Computational Modeling of Neuronal Systems

(Advanced Topics in Mathematical Physiology: G63.2855.001, G80.3042.004)

Thursday, 9:30-11:20am, WWH Rm 1314.
Prerequisites: familiarity with linear algebra, applied differential equations, statistics and probability.
Grad credit: 3 points

John Rinzel, rinzel@cns.nyu.edu, x83308, Courant Rm 521, CNS Rm 1005

This course will focus on computational modeling of neuronal systems, from cellular to system level, from models of physiological mechanisms to more abstract models of information encoding and decoding. We will address the characterization of neuronal responses or identification of neuronal computations; how they evolve dynamically; how they are implemented in neural ware; and how they are manifested in human/animal behaviors. Modeling will involve deterministic and stochastic differential equations, information theory, and Bayesian estimation and decision theory. Lecturers from NYU working groups will present foundational material as well as current research. Examples will be from various neural contexts, including visual and auditory systems, decision-making, motor control, and learning and memory.

Students will undertake a course project to simulate a neural system, or to compare a model to neural data. Abstract (Nov 15), written report and oral presentation (Dec 13). There may be occasional homework.

Computational Neuroscience

What "computations" are done by a neural system?

How are they done?

WHAT?

Feature detectors, eg visual system.
Coincidence detection for sound localization.
Memory storage.
Code: firing rate, spike timing.

Statistics of spike trains
Information theory
Decision theory
Descriptive models

HOW?

Molecular & biophysical mechanisms at cell & synaptic levels – firing properties, coupling.
Subcircuits.
System level.

Course Schedule.

* JR away

Introduction to mechanistic and descriptive modeling, encoding concepts.

Sept 6	Rinzel: "Nonlinear neuronal dynamics I: mechanisms of cellular excitability and oscillations"
Sept 17	Rinzel: "Nonlinear neuronal dynamics II: networks."
Sept 20*	Simoncelli: "Descriptive models of neural encoding: LNP cascade"
Sept 27*	Paninski: "Fitting LIF models to noisy spiking data"

Decision-making. Glimcher: Decisions, Uncertainty, and the Brain.

Oct 4	Glimcher: The Science of Neuroeconomics
Oct 11	Daw: "Valuation and/or reinforcement learning"
Oct 18	Rinzel: "Network models (XJ Wang et al) for decision making"

Vision.

Oct 25	Movshon: "Cortical processing of visual motion signals"
Nov 1	Rubin/Rinzel: "Dynamics of perceptual bistability"
Nov 8*	Cai/Rangan: "Large-scale model of cortical area V1."
Nov 15	Tranchina: "Synaptic depression: from stochastic to rate model; application to a model of cortical suppression."
Nov 22	no class (Thanksgiving)

Synchronization/correlation.

Nov 29	Pesaran: "Correlation between different brain areas"
Dec 6	Reyes: "Feedforward propagation in layered networks"

Nonlinear Dynamics of Neuronal Systems -- cellular level

John Rinzel

Computational Modeling of Neuronal Systems

Fall 2007

References on Nonlinear Dynamics

Rinzel & Ermentrout. Analysis of neural excitability and oscillations. In Koch & Segev (see below). Also as “Meth3” on www.pitt.edu/~phase/

Borisyuk A & Rinzel J. Understanding neuronal dynamics by geometrical dissection of minimal models. In, Chow et al, eds: Models and Methods in Neurophysics (Les Houches Summer School 2003), Elsevier, 2005: 19-72.

Izhikevich, EM: Dynamical Systems in Neuroscience. The Geometry of Excitability and Bursting. MIT Press, 2007.

Edelstein-Keshet, L. Mathematical Models in Biology. Random House, 1988.

Strogatz, S. Nonlinear Dynamics and Chaos. Addison-Wesley, 1994.

References on Modeling Neuronal System Dynamics

Koch, C. Biophysics of Computation, Oxford Univ Press, 1998. Esp. Chap 7

Koch & Segev (eds): Methods in Neuronal Modeling, MIT Press, 1998.

Wilson, HR. Spikes, Decisions and Actions, Oxford Univ Press, 1999.

Dynamics of Excitability and Oscillations

Cellular level
(spiking)

Network level
(firing rate)

Hodgkin-Huxley model

Wilson-Cowan model

Membrane currents

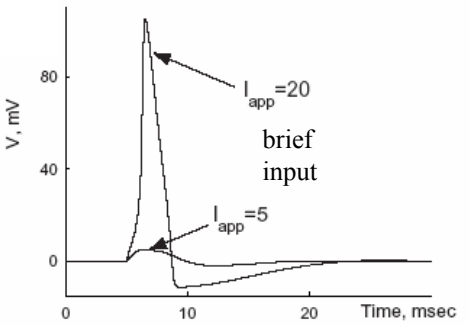
Activity functions

Activity dynamics in the phase plane

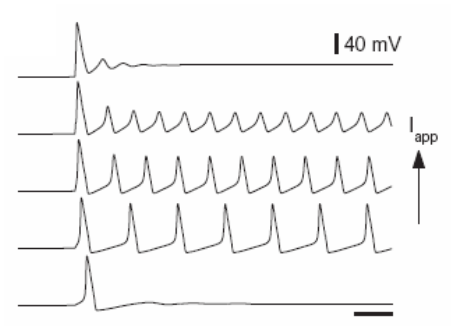
Response modes: Onset of repetitive activity (bifurcations)

Nonlinear Dynamical Response Properties

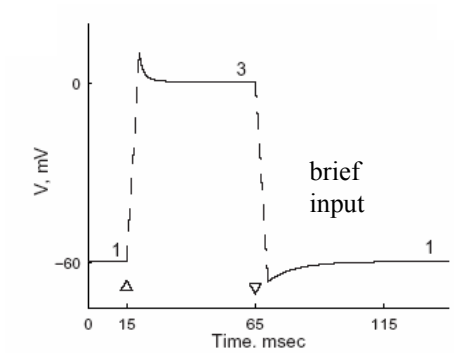
Cellular: "HH"



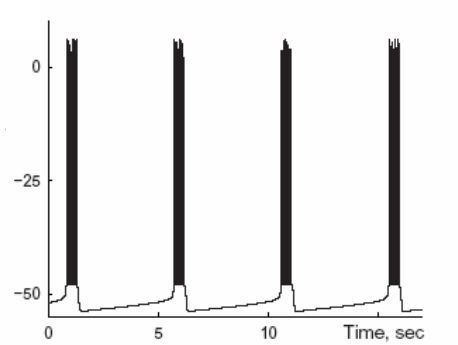
Excitability



Repetitive activity

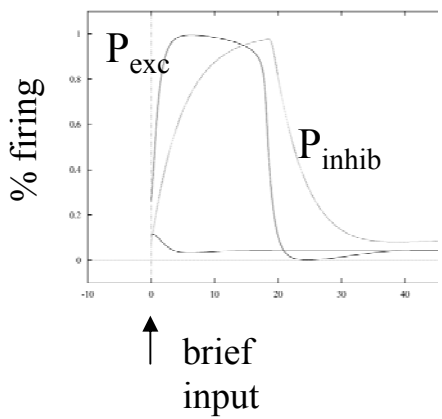


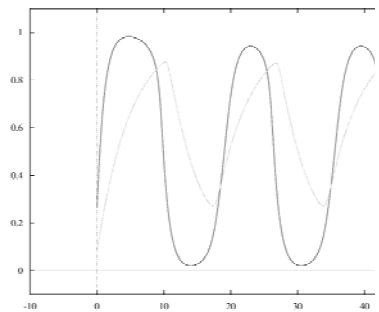
Bistability

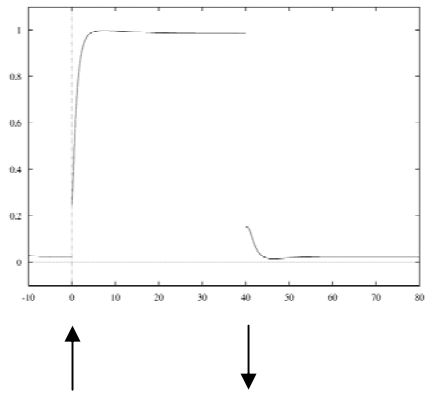


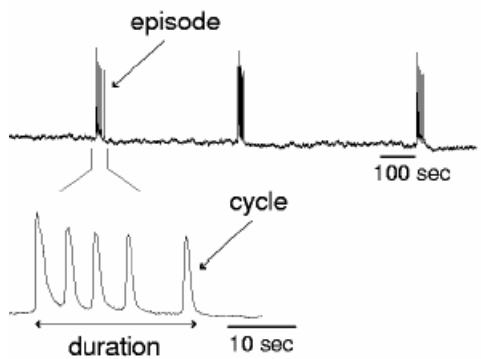
Bursting

Network: Wilson-Cowan
(Mean field)



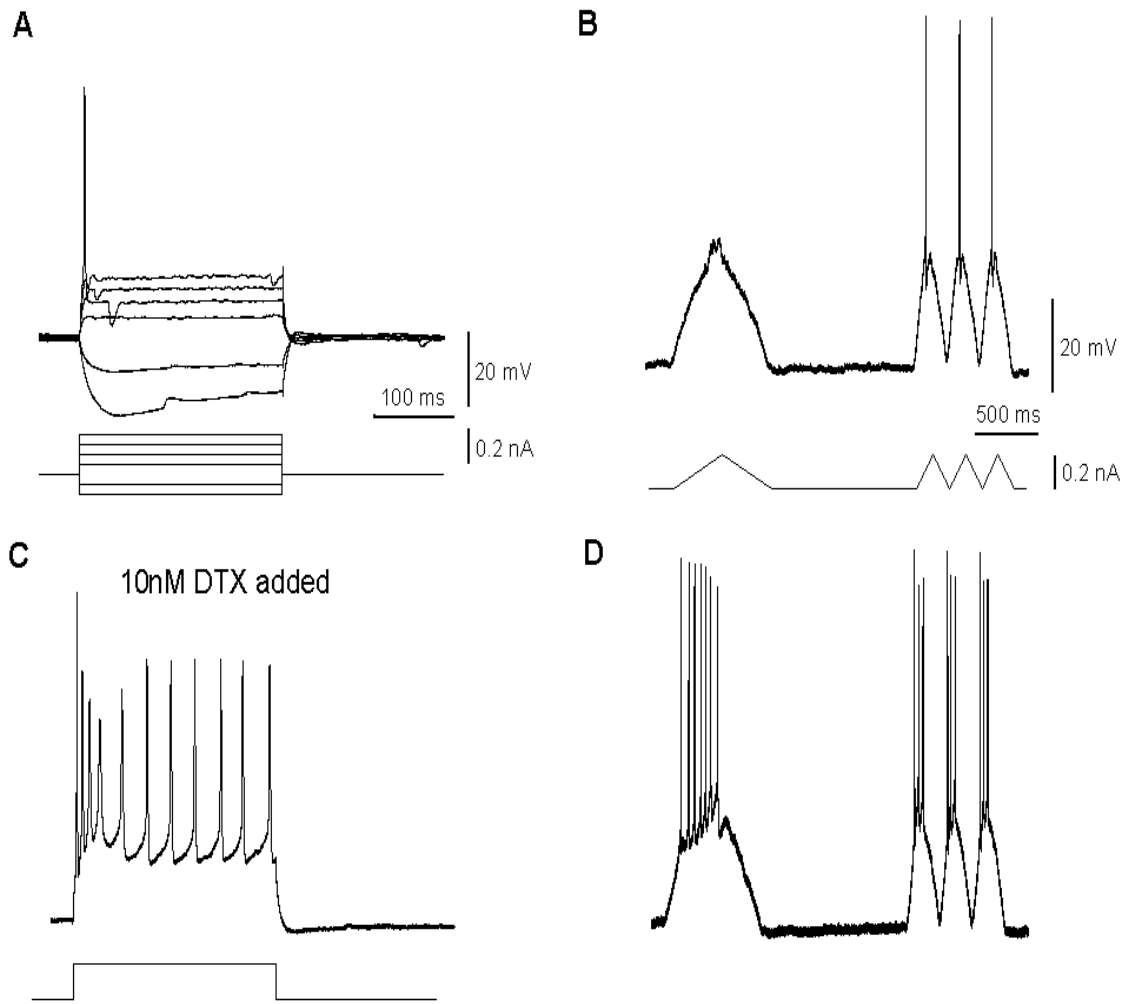






Auditory brain stem neurons fire phasically, not to slow inputs. Blocking I_{KLT} may convert to tonic.

J Neurosci, 2002



Take Home Messages

Excitability/Oscillations : fast autocatalysis + slower negative feedback

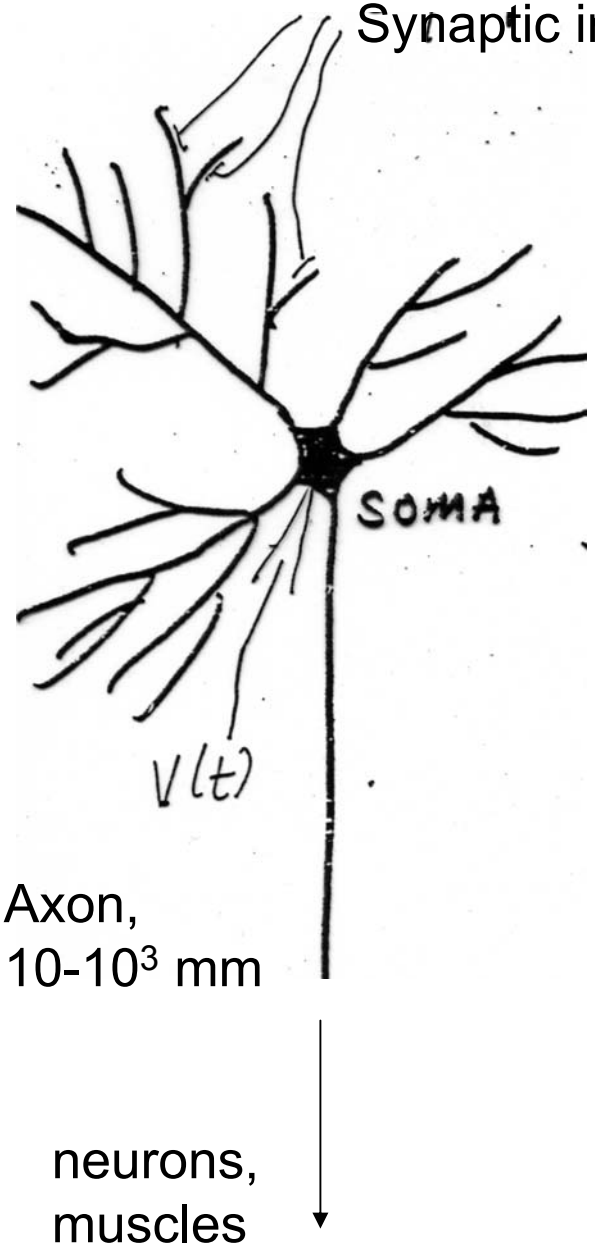
Value of reduced models

Time scales and dynamics

Phase space geometry

Different dynamic states – “Bifurcations”; concepts and methods are general.

XPP software:http://www.pitt.edu/~phase/ (Bard Ermentrout’s home page)



"Classical" neuron

Dendrites, 0.1 to 1 mm long

Signals: V_m ~ 100 mV membrane potential
O(msec)
ionic currents

Membrane with ion channels –
variable density over surface.

Dendrites – graded potentials,
linear in classical view

Axon – characteristic impulses, propagation

Electrical Activity of Cells

- V = V(x,t) , distribution within cell
  - uniform or not?, propagation?
- Coupling to other cells
- Nonlinearities
- Time scales

Current balance equation for membrane:

$$C_m \frac{\partial V}{\partial t} + I_{ion}(V) = \frac{d}{4R_i} \frac{\partial^2 V}{\partial x^2} + I_{app} + \text{coupling}$$

capacitive | channels | cable properties | other cells

Coupling: $\sum_j g_{c,j}(V_j - V)$ "electrical" - gap junctions

other cells

$\sum_j g_{syn,j}(V_j(t))\,(V_{syn} - V)$ chemical synapses

$I_{ion} = I_{ion}(V, \mathbf{W})$ generally nonlinear

$= \sum_k g_k(V, \mathbf{W})\,(V - V_k)$

channel types

$\partial \mathbf{W}/\partial t = \mathbf{G}(V, \mathbf{W})$

gating dynamics

J. Physiol. (1952) 117, 500–544

A QUANTITATIVE DESCRIPTION OF MEMBRANE CURRENT AND ITS APPLICATION TO CONDUCTION AND EXCITATION IN NERVE

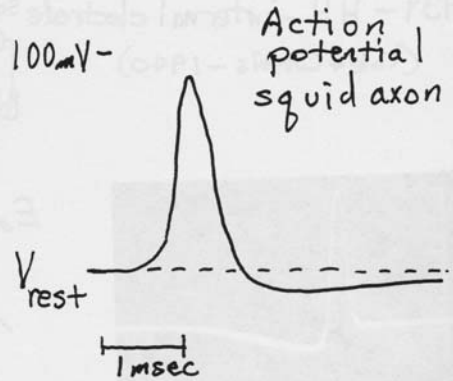
By A. L. HODGKIN and A. F. HUXLEY

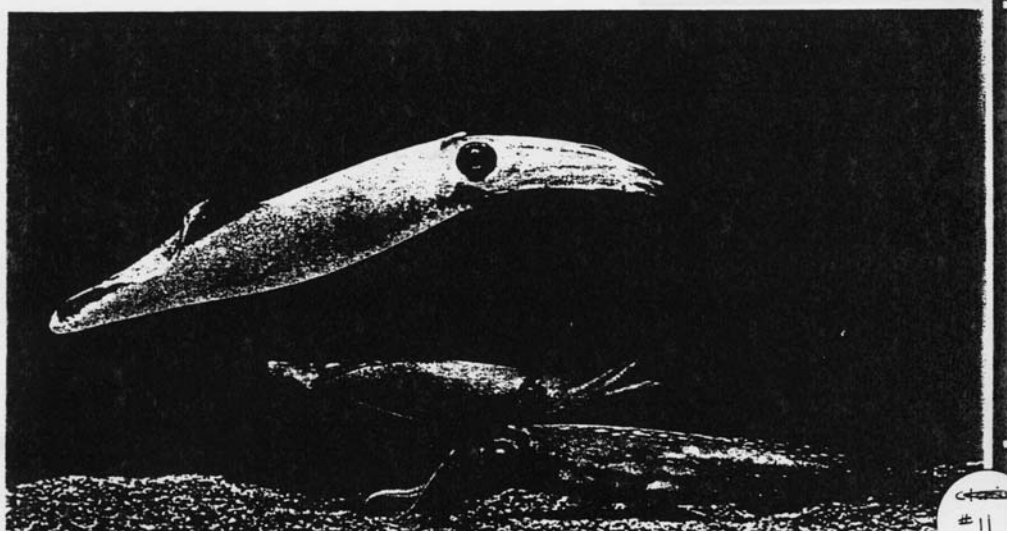
From the Physiological Laboratory, University of Cambridge

(*Received* 10 *March* 1952)

Alan Lloyd Hodgkin

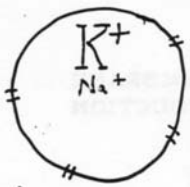
Andrew Fielding Huxley





Nobel Prize, 1959

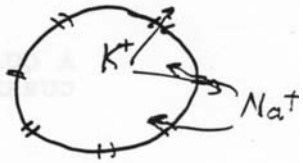
Classical Membrane Theory — Bernstein (1902)



rest

K^+
Na^+

"Breakdown"



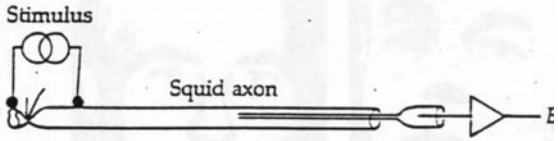
"activity"

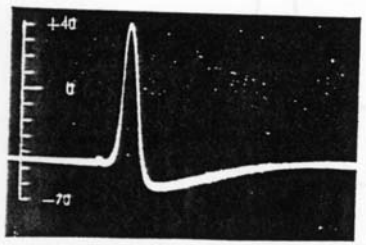
$E_m \uparrow 0$

$$E_m \doteq E_K = \frac{RT}{F} \ln \frac{[K^+]_{ext}}{[K^+]_{int}}$$

$$= -75\ mV$$

1939 – HH – internal electrode (Cole + Curtis – 1940)





E_m reverses — Bernstein disproved.

