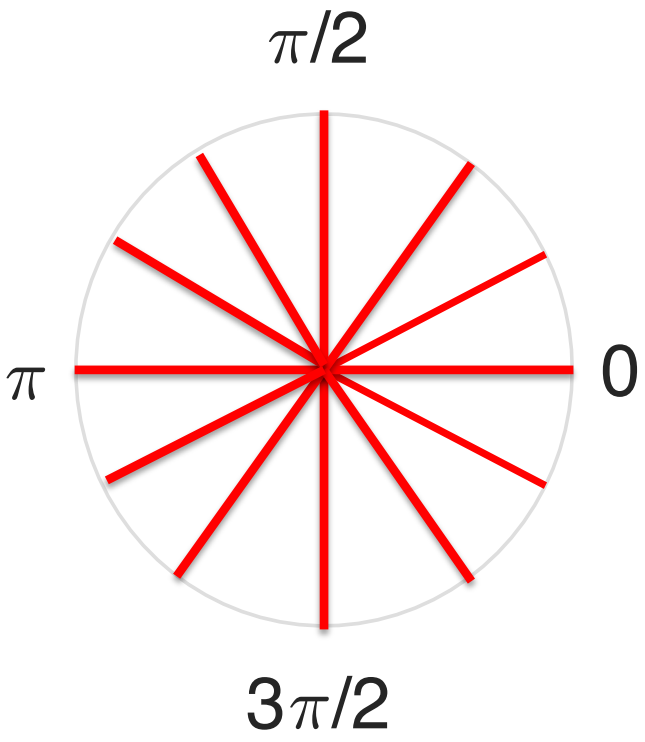
Arbitrary Phase Relationships

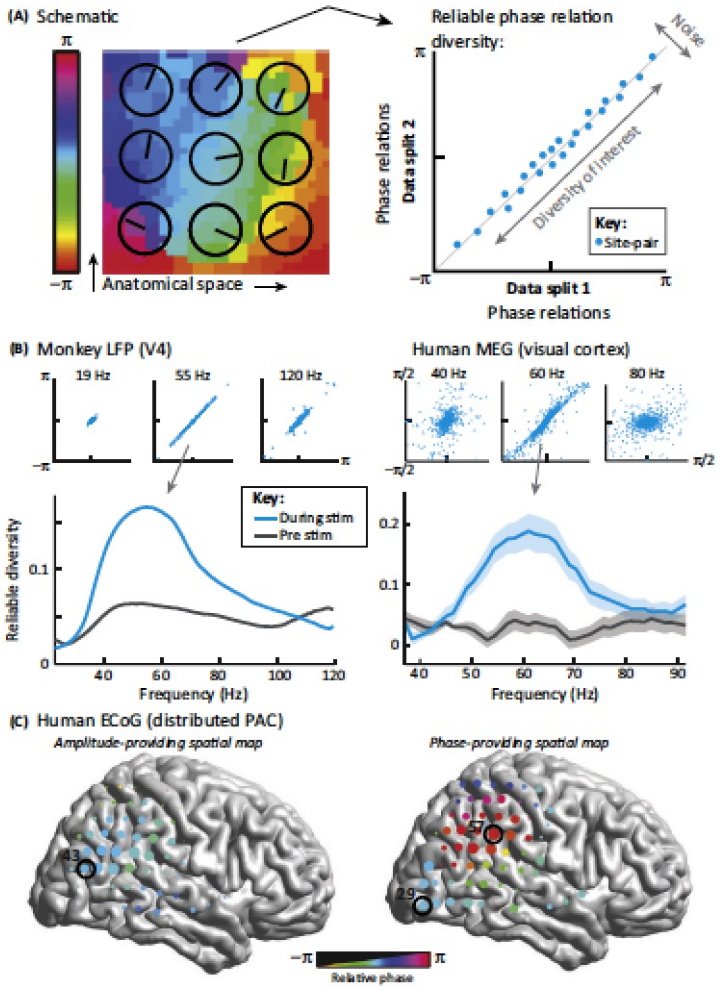
(as observed in vivo in the cortex)



Arbitrary Phase Relationships

(as observed in vivo in the cortex)

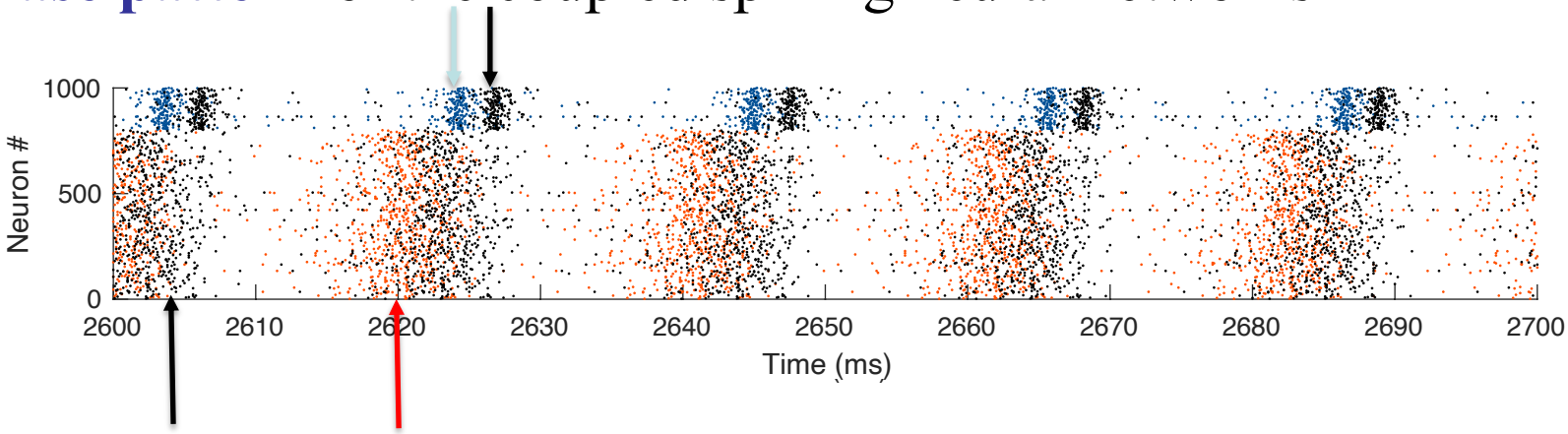




E Maris et al, (2016) Trends in neurosciences

Global PRC for the whole double-network

Phase pattern of the coupled spiking neural networks

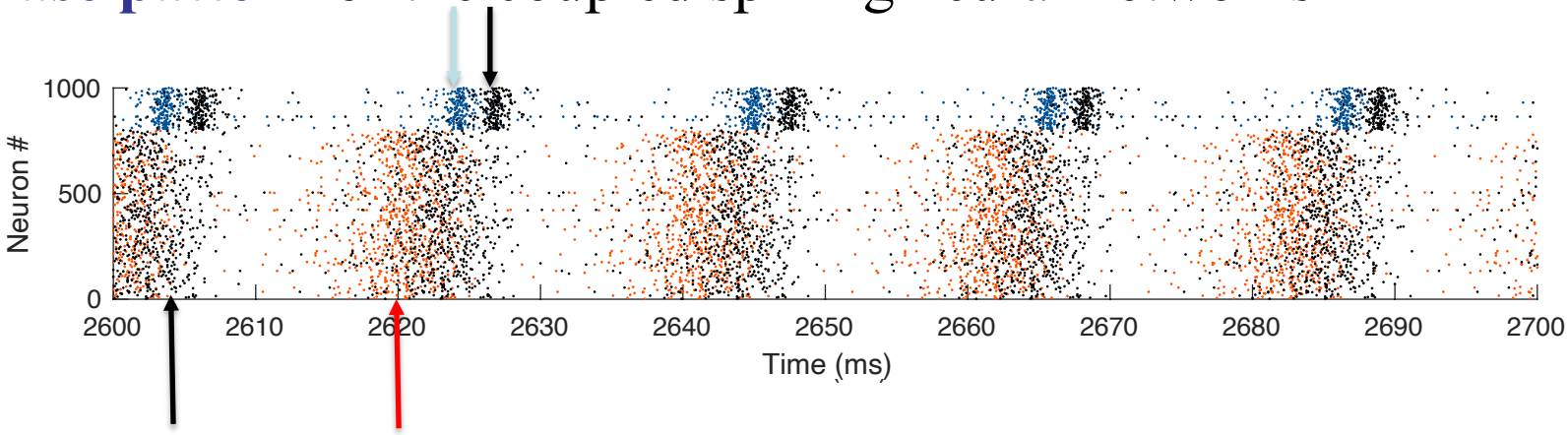


Will gPRC be different if we stimulate the leader or the follower?