WW II – '39-'47

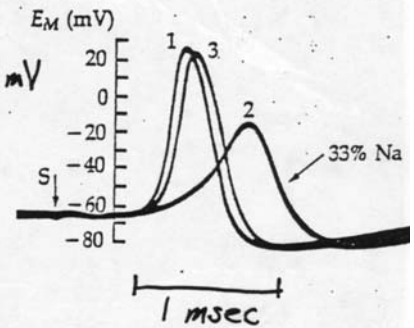
Sodium Hypothesis. Hodgkin + Katz (1949)

Selective change g_{Na} , g_K during a.p.

rest: $g_K \gg g_{Na} \Rightarrow E_m \doteq E_K$

a.p.: $g_{Na} \gg g_K \Rightarrow E_m \uparrow E_{Na} = +60\ mV$

$\therefore [Na^+]_{ext}$ affects a.p.



Replace E by V

Current Balance:

(no coupling, no cable properties, "steady state")

$$0 \approx g_K(V-V_K) + g_{Na}(V-V_{Na}) + g_L(V-V_L)$$

$$\rightarrow \quad V \approx \frac{g_K V_K + g_{Na} V_{Na} + g_L V_L}{g_K + g_{Na} + g_L}$$

HH Recipe:

V-clamp ➔ I_{ion} components

Predict I-clamp behavior?

$I_K(t)$ is monotonic; activation gate, n
$I_{Na}(t)$ is transient; activation, m and inactivation, h

e.g., $g_K(t) = I_K(t) /(V-V_K) = G_K\, n^4(t)$
with $V=V_{clamp}$
gating kinetics:
$dn/dt = \alpha(V)\,(1-n) - \beta(V)\, n$
$= (n_\infty(V) - n)/\tau_n(V)$
$n_\infty(V)$ increases with V.

mass action for "subunits" or HH-"particles"

$$\text{OFF } P \underset{\beta(V)}{\overset{\alpha(V)}{\rightleftharpoons}} P^* \text{ ON}$$

$I_{Na}(t) = G_{Na} m^3(t)\, h(t)\, (V-V_{Na})$

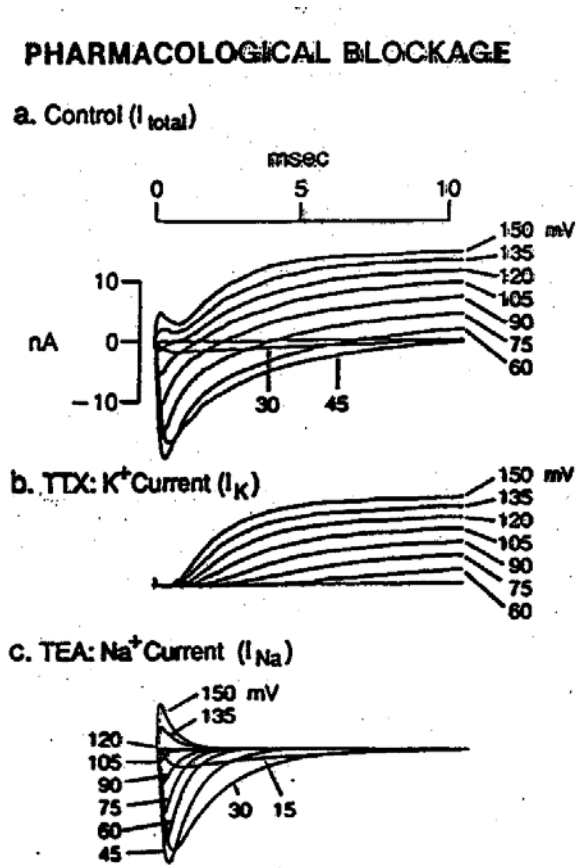


Figure 7. *B*, Separation of ionic currents by use of nerve poisons. *a*, Response in normal seawater; different amplitudes of voltage steps are indicated on the right (in mV). *b*, Response due to I_K when I_{Na} is blocked by tetrodotoxin (TTX). *c*, Response due to I_{Na} when I_K is blocked by tetraethylammonium (TEA). (From Hille, 1977).

ing into the cell) followed by an outward movement of positive current (see Figure 9; solid line).

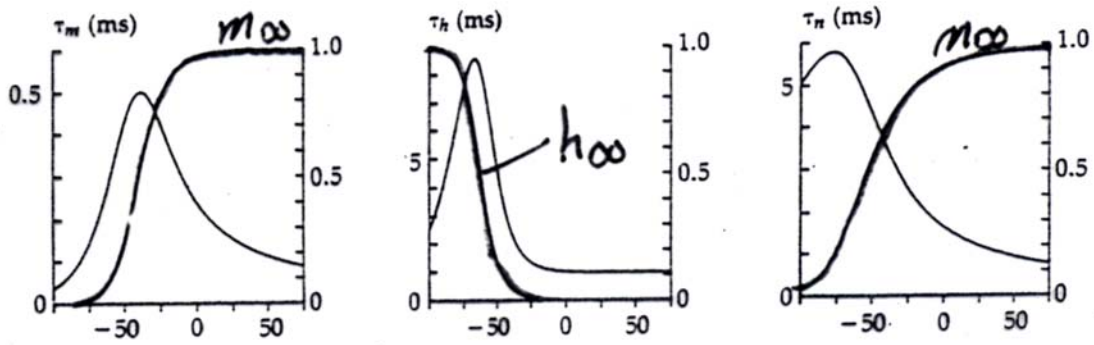
At this point, we need to define a bit of terminology that will be useful. In simple terms, ionic current through excitable membranes is controlled by two factors: (1) an ion-selective pore through which only certain ions can flow, and (2) a gate or gates that open(s) and close(s) the pore to allow ionic flux. The turning on of a current is known as the *activation* of the current and the opposite of activation is known as *deactivation*. These processes occur when an *activation gate* opens or closes. If a current turns on and then off despite a constant change in membrane potential, it is said to *inactivate*. The reverse of inactivation is *deinactivation*. Inactivation and

HH Equations

$$C_m\, dV/dt + G_{Na}\, m^3\, h\, (V-V_{Na}) + G_K\, n^4\, (V-V_K) + G_L\, (V-V_L) = I_{app}$$

space-clamped

$$dm/dt = \varphi\, [m_\infty(V)-m]/\tau_m(V)$$
$$dh/dt = \varphi\, [h_\infty(V) - h]/\tau_h(V)$$
$$dn/dt = \varphi\, [n_\infty(V) - n]/\tau_n(V)$$



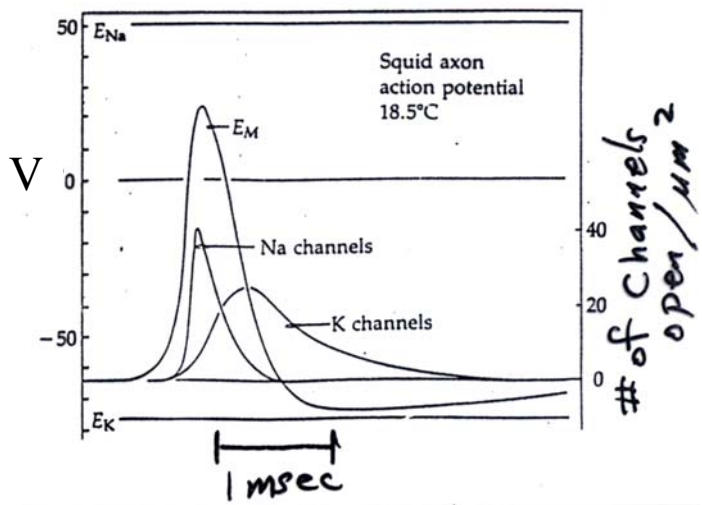
φ, temperature correction factor

$= Q_{10}$**[(temp-$temp_{ref}$)/10]

HH: $Q_{10}=3$

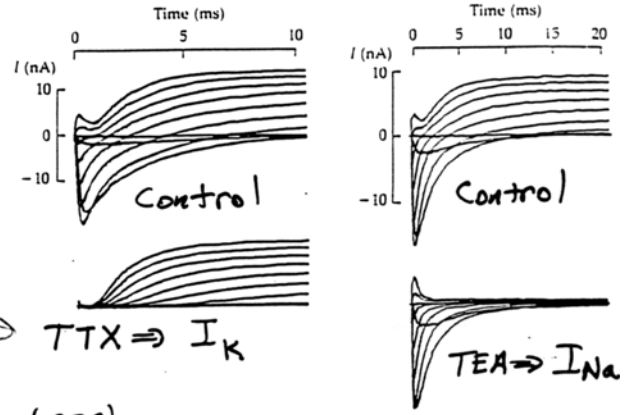
Reconstruct action potential

Time course
Velocity
Threshold
Refractory period
Ion fluxes
Repetitive firing?

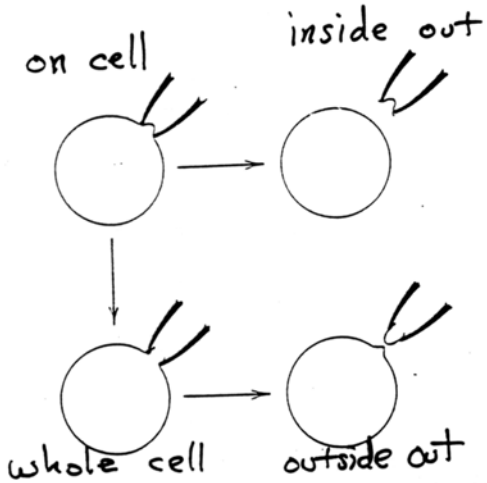


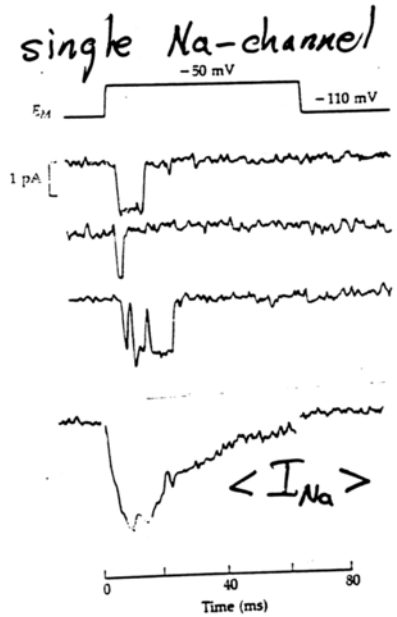
Biophysical Developments

Pharmacological Blockade



Patch Clamp – Neher (1979)





Other channels

I_{Ca} — T, L, N types

I_A — K^+ w/ inactivation

I_{K-Ca} — K^+ activated by V & by $[Ca^{2+}]_{int}$

HH model is a mean-field model that assumes an adequate density of channels.

1 μm^2 has about 100 Na^+ and K^+ channels.

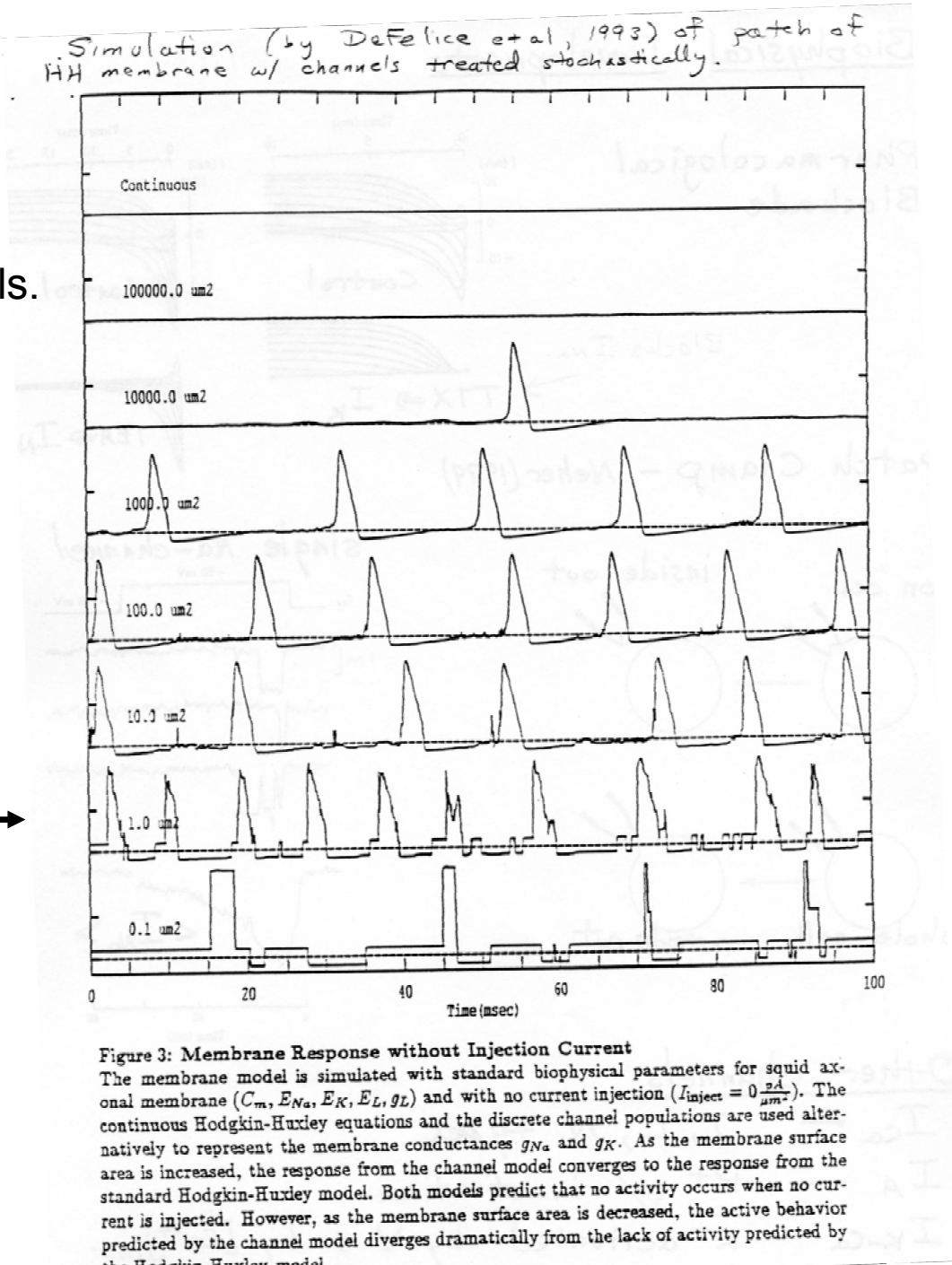


Figure 3: Membrane Response without Injection Current
The membrane model is simulated with standard biophysical parameters for squid axonal membrane ($C_m, E_{Na}, E_K, E_L, g_L$) and with no current injection ($I_{inject} = 0 \frac{pA}{\mu m^2}$). The continuous Hodgkin-Huxley equations and the discrete channel populations are used alternatively to represent the membrane conductances g_{Na} and g_K. As the membrane surface area is increased, the response from the channel model converges to the response from the standard Hodgkin-Huxley model. Both models predict that no activity occurs when no current is injected. However, as the membrane surface area is decreased, the active behavior predicted by the channel model diverges dramatically from the lack of activity predicted by the Hodgkin-Huxley model.