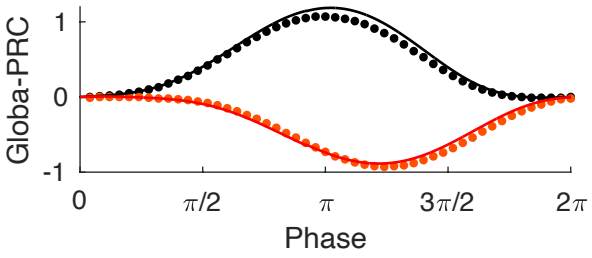
If yes, then we have dynamically induced a directionality in how inputs are processed

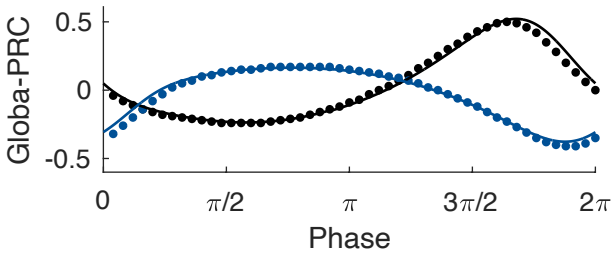
Causal Directionality

Phase pattern of the coupled spiking neural networks



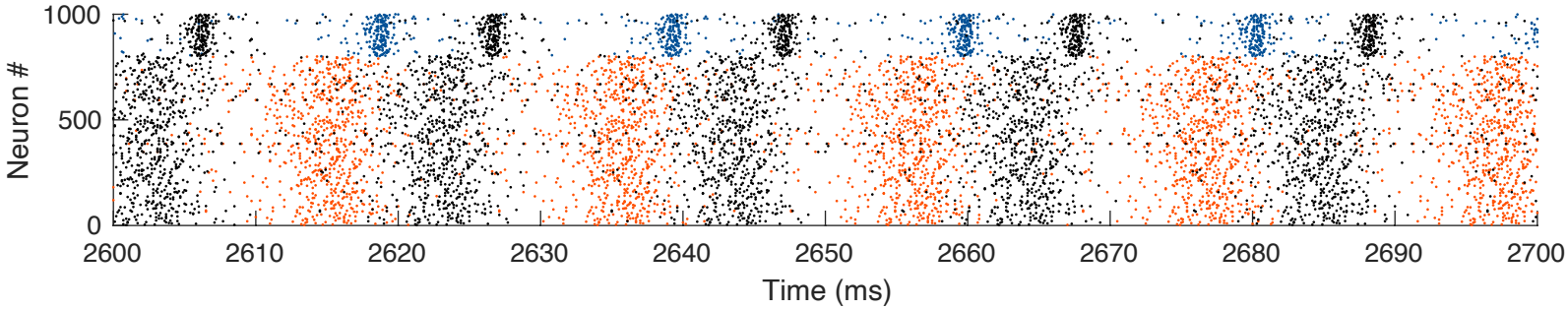
Global PRC



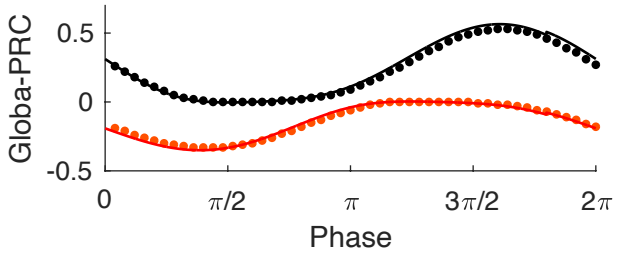


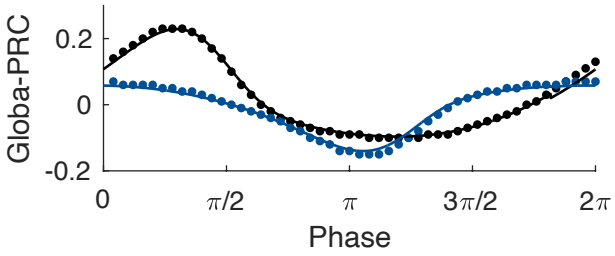
Causal Directionality

Phase pattern of the coupled spiking neural networks



Global PRC





What have we learned?

- Bifurcations as basis for oscillations (Hopfs, SNICs, Homoclinics)
  - Loss of stability at fixed points
  - Gain of stability by limit cycles
  - Apply to both single neurons and networks
- Can reduce complex models to phase-oscillators
  - Kuramoto phase oscillator, other phase models.
  - Phase Response Curve to characterize response
  - Can analyse synchrony of rhythms by weak coupling theory
- Network rhythms arise by interaction of excitation and inhibition
  - PING/ING etc….
  - Can use mathematical reduction to study large scale rhythms in networks
  - Can use phase-response curve formalism to analyse directionality of signals
- Open questions: Function? Rhythms without oscillators?

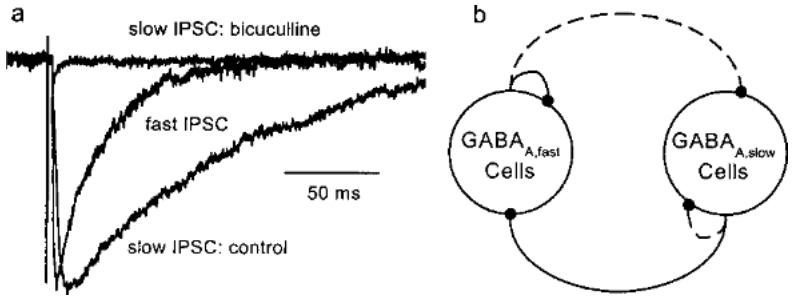
More fun with ERMs: theta-gamma

cross-frequency coupling

interaction between θ-rhytms (5-10 Hz) and γ-oscillations (20-100 Hz)

Which is the origin of this CFC ?

White, Banks, Pearce, and Kopell, PNAS 2000 : θ-γ CFC - interaction of two inhibitory populations



Two classes of interneurons identified in the Hippocampus (CA1)

- $\text{GABA}_{A,fast}$ $\tau_d \simeq 9$ ms
- $\text{GABA}_{A,slow}$ $\tau_d \simeq 50$ ms

two coupled neural masses A and B with exponential synapses:

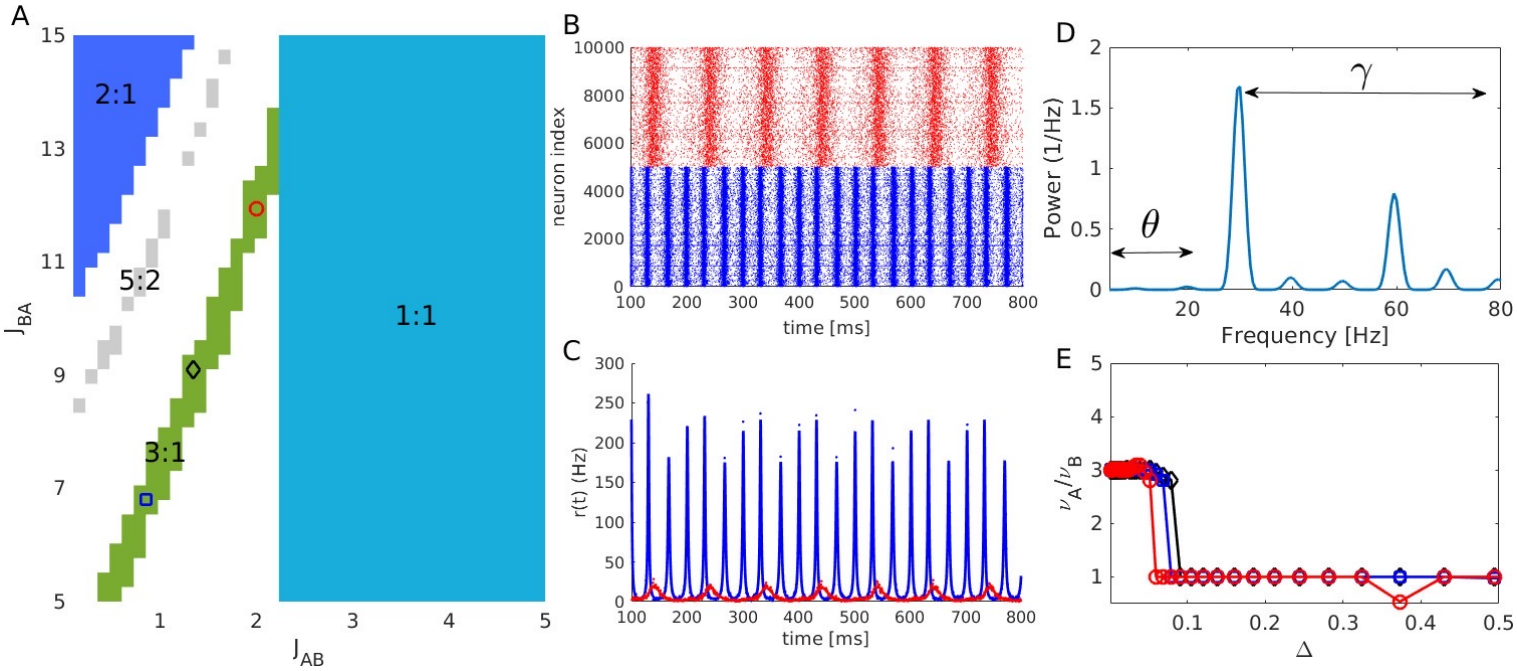
A slow inhibitory population A with self-coupling $J_{AA} = -2$,