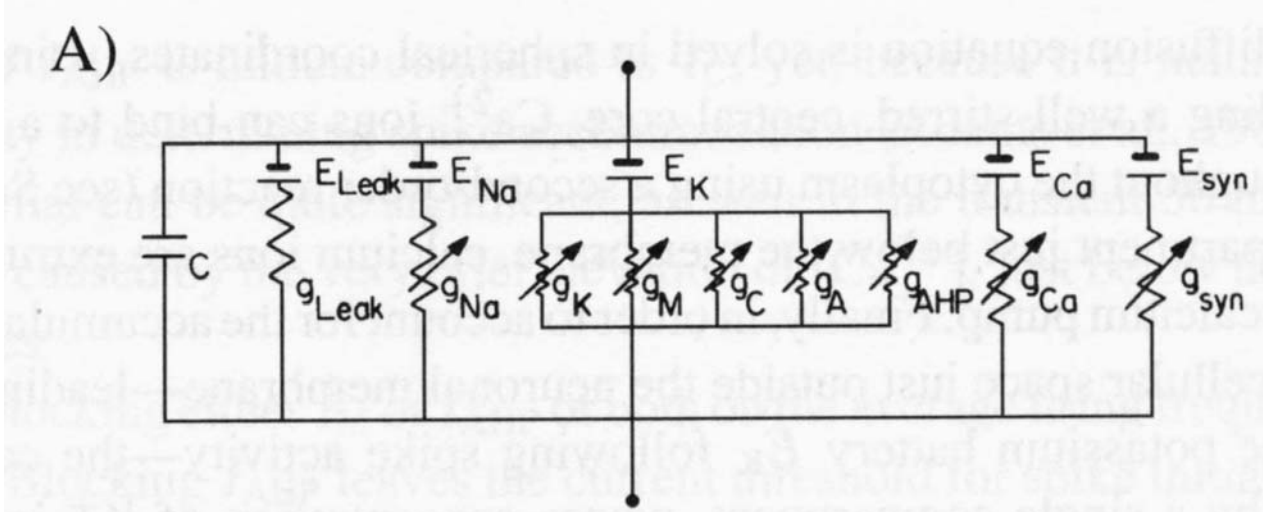
Bullfrog sympathetic Ganglion “B” cell

Cell is “compact”, electrically … but not for diffusion Ca^{2+}

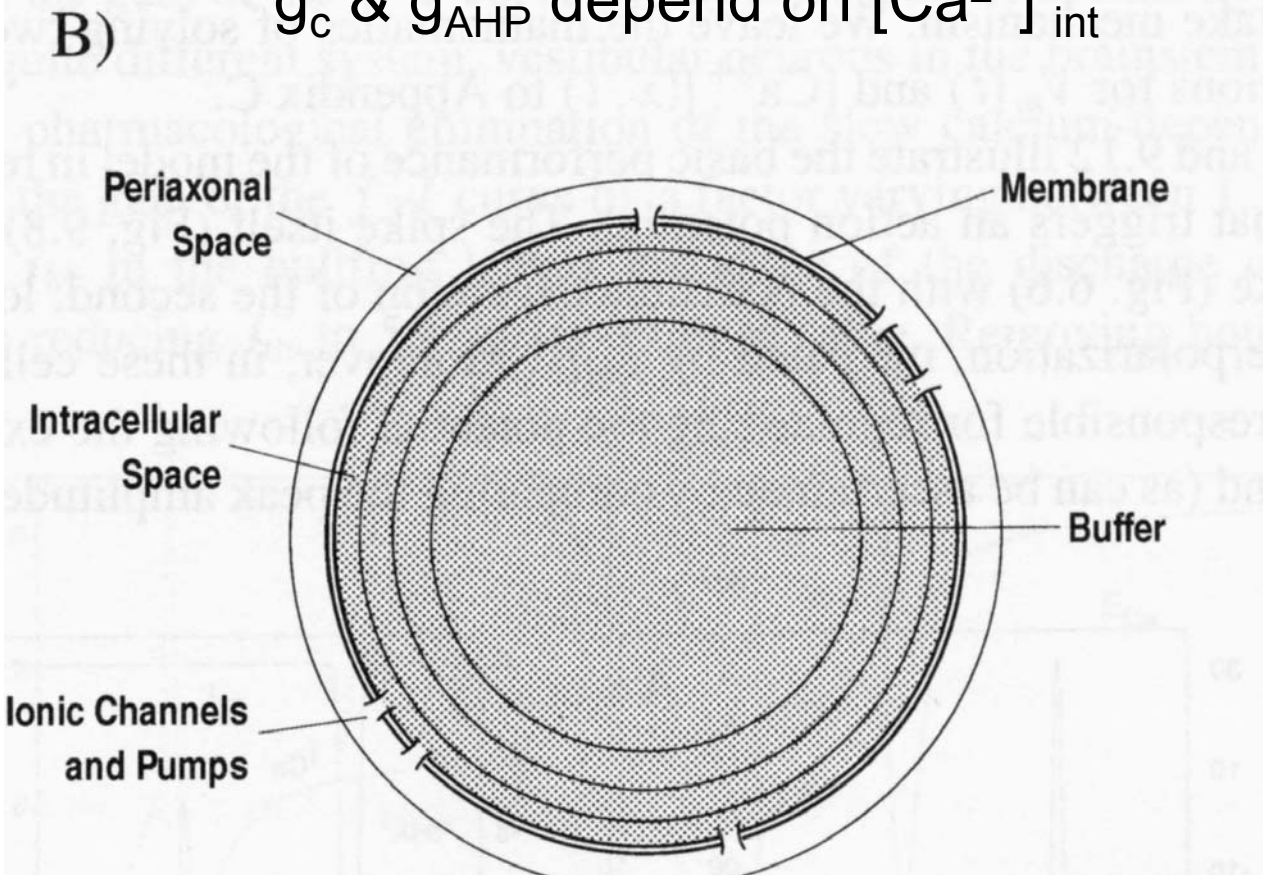
MODEL:

“HH” circuit
+ $[Ca^{2+}]_{int}$
+ $[K^{+}]_{ext}$

Yamada, Koch, Adams ‘89

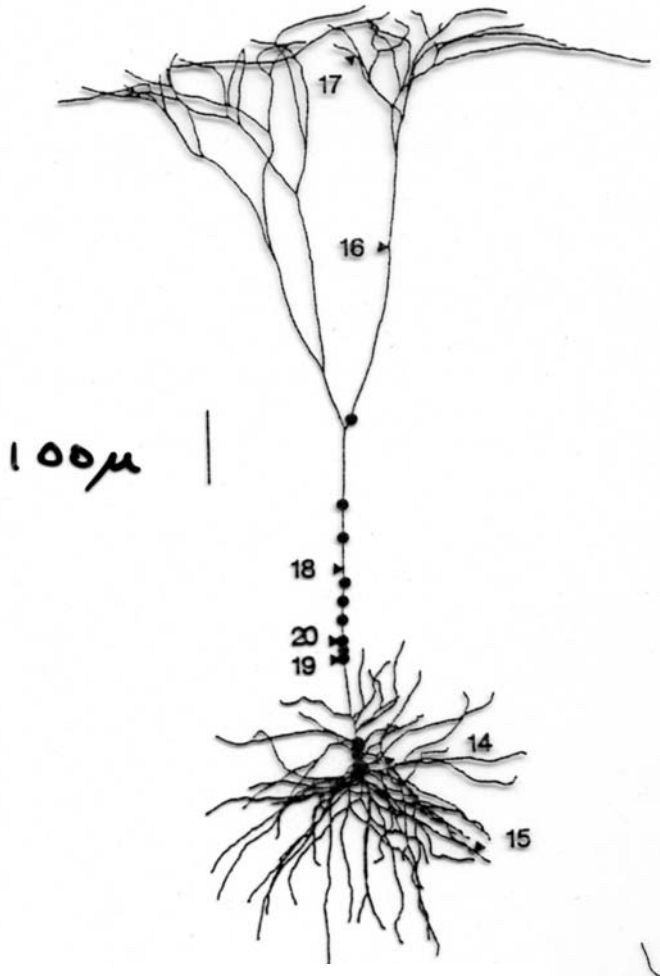


g_c & g_{AHP} depend on $[Ca^{2+}]_{int}$



Cortical Pyramidal Neuron

Complex dendritic branching
Nonuniformly distributed channels



Koch, Douglas,
Wehmeier '90

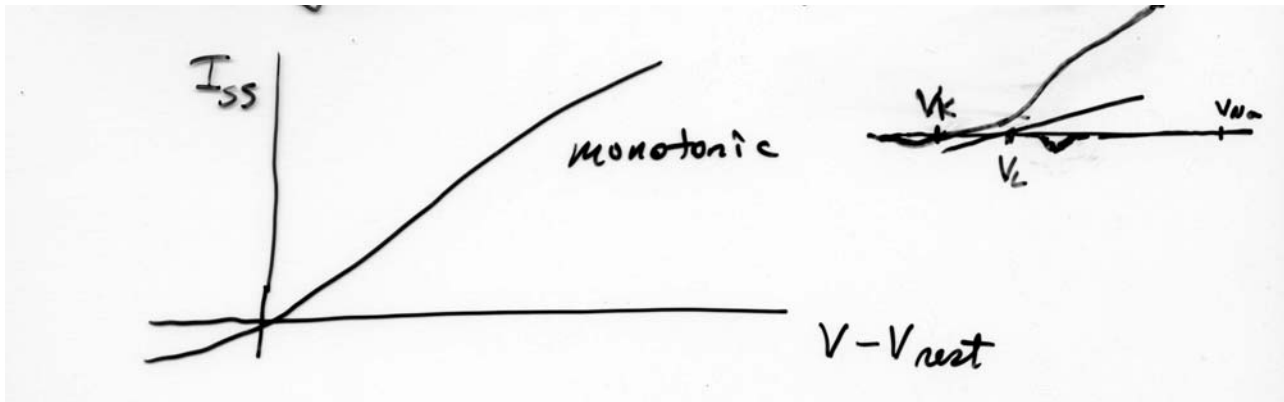
Pyramidal Neuron with axonal tree

Schwark & Jones, '89

HH action potential – biophysical time scales.

I-V relations: $I_{SS}(V)$ steady state $I_{inst}(V)$ "instantaneous"

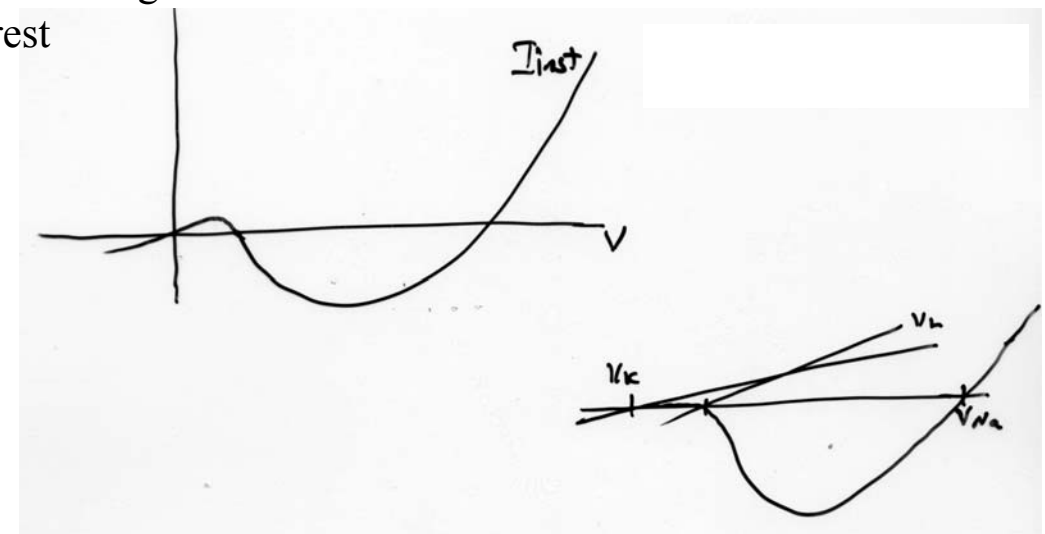
HH: $I_{SS}(V) = G_{Na}\, m_\infty^3(V)\, h_\infty(V)\, (V-V_{Na}) + G_K\, n_\infty^4(V)\, (V-V_K) + G_L\, (V-V_L)$



$I_{inst}(V) = G_{Na}\, m_\infty^3(V)\, h\, (V-V_{Na}) + G_K\, n\, (V-V_K) + G_L\, (V-V_L)$

fast

slow, fixed at holding values e.g., rest

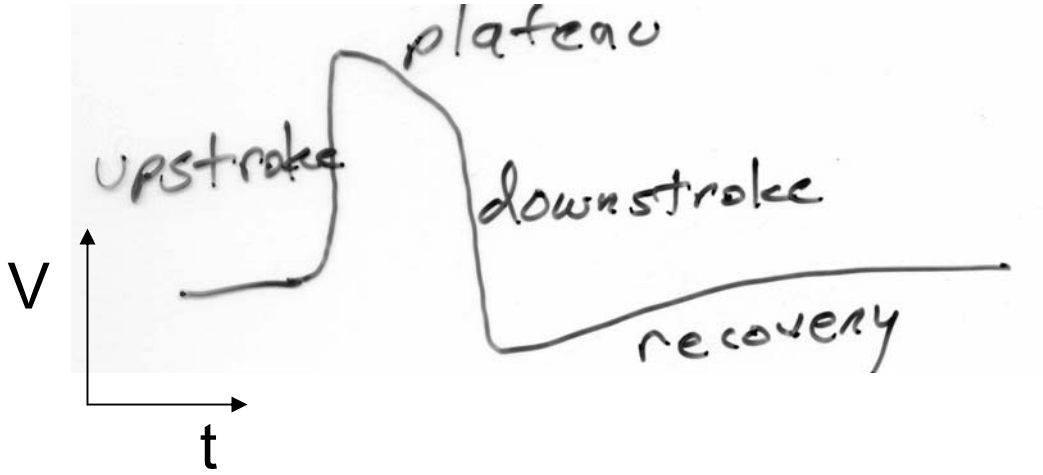


Dissection of HH Action Potential

Fast/Slow Analysis - based on time scale differences

h, n are slow relative to V,m

Idealize AP to 4 phases



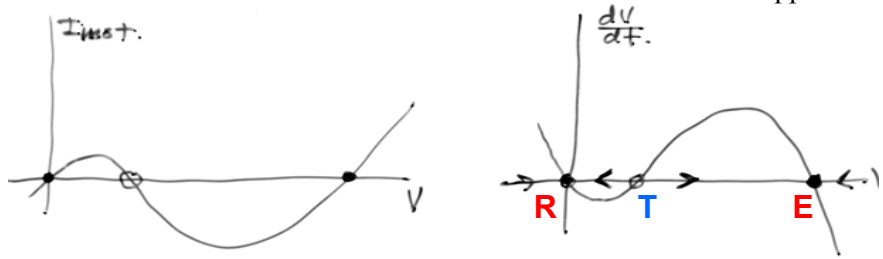
h,n – constant during upstroke and downstroke