- A fast inhibitory population B with self-coupling $J_{BB} = -11$
- Cross-couplings J_{AB} (A → B) and J_{BA} $(B \rightarrow A)$
-

Modified from talk by Torcini 2021

More fun with ERMs: theta-gamma

- Fast population: $\tau_{A,d} = 9$ ms - Slow population: $\tau_{B,d} = 50$ ms
- $3:1$ phase-locking mode: $\theta - \gamma$ coupling



$\Rightarrow$ For large values of J_{AB} and disorder Δ, the $3:1$ locked mode is lost

[Ceni, Olmi, AT, Angulo Garcia, Chaos (2020)]

Modified from talk by Torcini 2021

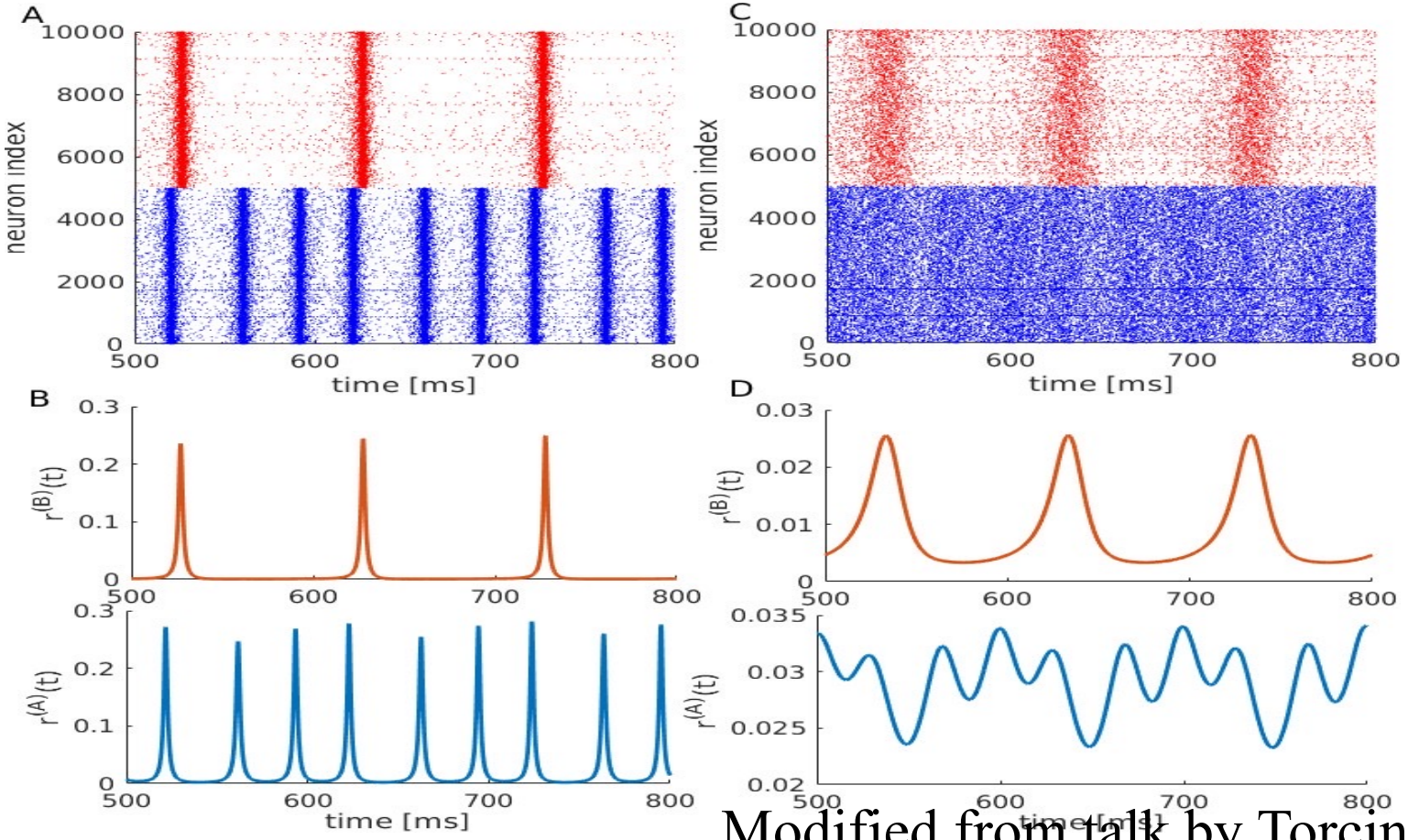
θ-γ Cross Frequency Coupling

$\Delta = 0.05$

Phase-phase CFC: slow population fast one are locked in phase

$\Delta = 0.2$

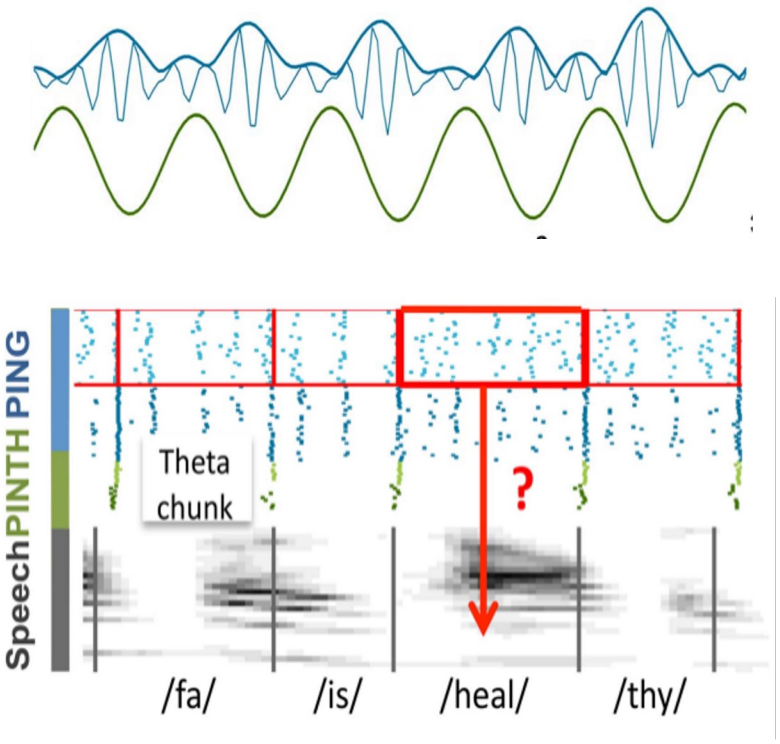
Phase-amplitude CFC: slow population modulates the fast amplitude