$$C\,dV/dt = -\,I_{inst}(V, m_\infty(V), h_R, n_R) + I_{app}$$

Upstroke…

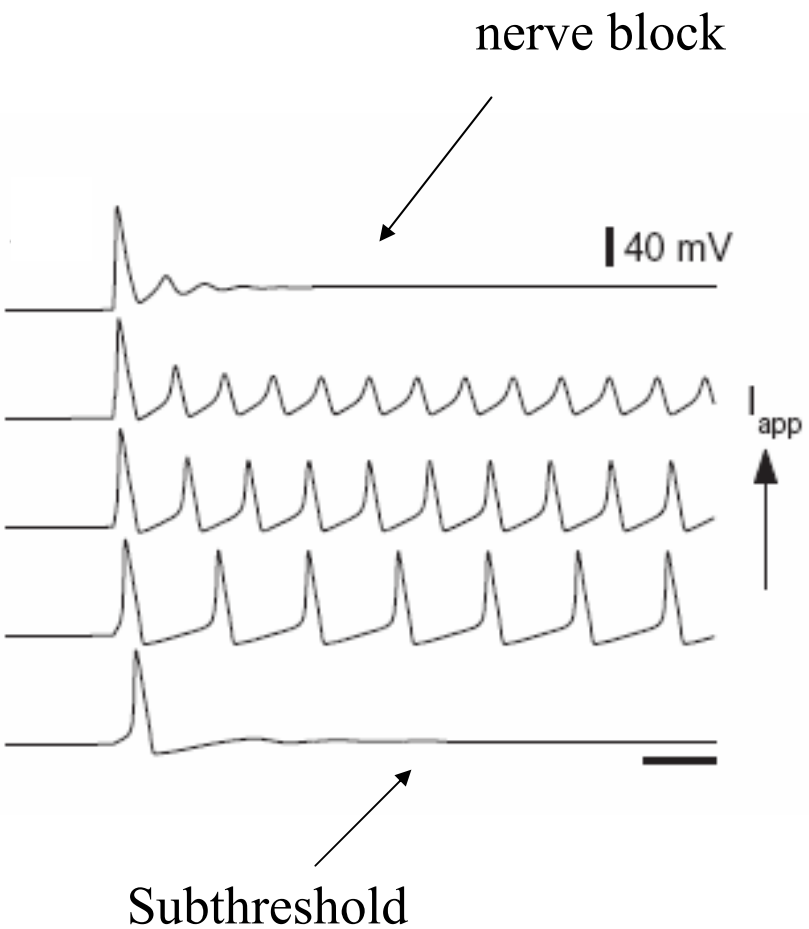
R and E – stable

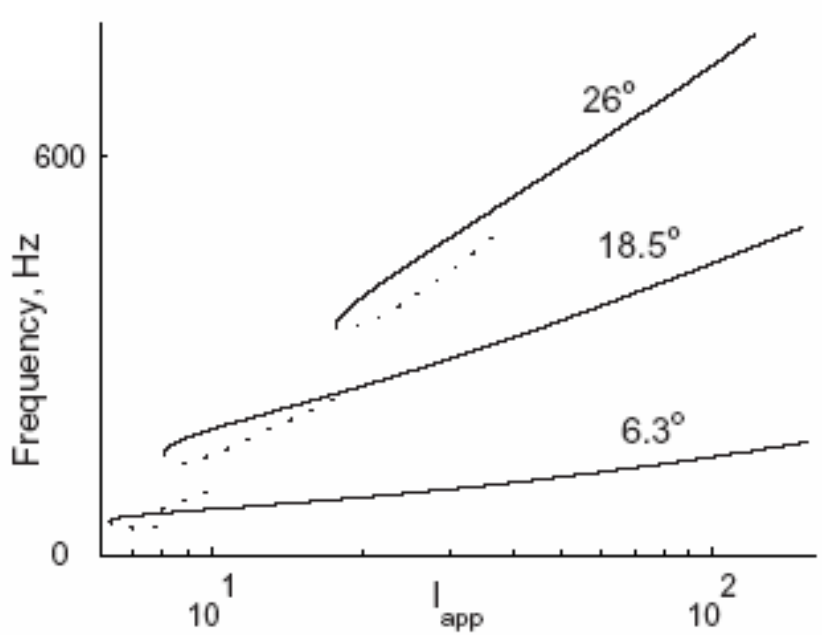
T - unstable



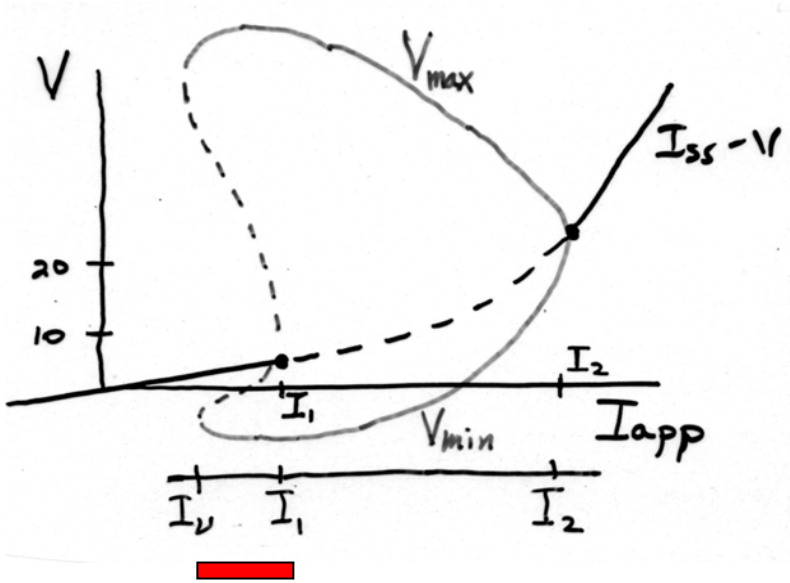
Repetitive Firing, HH model and others

Response to current step





Repetitive firing in HH and squid axon -- bistability near onset

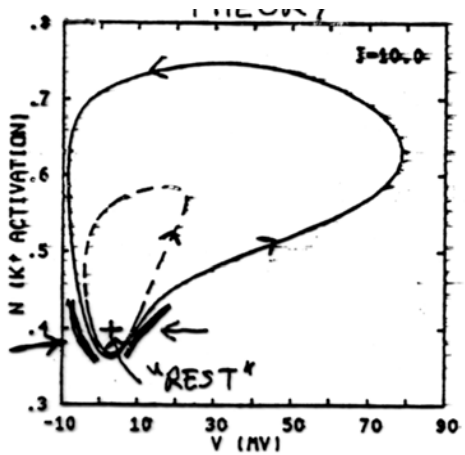


Interval of bistability

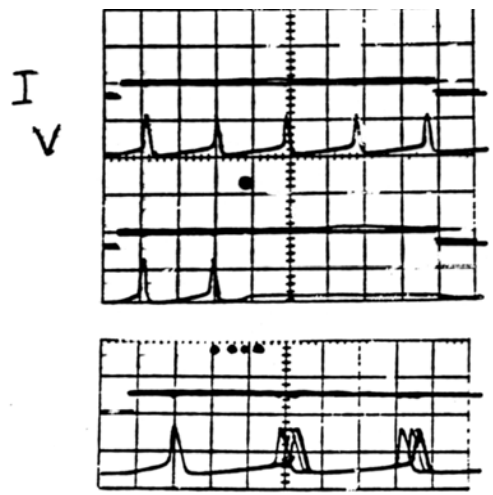
Rinzel & Miller, '80

Linear stability: eigenvalues of 4x4 matrix. For reduced model w/ $m=m_\infty(V)$: stability if $\partial I_{inst}/\partial V + C_m/\tau_n > 0$.

HH eqns



Squid axon



Guttman, Lewis & Rinzel, '80

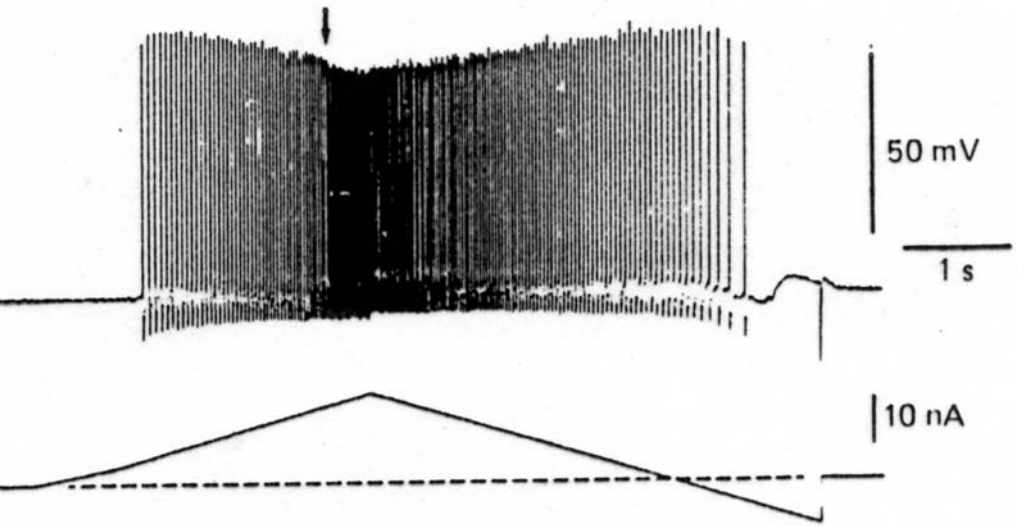
Bistability in motoneurons

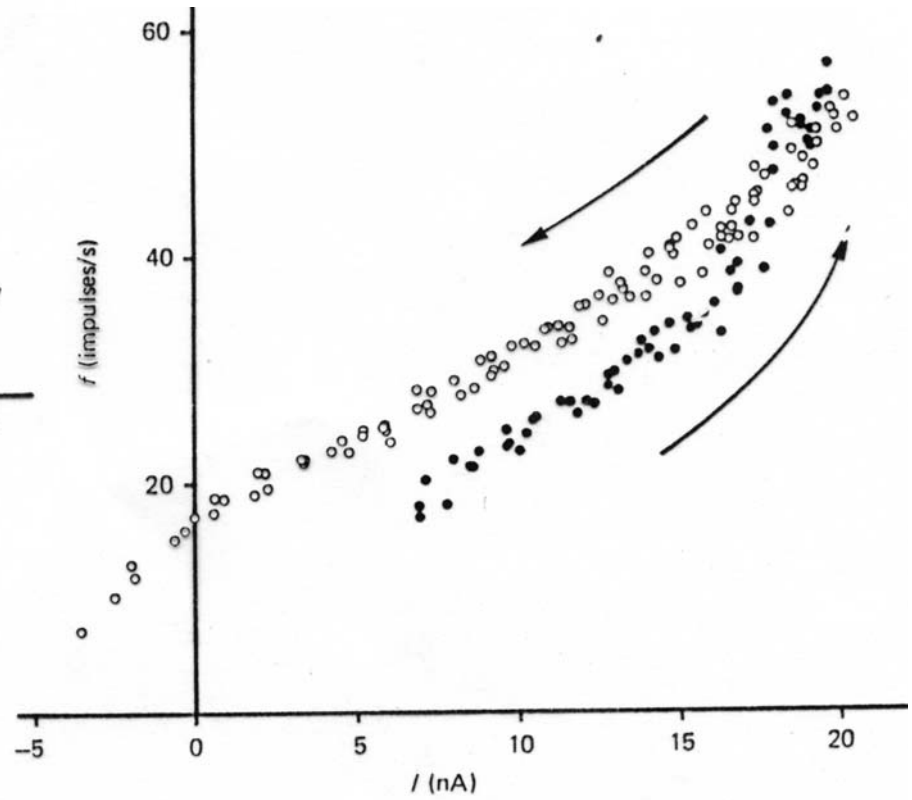
Journal of Physiology (1988), **405**, *pp.* 345–367
With 10 text-figures
Printed in Great Britain

BISTABILITY OF α-MOTONEURONES IN THE DECEREBRATE CAT A
IN THE ACUTE SPINAL CAT AFTER INTRAVENOUS 5-HYDROXYTRYPTOPHAN

BY JØRN HOUNSGAARD, HANS HULTBORN*, BO JESPERSEN AND OLE KIEHN

From the Department of Neurophysiology, The Panum Institute, University o Copenhagen, Blegdamsvej 3C, DK-2200 Copenhagen N, Denmark

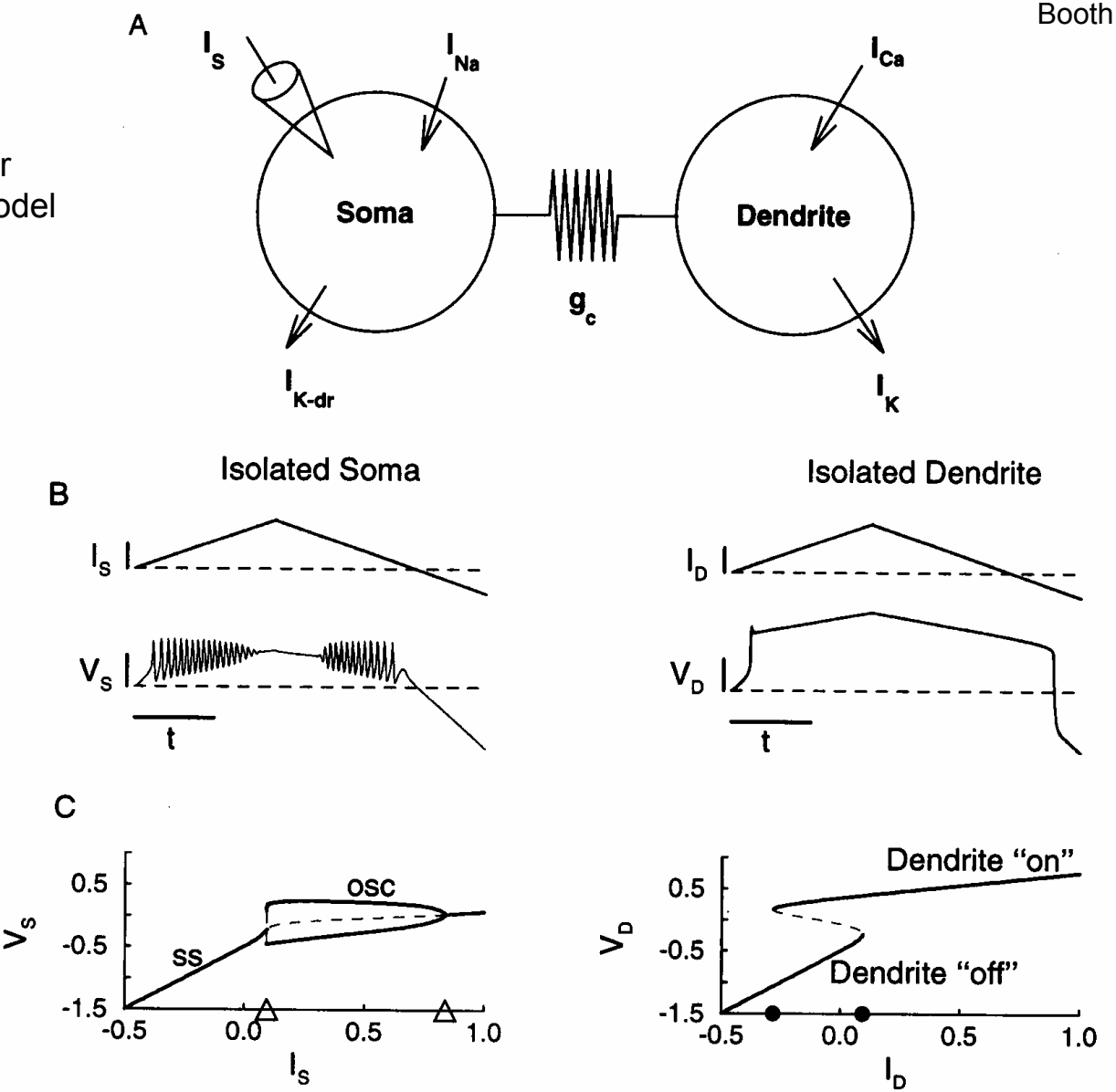




2-compartment model; plateau generator in dendrite

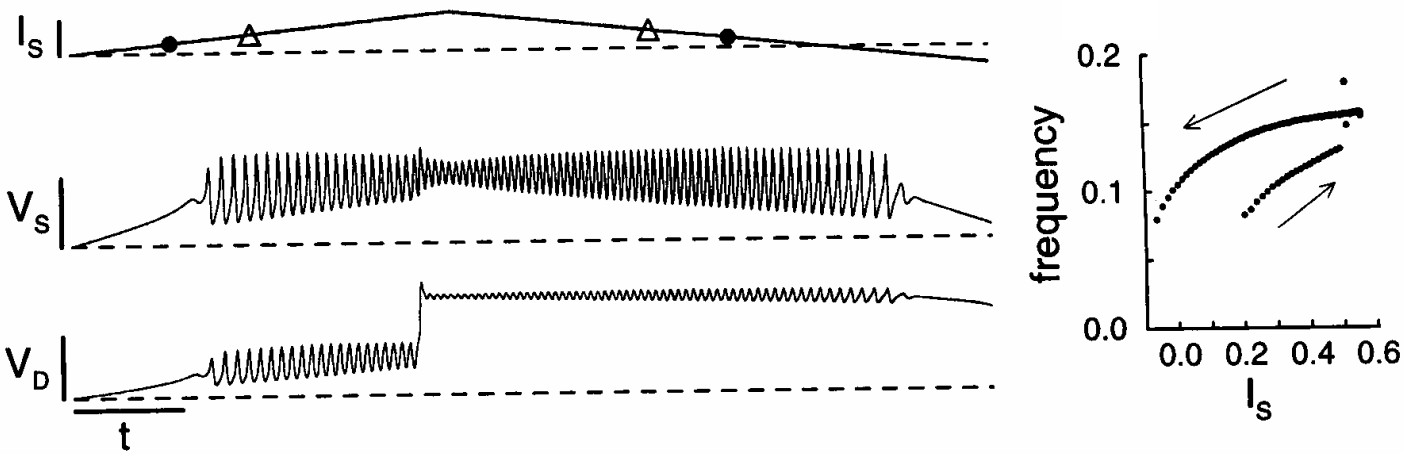
Booth & Rinzel, '95

Morris-Lecar membrane model

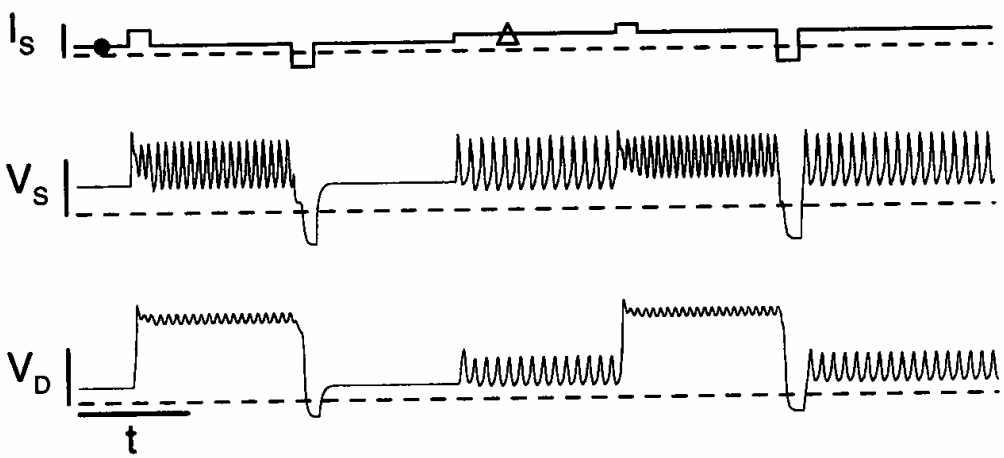


Bistability in 2-compt model

Response to up/down ramp; hysteresis



Switching between states



Two-variable Model ➔ Phase Plane Analysis

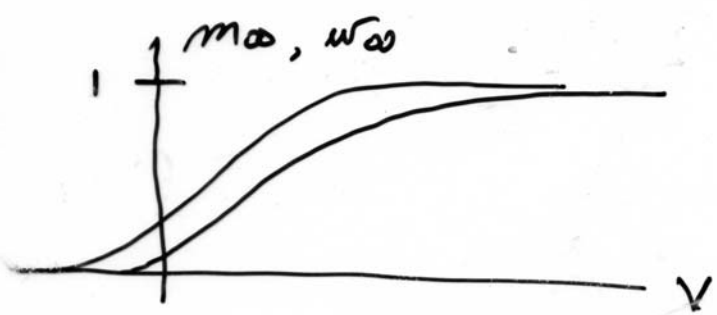
I_{Ca} – fast, non-inactivating
I_K -- “delayed” rectifier, like HH’s I_K

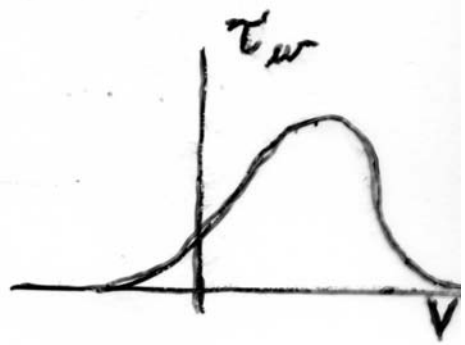
Morris & Lecar, ’81 – barnacle musclel

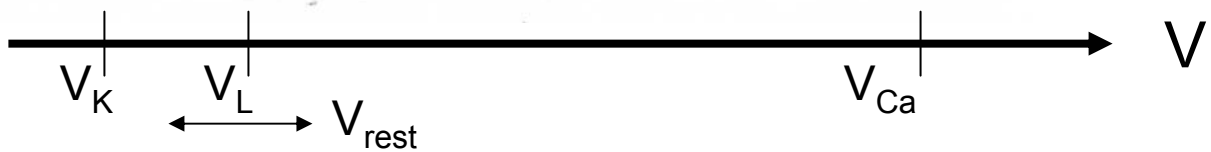
$$C\frac{dV}{dt} = -\bar{g}_{Ca}\, m_\infty(V)(V-V_{Ca}) - \bar{g}_K\, w\,(V-V_K) - g_L(V-V_L) + I$$

$$\frac{dw}{dt} = \phi\,\frac{w_\infty(V)-w}{\tau_w(V)}$$

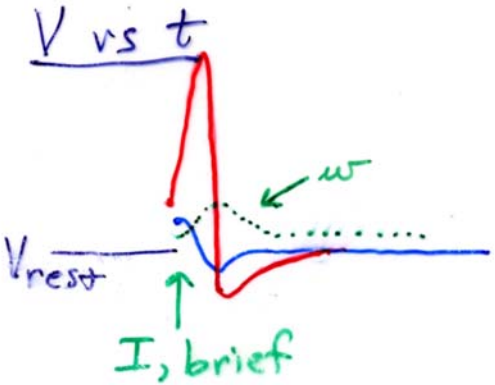
← negative feedback: slow

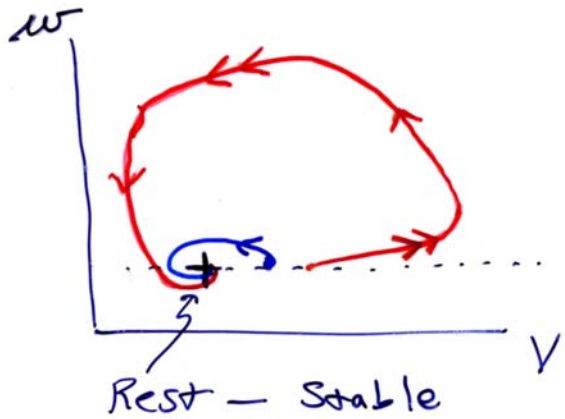


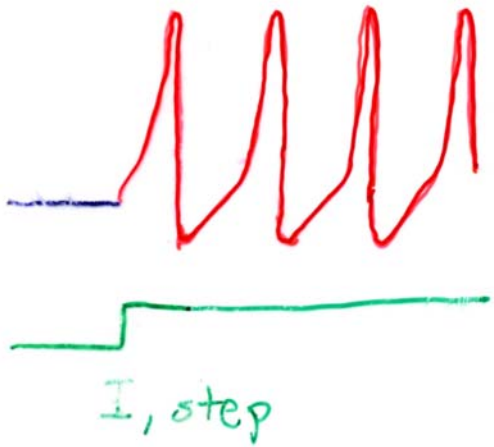


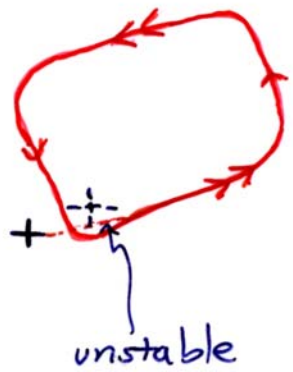


Phase Plane & Attractors









Effect of Perturbations

P.P. Analysis

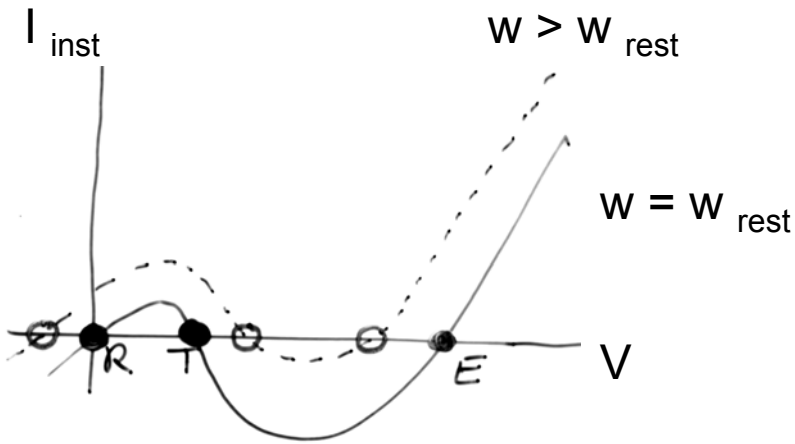
Get the Nullclines

$dV/dt = - I_{inst}(V,w) + I_{app}$

$dw/dt = \varphi [w_\infty(V) - w] / \tau_w(V)$

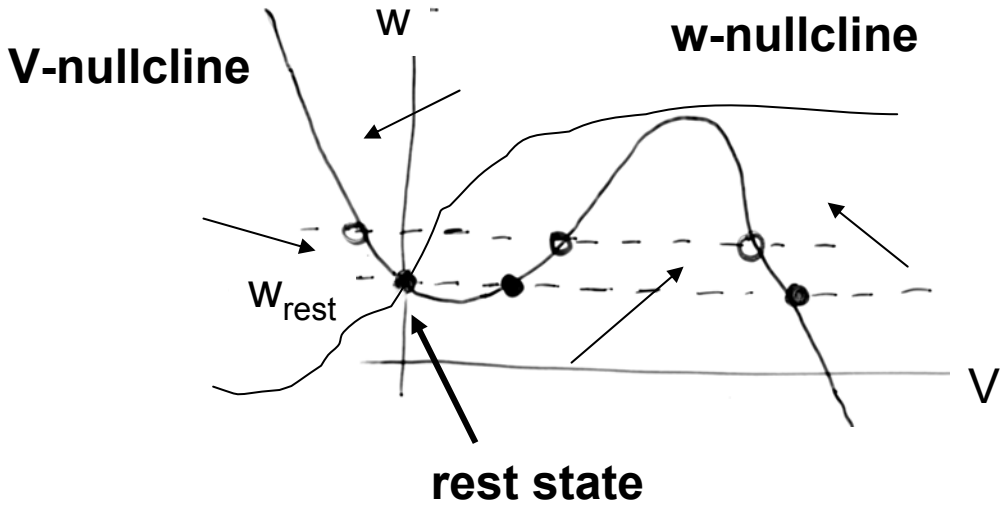
$dV/dt = 0$

$I_{inst}(V,w) = I_{app}$

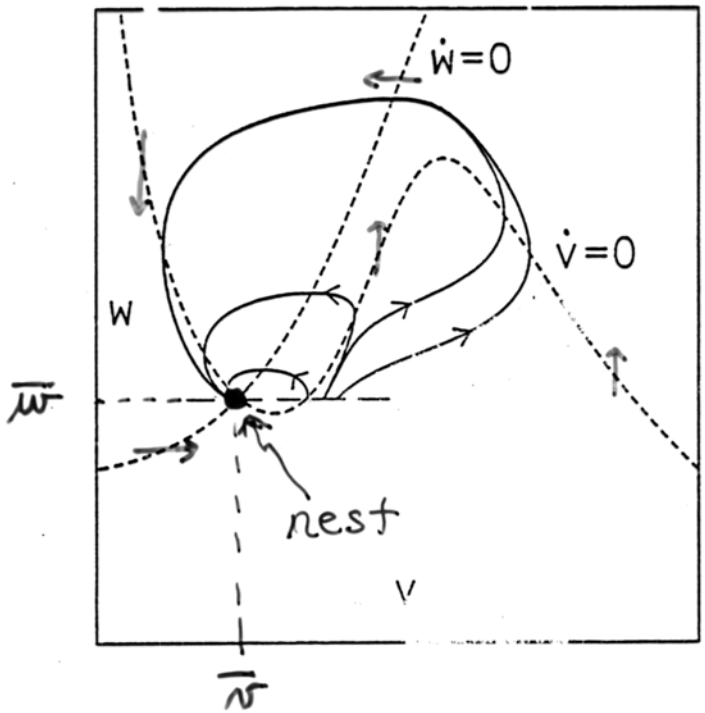


$dw/dt = 0$

$w = w_\infty(V)$



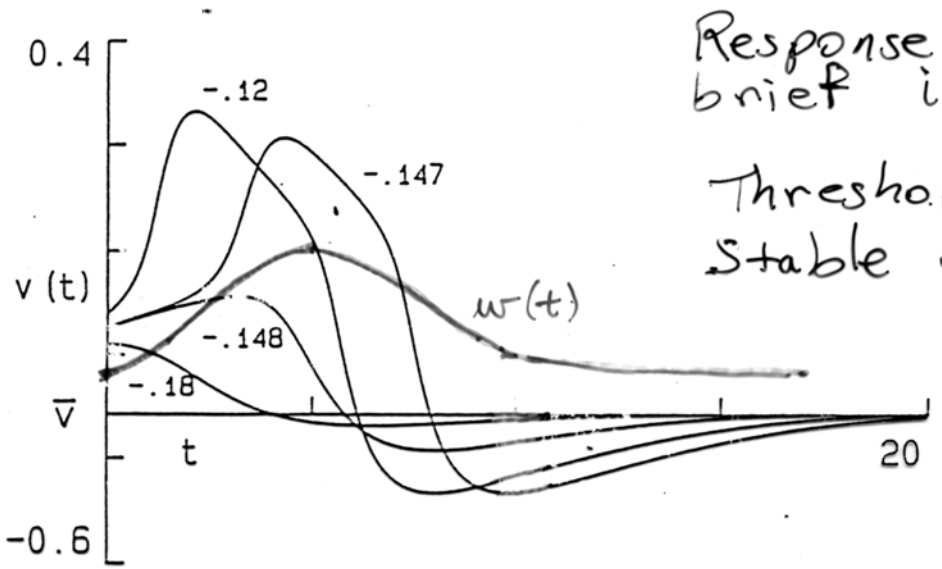
ML model
- excitable regime



V̇=0
3 Branches:
"R, T, E"

Case of small φ

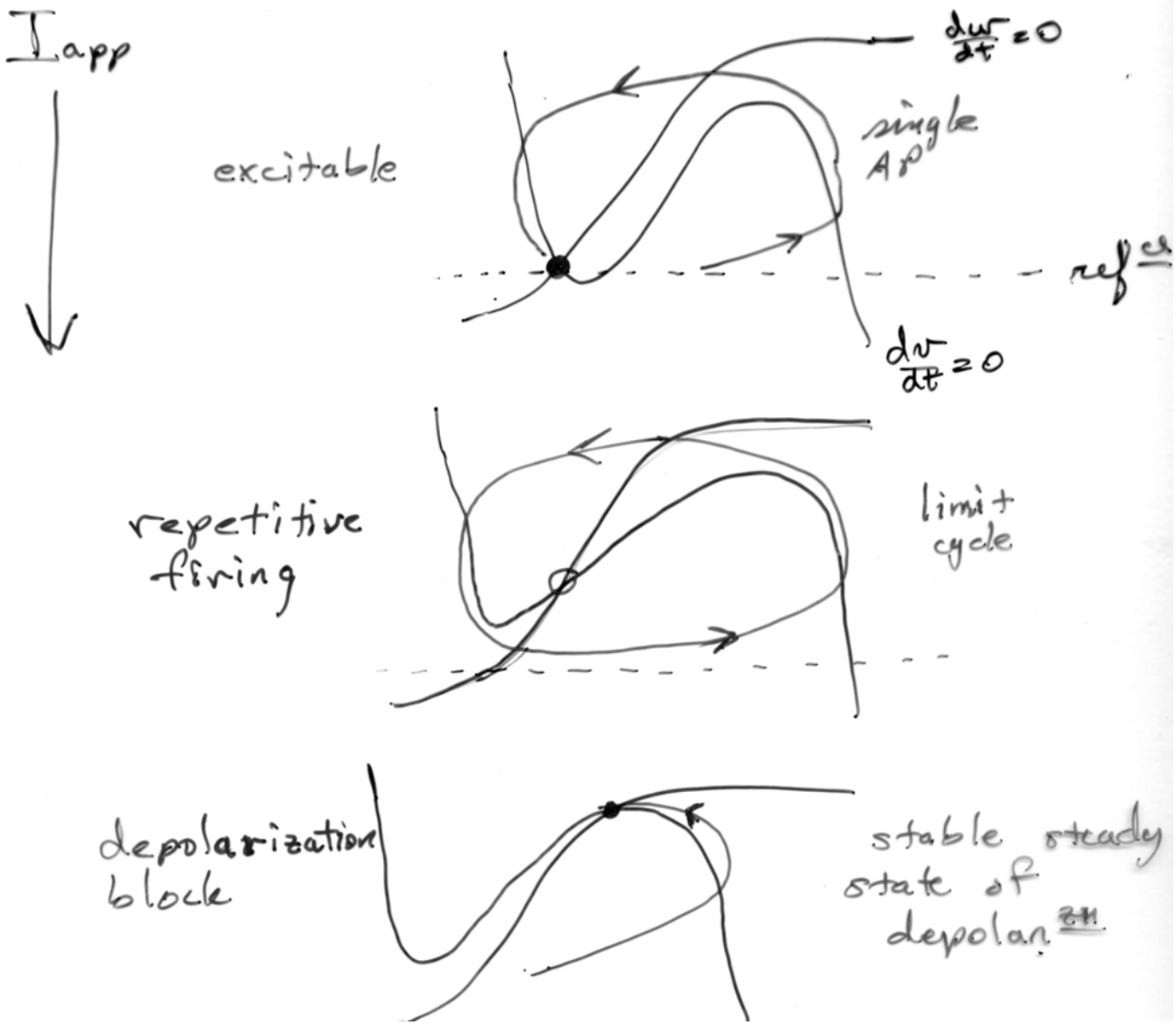
traj hugs V-nullcline - except for up/down jumps.



Response to brief i(t).

Threshold.
Stable rest.

Repetitive Firing in phase plane for M-L model



Repetitive Activity in ML (& HH)

"rest" – unstable

↑ only if on middle branch (math)

Onset is via Hopf bifurcation

Condition for instability*:

damped $i < i_1$ (Hopf) growing $i > i_1$

$$\frac{1}{C_m}\frac{\partial i_{ion}}{\partial v} < -\frac{\phi}{\tau_w}$$

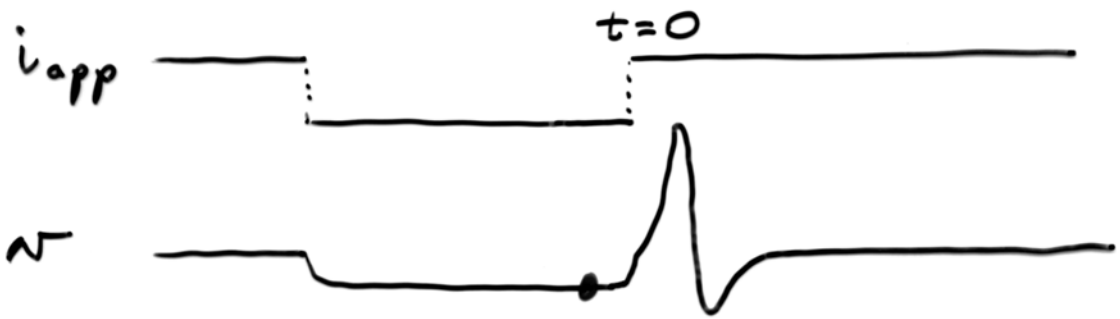
"negative" resistance – destabilizing

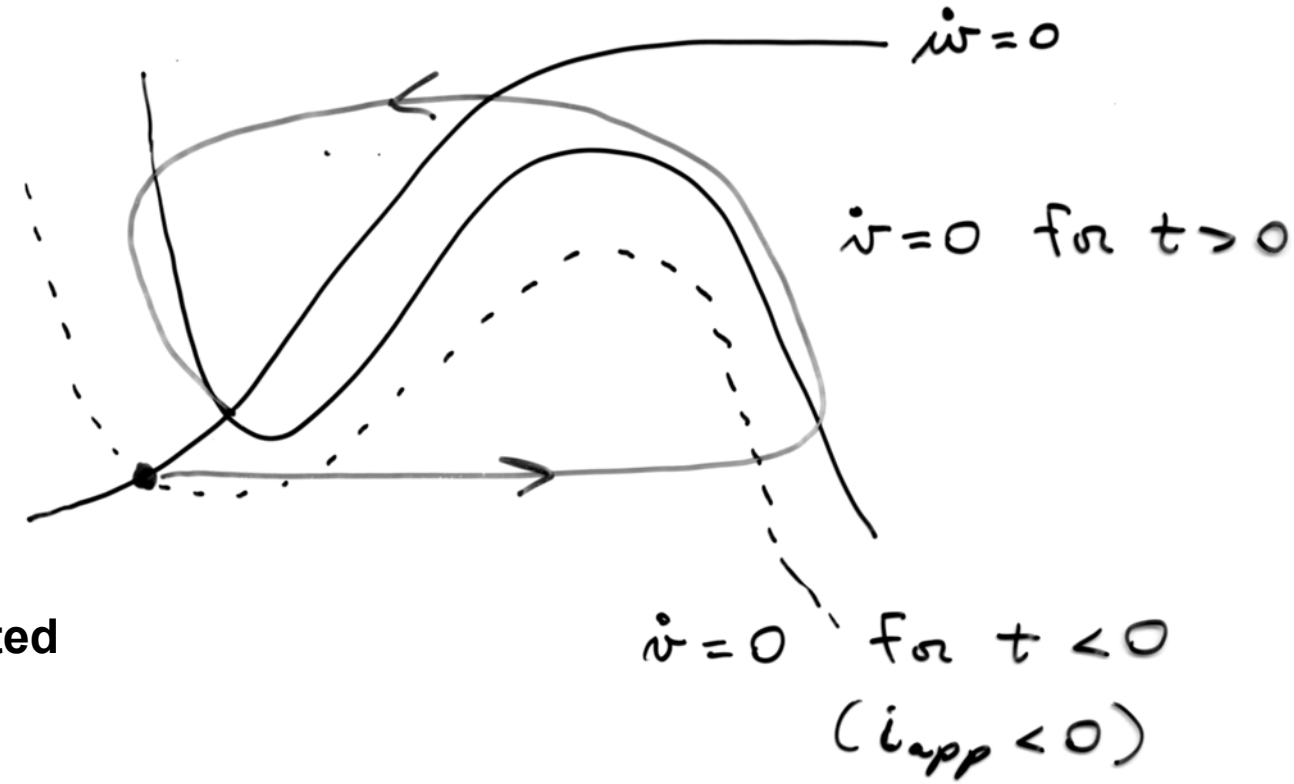
* near "rest" i_{ss} – monotone

"Type II" onset

Hodgkin '48

Anode Break Excitation or Post-Inhibtory Rebound (PIR)