Modified from talk by Torcini 2021

Theta-gamma code for speech

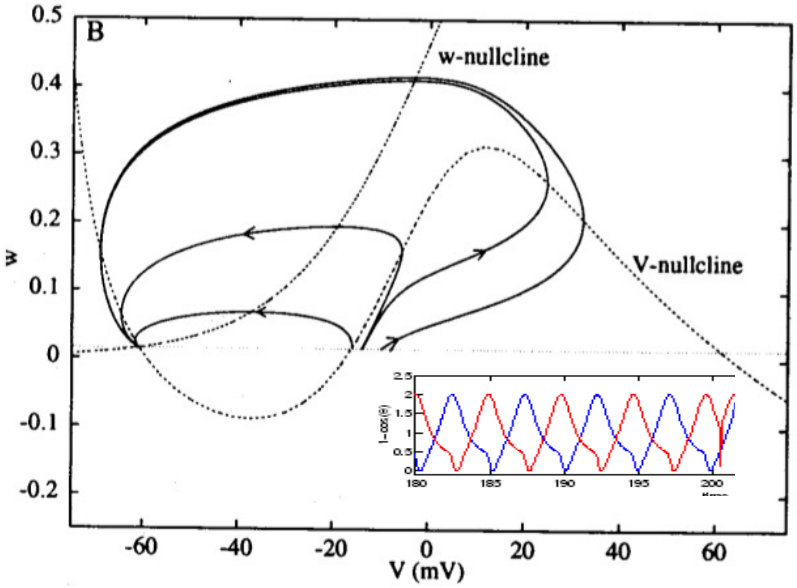
- Theta rhythm (4-8 Hz) chunks of syllables by tracking of speech envelope
- Gamma rhythm (>40 Hz) encodes phonemes and acoustic features
- Coupled theta-gamma performs packaging phonetic segments into syllabic units

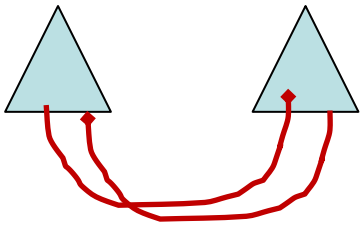


Hyafi, et al. Elife, 2015 [6] Hovsepyan, et al. Nature communications, 2020.

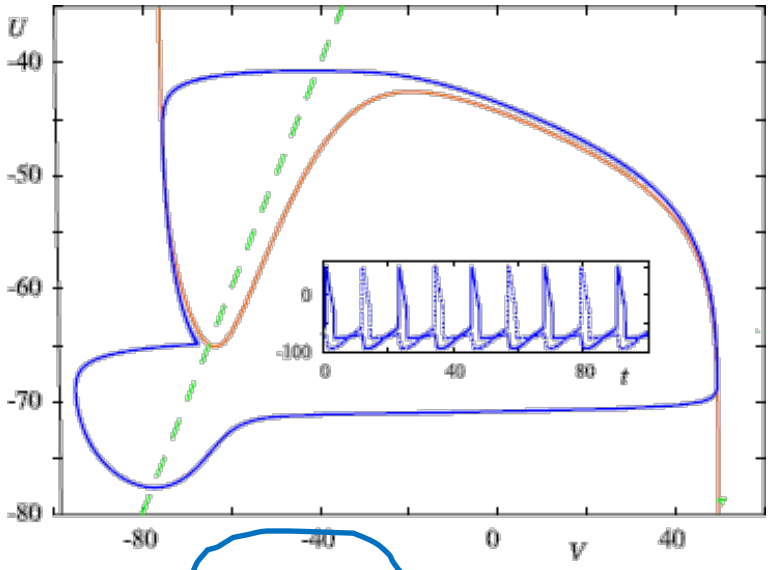
Sustained oscillations in excitable circuits

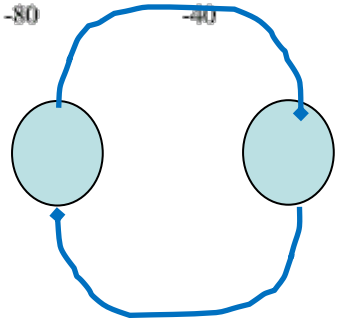
Mutual excitation





Mutual inhibition





Ashwin, P., Coombes, S. & Nicks, R. Mathematical Frameworks for Oscillatory Network Dynamics in Neuroscience. *J. Math. Neurosc.* **6**, 2 (2016). https://doi.org/10.1186/s13408-015-0033-6