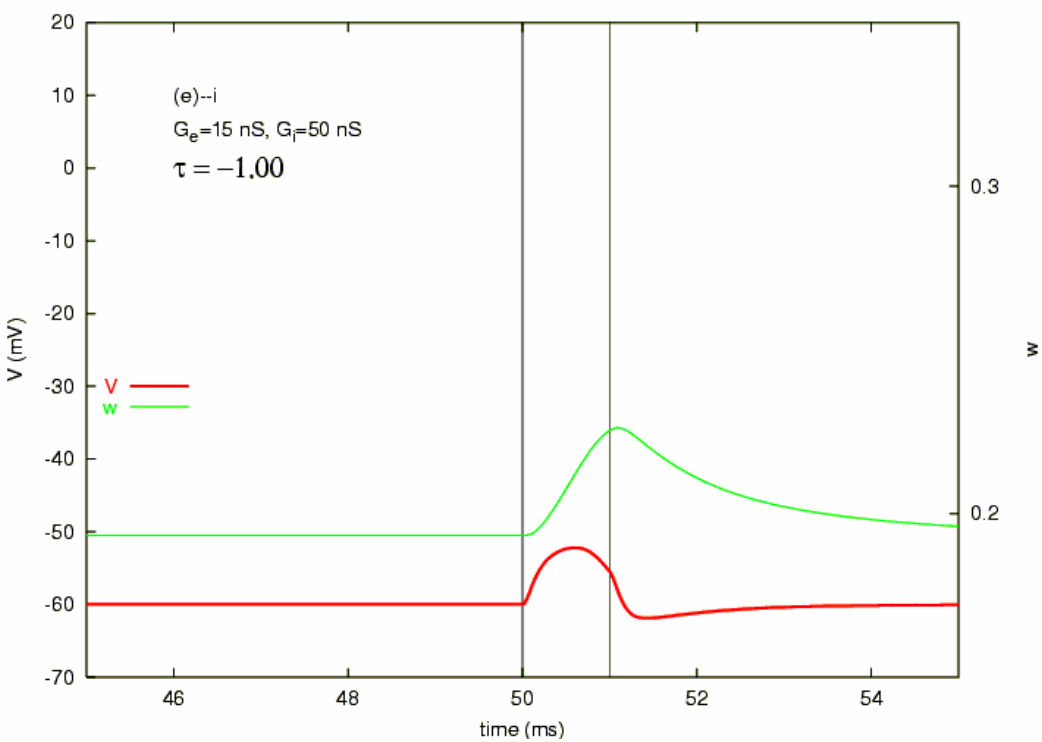


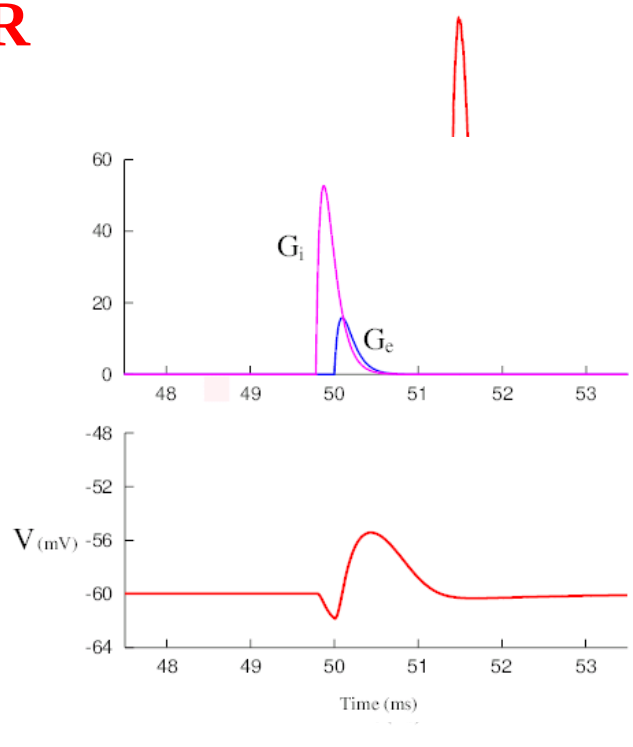


I_K - deactivated

Post inhibitory facilitation, PIF, transient form of PIR

Subthreshold nonlinearities:
ipsp can enhance epsp,
and lead to spiking



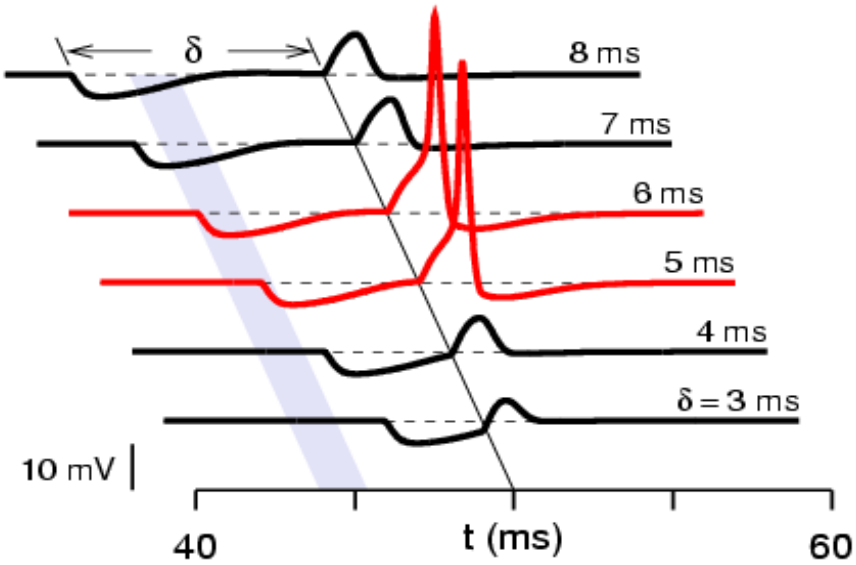


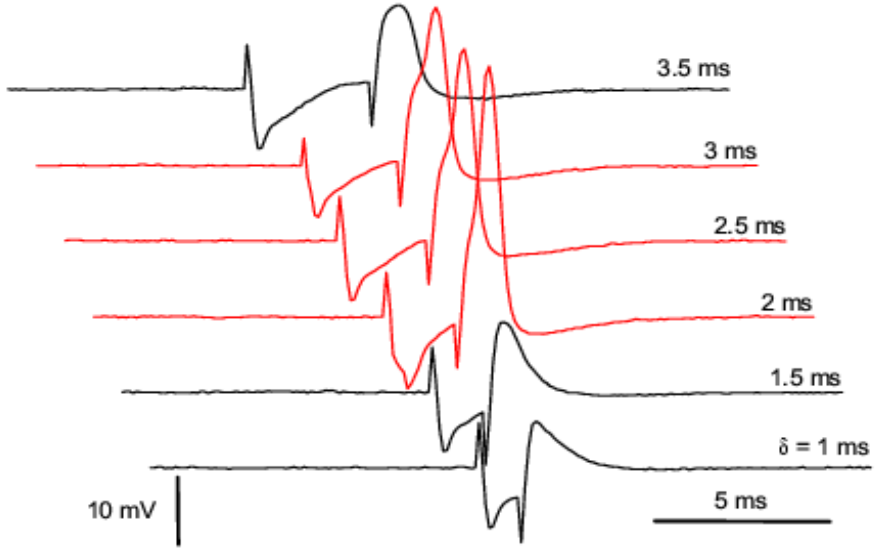
Model of "coincidence-detecting" cell in auditory brain stem.
Has a subthreshold K^+ current I_{KLT} .

Theory

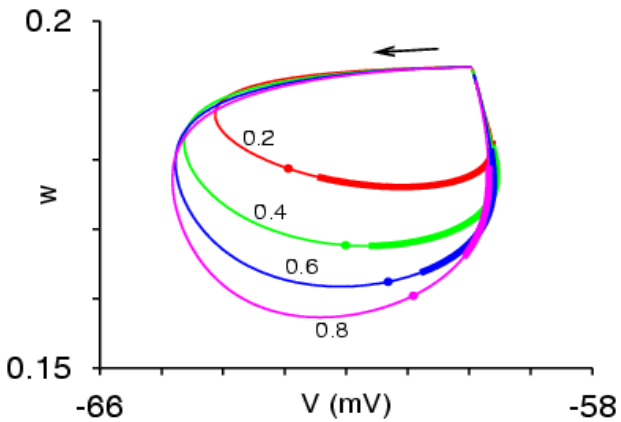
PIF

Experiment (Gerbil MSO, slice)

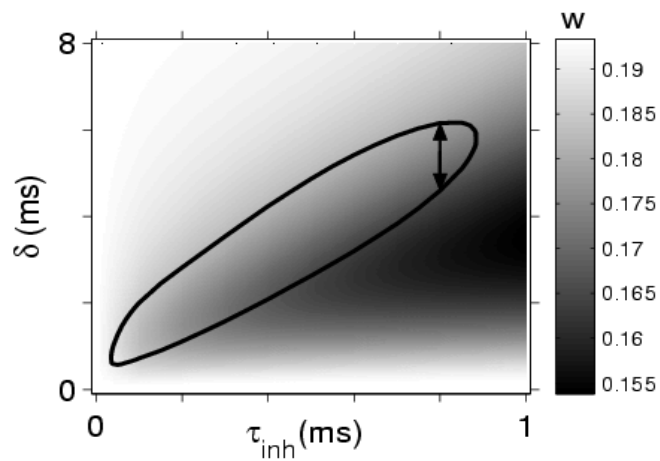




Competing factors:
hyperpolar'zn (farther from V_{th})
and hyperexcitable (reduced w)

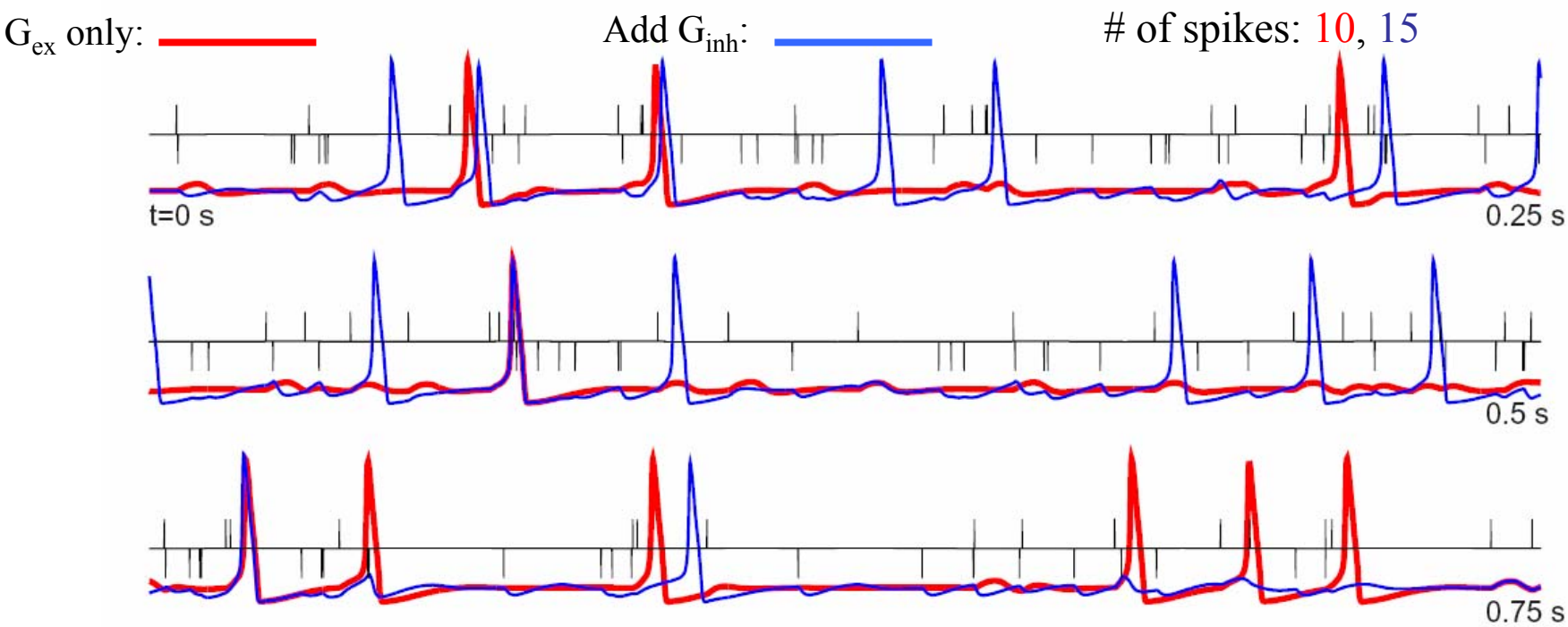


Dependence on τ_{inh}



Boosting Spontaneous Rate with Fast Inhibition, via PIF

Firing Response to Poisson-G_{ex} train Enhanced by Inhibition (Poisson-G_{inh})

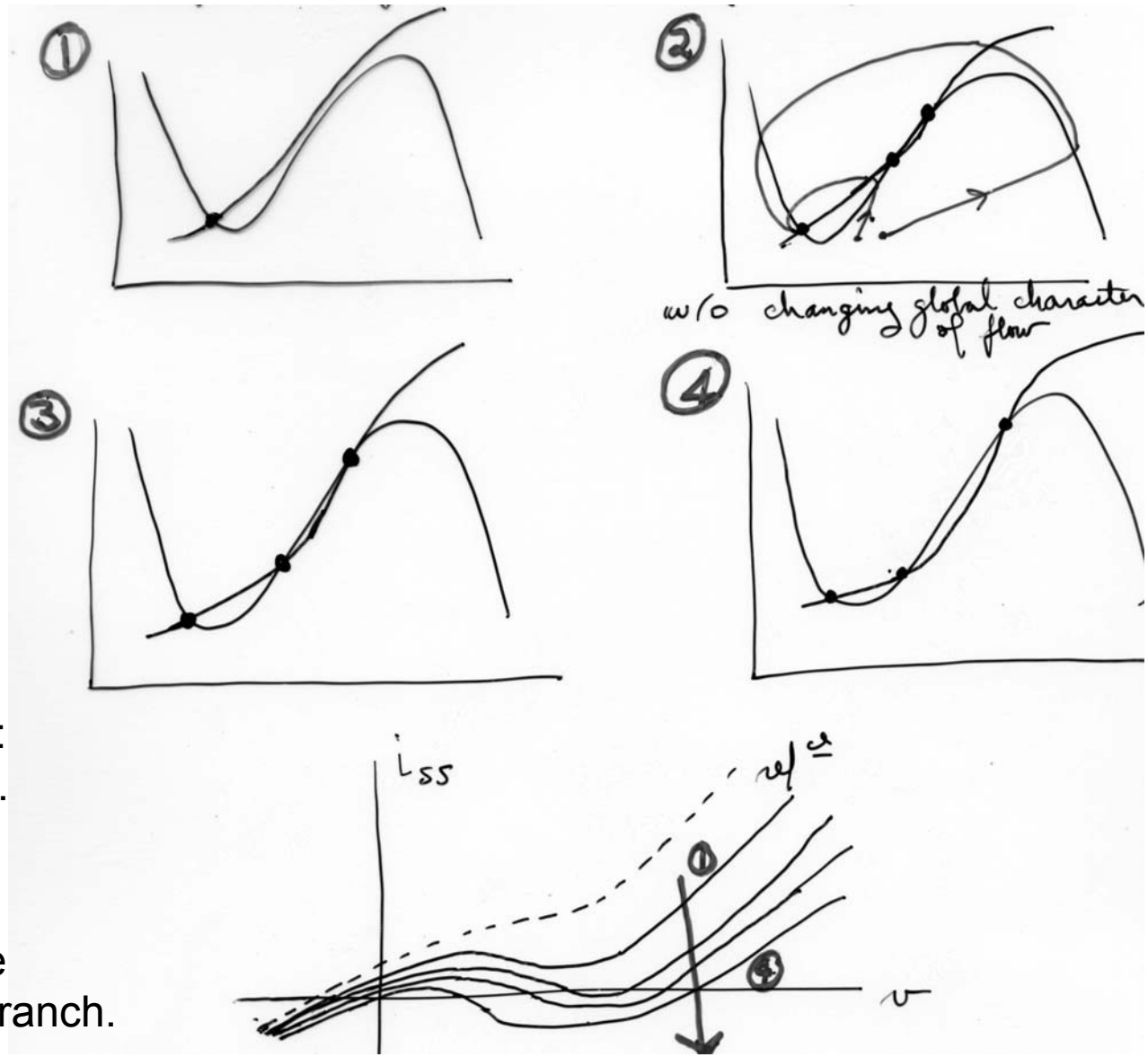


Std HH model; 100 Hz inputs; $\tau_{ex} = \tau_{inh} = 1$ ms

w/ R Dodla. PhysRev E, in press

Adjust param's → changes nullclines: case of 3 "rest" states

3 states → I_{ss}
is N-shaped



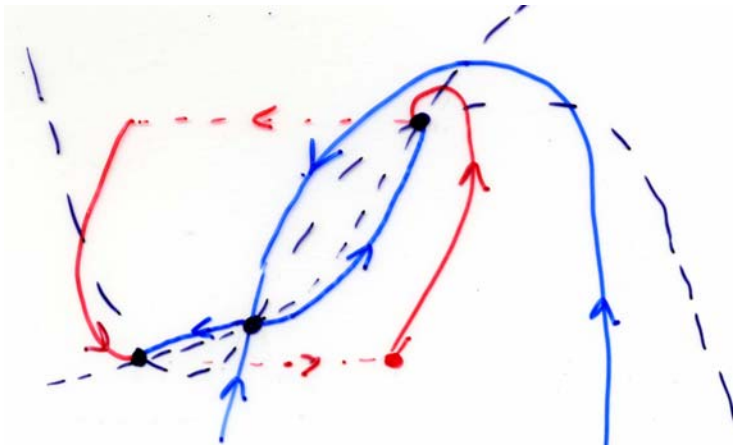
Stable or Unstable?

3 states – not necessarily:
stable – unstable – stable.

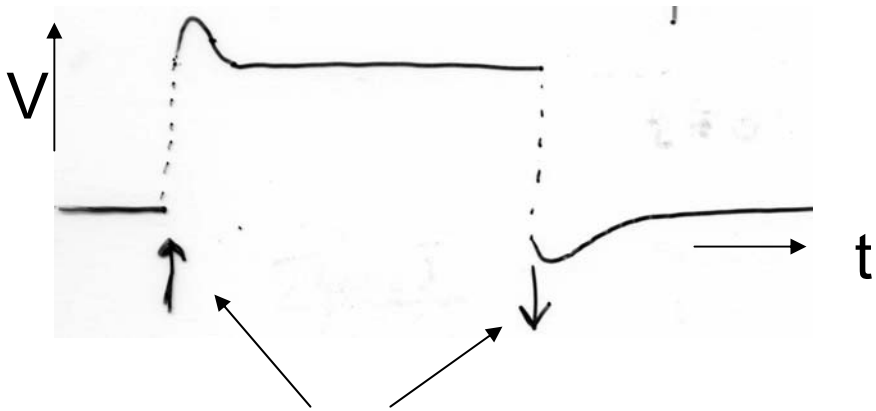
Φ small enough,
then both upper/middle
unstable if on middle branch.

ML: φ large → 2 stable steady states

Neuron is bistable: plateau behavior.



Saddle point, with
stable and unstable manifolds

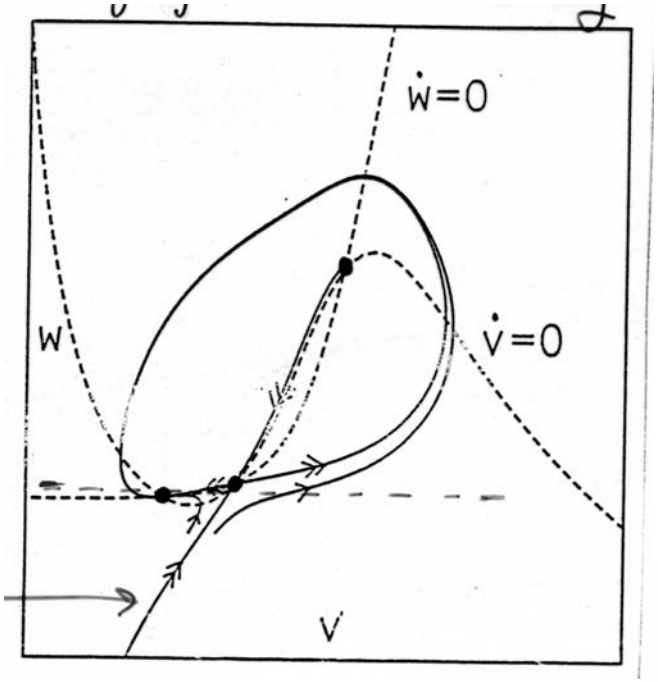


I_{app} switching pulses

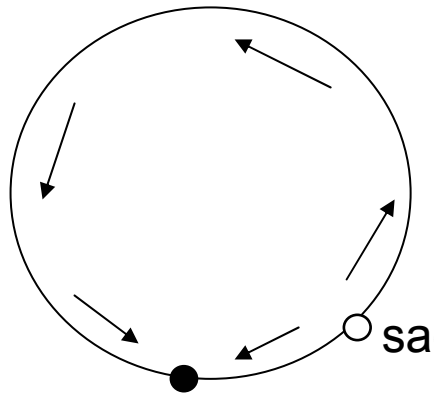
e.g., HH with
V_K = 24 mV

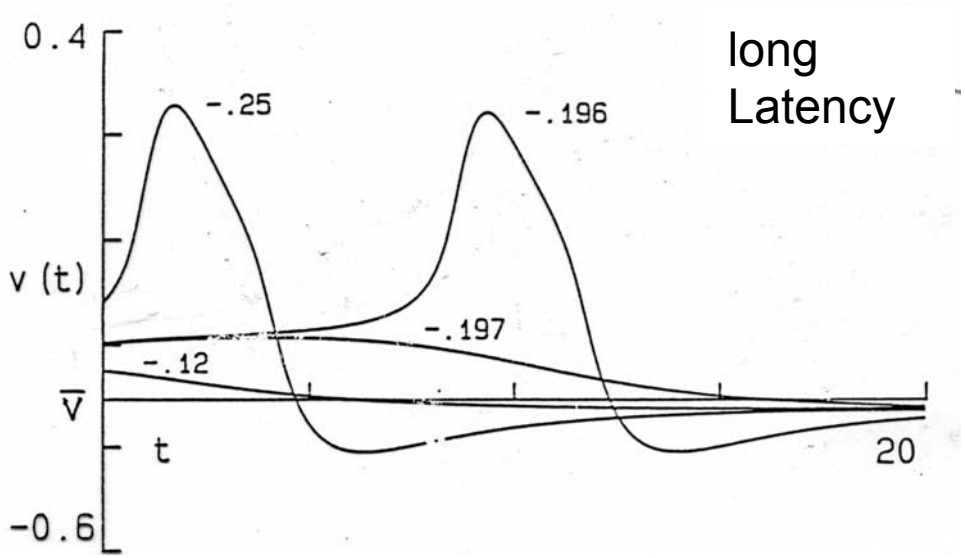
ML: φ small → both upper states are unstable

Neuron is excitable with strict threshold.



threshold
separatrix





I_{ss} must be N-shaped.

$I_{K\text{-}A}$ can give long latency but not necessary.

Onset of Repetitive Firing – 3 rest states

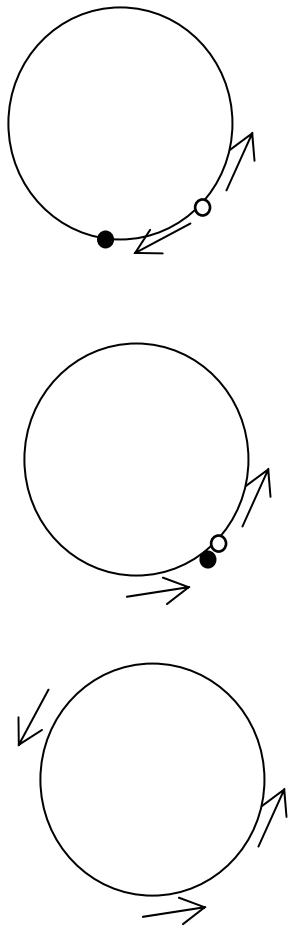
SNIC- saddle-node on invariant circle

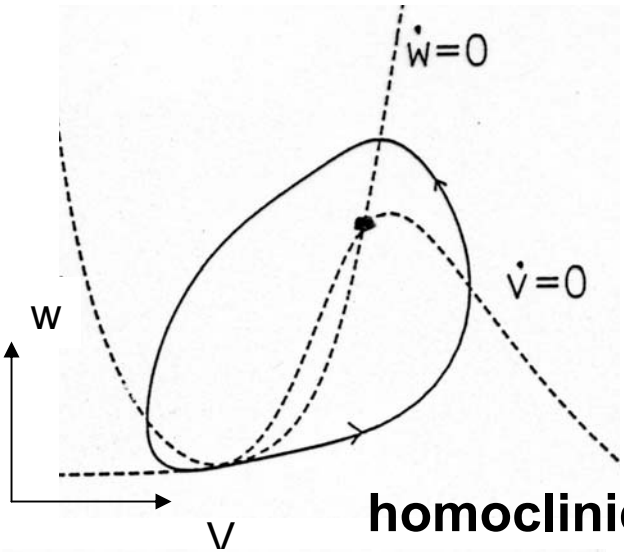
excitable

I_{app}

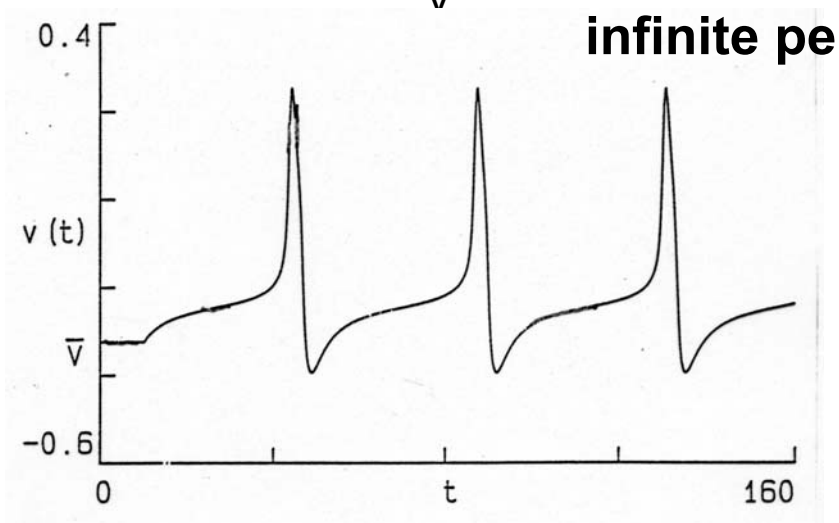
saddle-node

limit cycle





homoclinic orbit; infinite period



emerge w/ large amplitude – zero frequency

ML: φ small

freq $\sim\sqrt{I-I_1}$

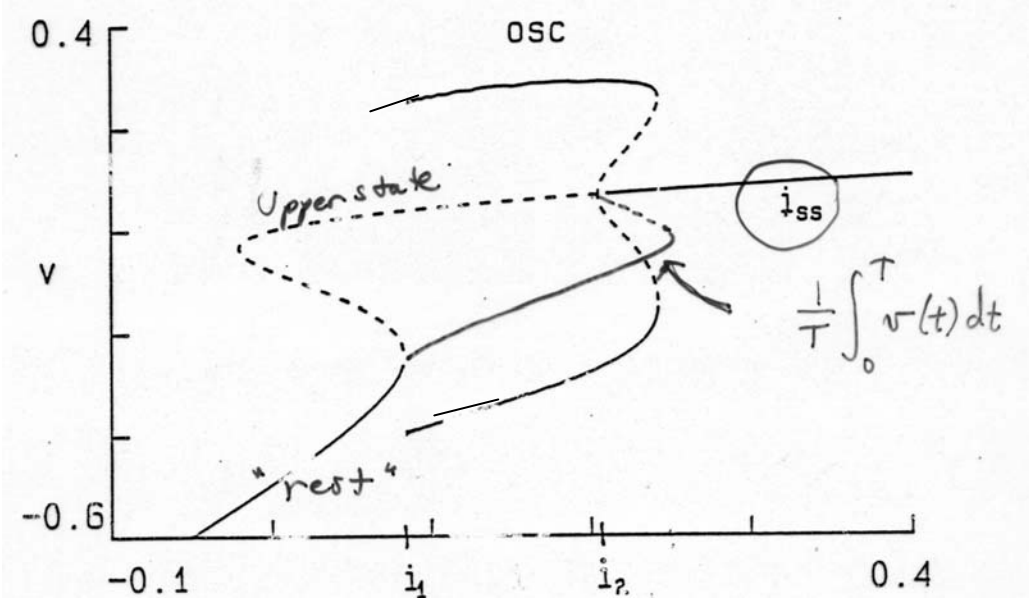
low freq but no conductances very slow

$I_{K\text{-}A}$? (Connor et al '77)

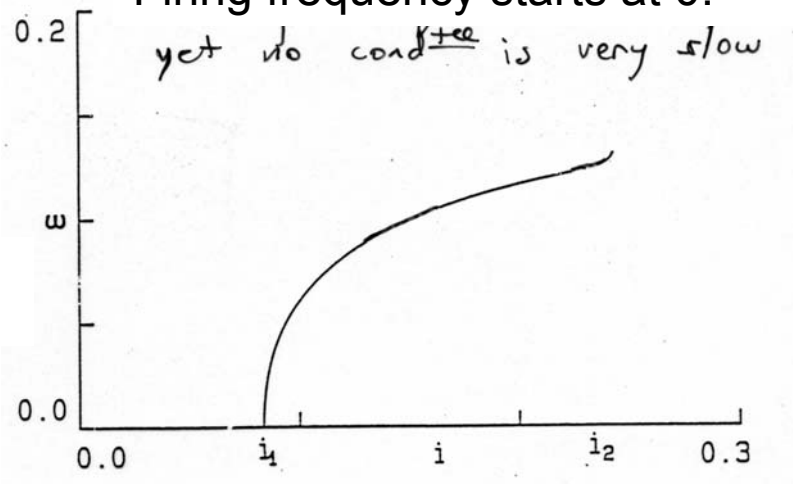
"Type I" onset

Hodgkin '48

Response/Bifurcation diagram



Firing frequency starts at 0.



Firing rate model (Amari-Wilson-Cowan) for dynamics of excitatory-inhibitory populations.

$$\tau_e \, dr_e/dt = -r_e + S_e(a_{ee} \, r_e - a_{ei} \, r_i + I_e)$$

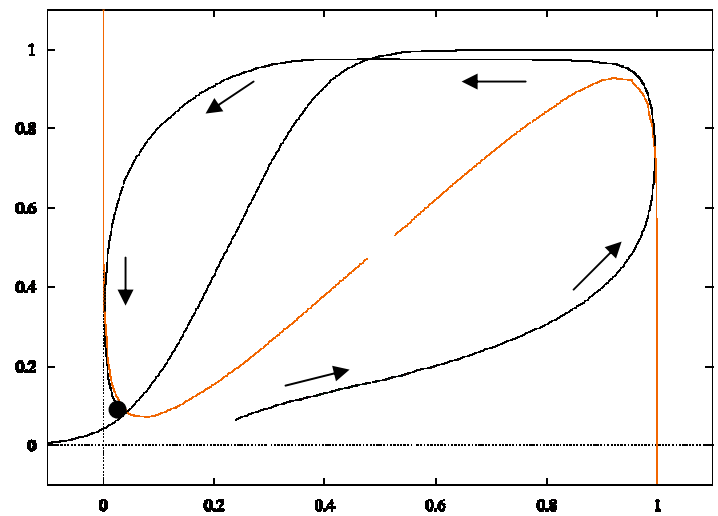
$$\tau_i \, dr_i/dt = -r_i + S_i(a_{ie} \, r_e - a_{ii} \, r_i + I_i)$$

$r_i(t)$, $r_e(t)$ -- average firing rate (across population and "over spikes")

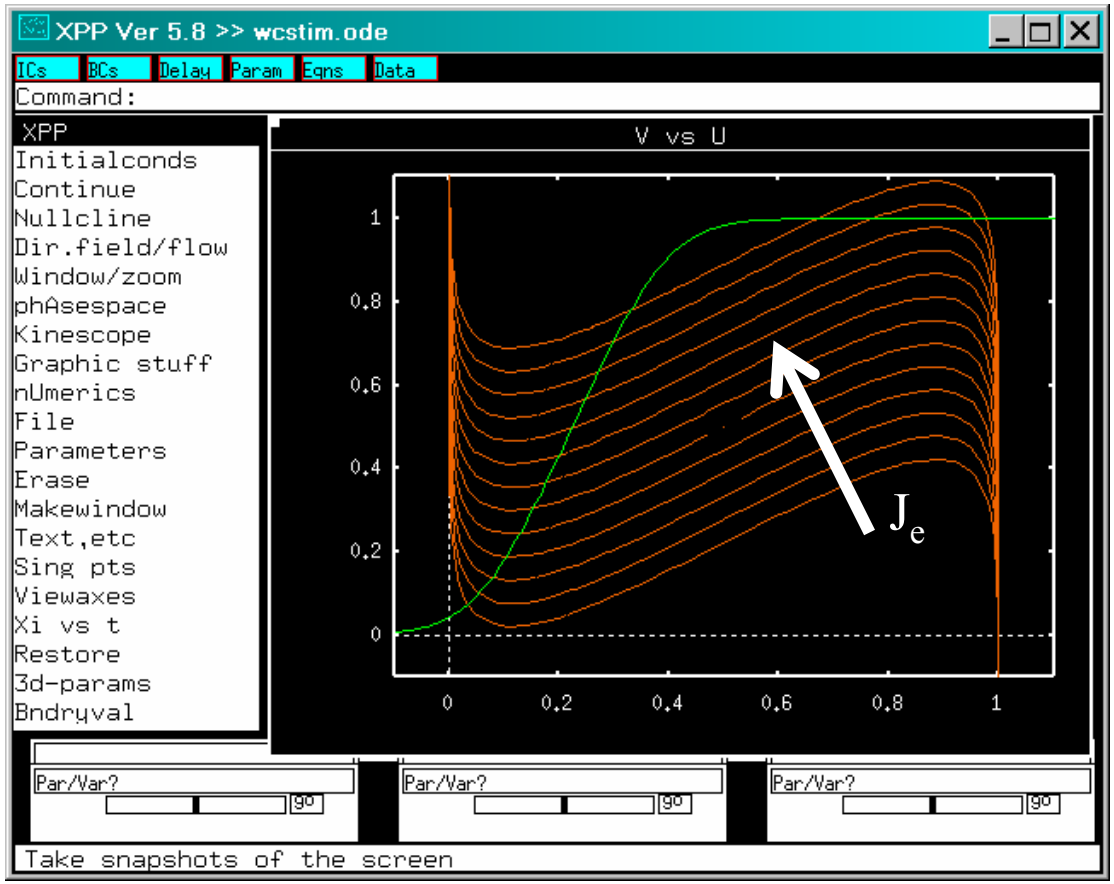
τ_e , τ_i -- "recruitment" time scale

S_e(input) , S_i(input) – input/output relations, sigmoids

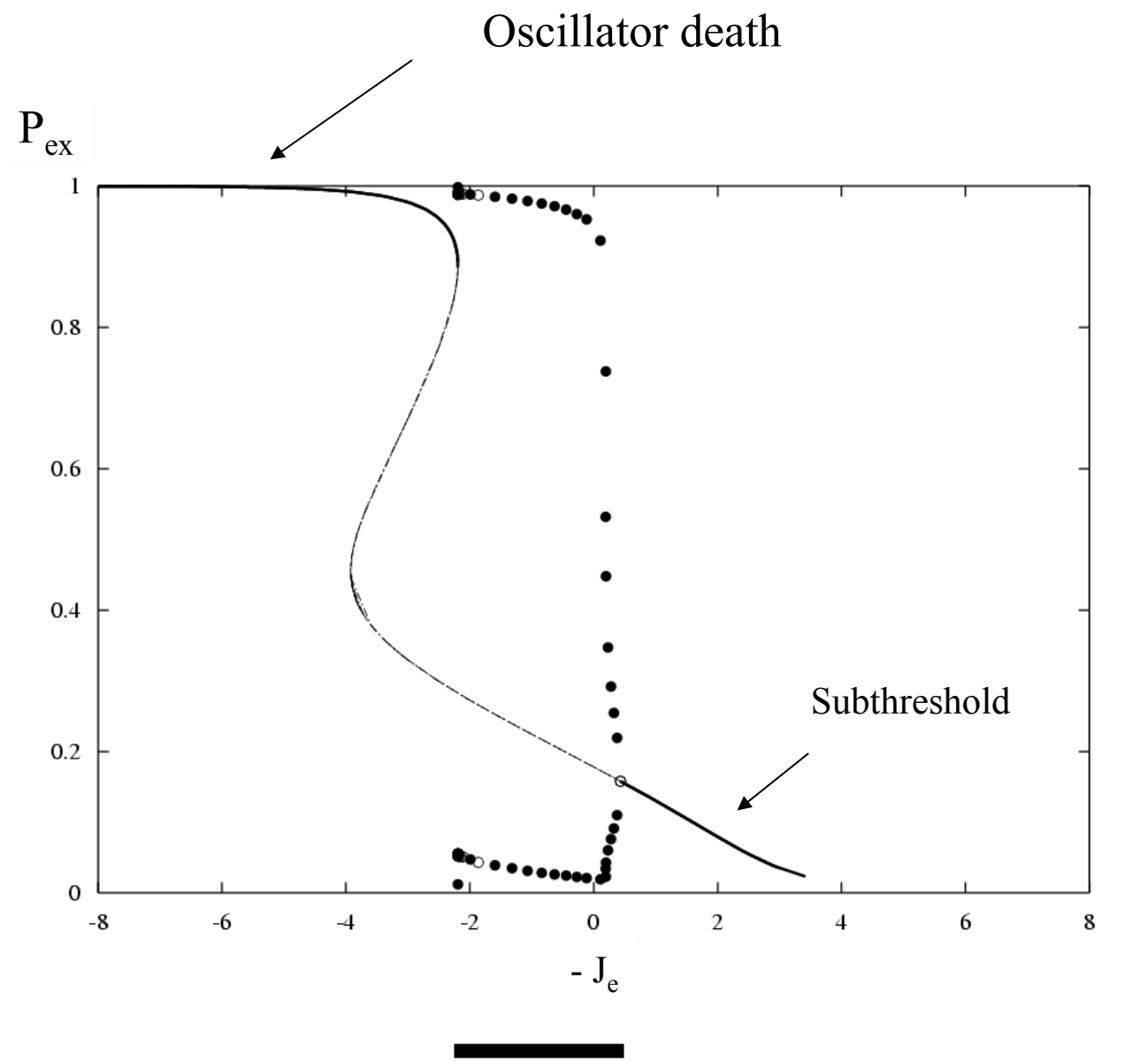
a_{ee} etc – "synaptic weights"



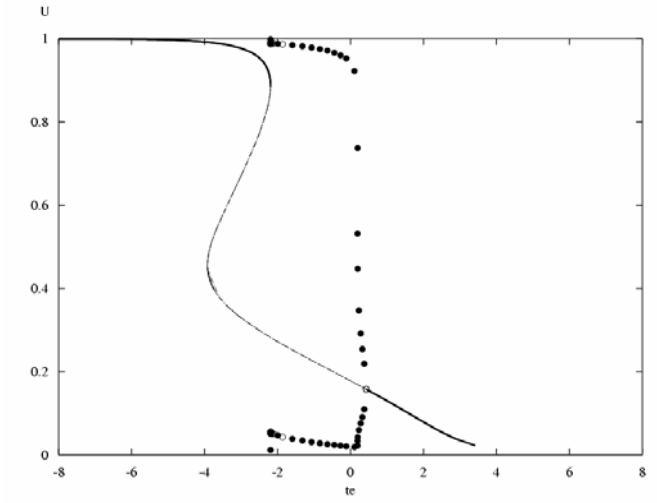
Wilson-Cowan Model
dynamics in the phase plane.



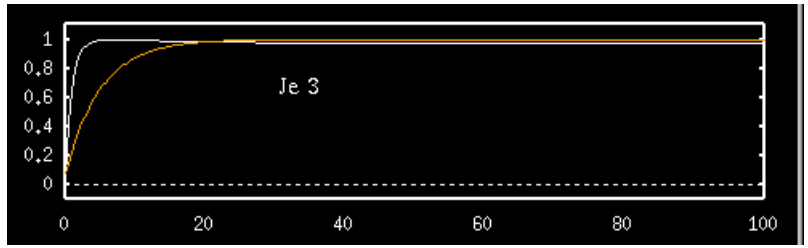
Phase plane, nullclines for range of J_e.

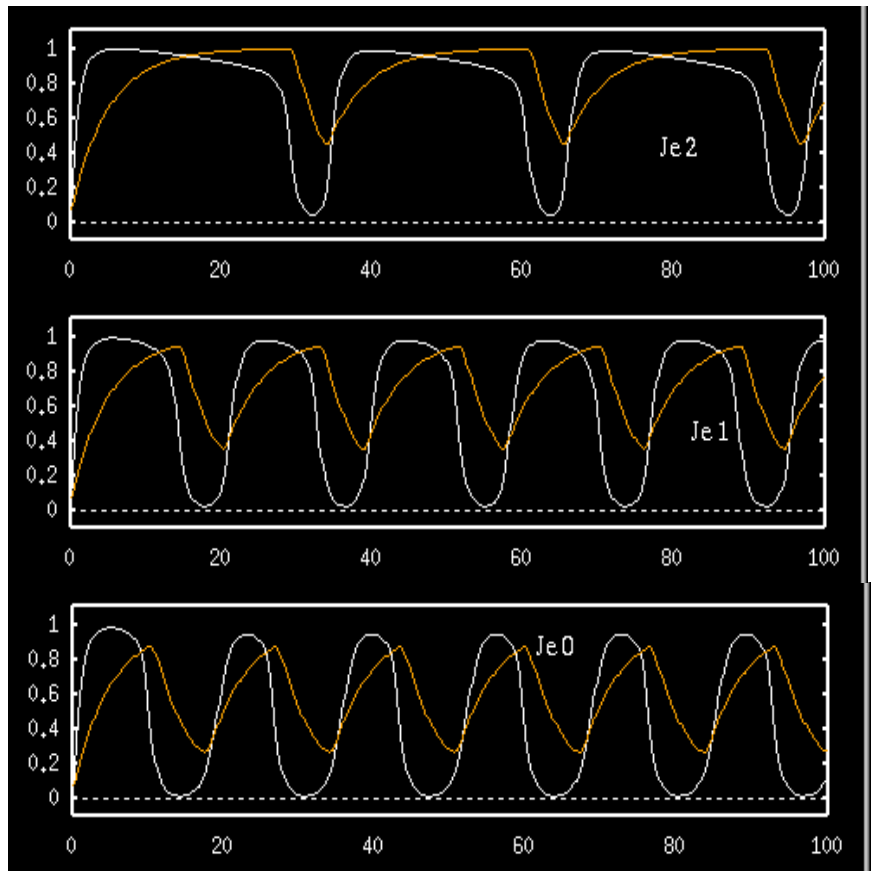


Regime of repetitive activity

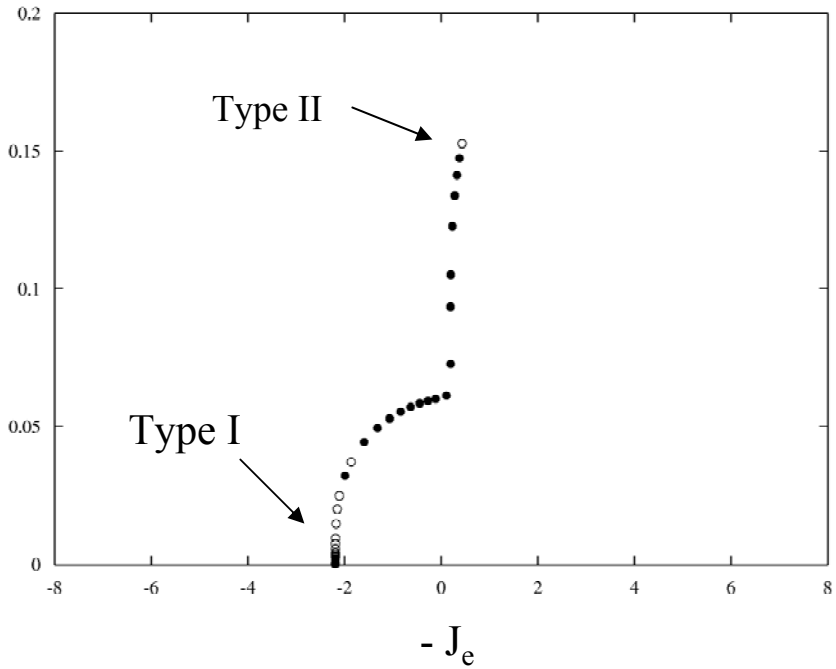


“Oscillator Death” but cells are firing





Frequency



Transition from Excitable to Oscillatory

Same for W-C network models.

Type II, min freq ≠ 0

I_{ss} monotonic

subthreshold oscill'ns

excitable w/o distinct threshold

excitable w/ finite latency

Type I, min freq = 0

I_{SS} N-shaped – 3 steady states

w/o subthreshold oscillations

excitable w/ "all or none" (saddle) threshold

excitable w/ infinite latency

Hodgkin '48 – 2 classes of repetiitive firing;
Also - Class I less regular ISI near threshold

J Neurophysiol 92: 2283–2294, 2004;
10.1152/jn.00109.2004.

Threshold Firing Frequency–Current Relationships of Neurons in Rat Somatosensory Cortex: Type 1 and Type 2 Dynamics

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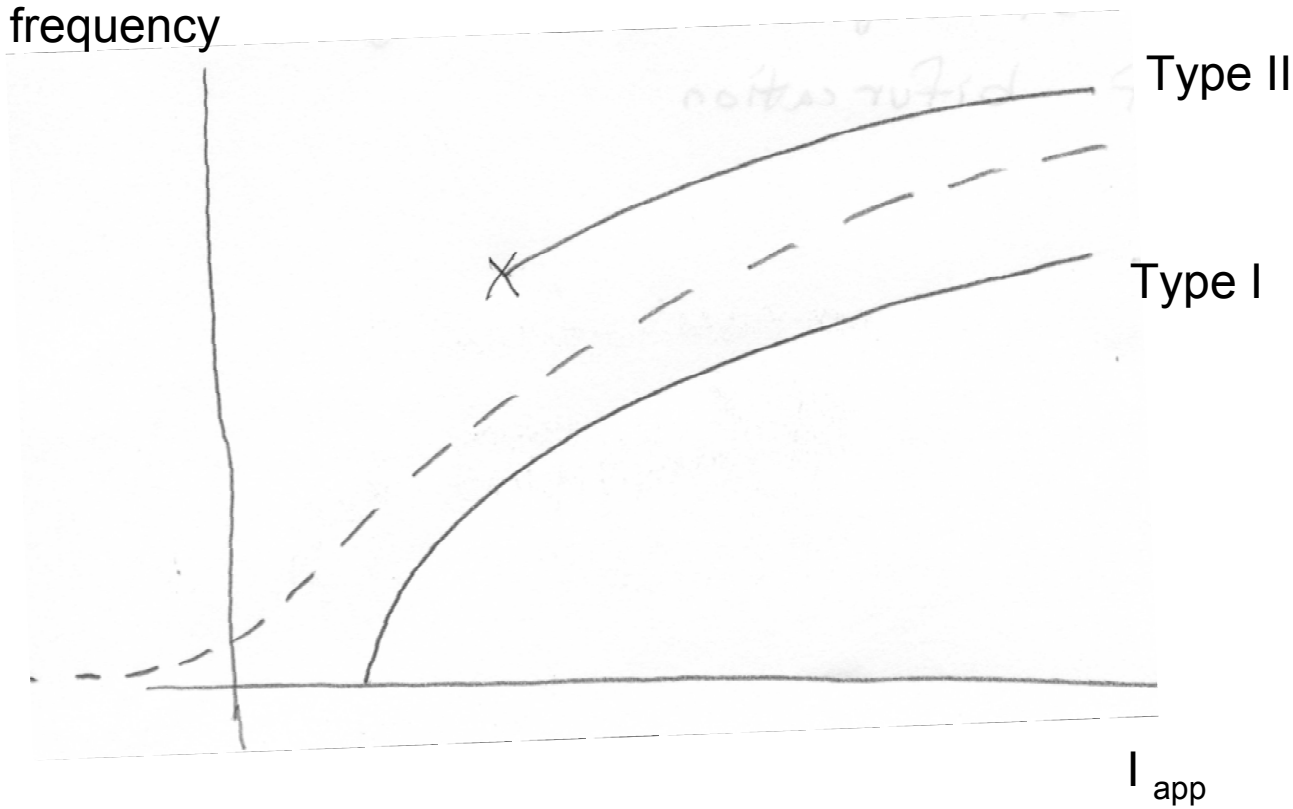
Tateno, T., A. Harsch, and H.P.C. Robinson. Threshold firing frequency–current relationships of neurons in rat somatosensory cortex: type 1 and type 2 dynamics. *J Neurophysiol* 92: 2283–2294, 2004; 10.1152/jn.00109.2004. Neurons and dynamical models of spike generation display two different types of threshold behavior, with steady current stimulation: type 1 [the firing frequency vs. current (*f–I*) relationship is continuous at threshold) and type 2 (discontinuous *f–I*)]. The dynamics at threshold can have profound effects on the encoding of input as spikes, the sensitivity of spike generation to input noise, and the coherence of population firing. We have examined the *f–I* and frequency–conductance (*f–g*) relationships of cells in layer 2/3 of slices of young (15–21 DIV) rat somatosensory cortex, focusing in detail on the nature of the threshold. Using white-noise stimulation, we also measured firing frequency and interspike interval variability as a function of noise amplitude. Regular-spiking (RS) pyramidal neurons show a type 1 threshold, consistent with their well-known ability to fire regularly at very low frequencies. In fast-spiking (FS) inhibitory interneurons, although regular firing is supported over a wide range of frequencies, there is a clear discontinuity in their *f–I* relationship at threshold (type 2), which has not previously been highlighted. FS neurons are unable to support maintained periodic firing below a critical frequency f_c, in the range of 10 to 30 Hz. Very close to threshold, FS cells switch irregularly between bursts of periodic firing and subthreshold oscillations. These characteristics mean that the dynamics of RS neurons are well suited to encoding inputs into low-frequency firing rates, whereas the dynamics of FS neurons are suited to maintaining and quickly synchronizing to gamma and higher-frequency input.

of these 2 types, which thus represent the behavior of a wide range of excitable membranes.

Even simple dynamical models of spike generation can exhibit both kinds of behavior, depending on their parameters (Morris and Lecar 1981; Rinzel and Ermentrout 1998). In these models, because of the different natures of dynamical bifurcation at threshold, type 1 behavior is associated with all-or-nothing spikes, whereas type 2 behavior is associated with graded spike amplitude and subthreshold oscillations. Recently, modeling studies have shown that the threshold type of the neuron profoundly affects the reliability of spike generation in the presence of noise (Gutkin and Ermentrout 1998; Robinson and Harsch 2002). Experimental classification of the responses of neurons in the cortex, however, has focused mostly on the form of the frequency vs. current (*f–I*) relationship in responses that are well above threshold (Connors and Gutnick 1990; Kawaguchi and Kubota 1997; Nowak et al. 2003); a clear classification of the continuity or discontinuity of the *f–I* relationship at threshold is lacking. Therefore in this paper we study the thresholds of 2 well-characterized types of cell—regular-spiking and fast-spiking neurons—and show that they follow type 1 and type 2 behaviors, respectively. We discuss what impact this could have on the roles of these 2 cell types in the cortical network.

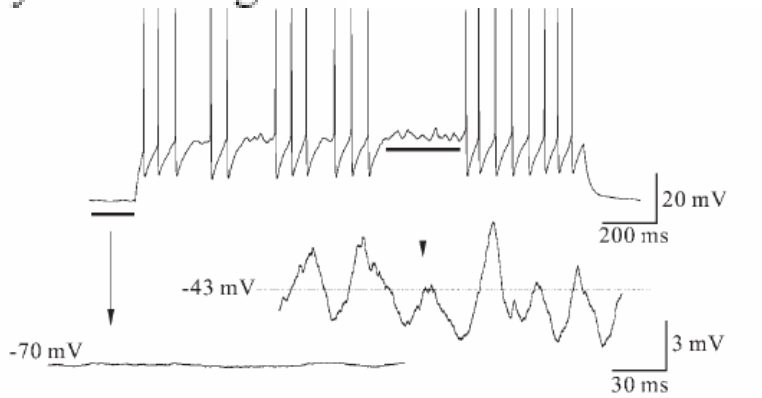
METHODS

Noise smooths the f-I relation

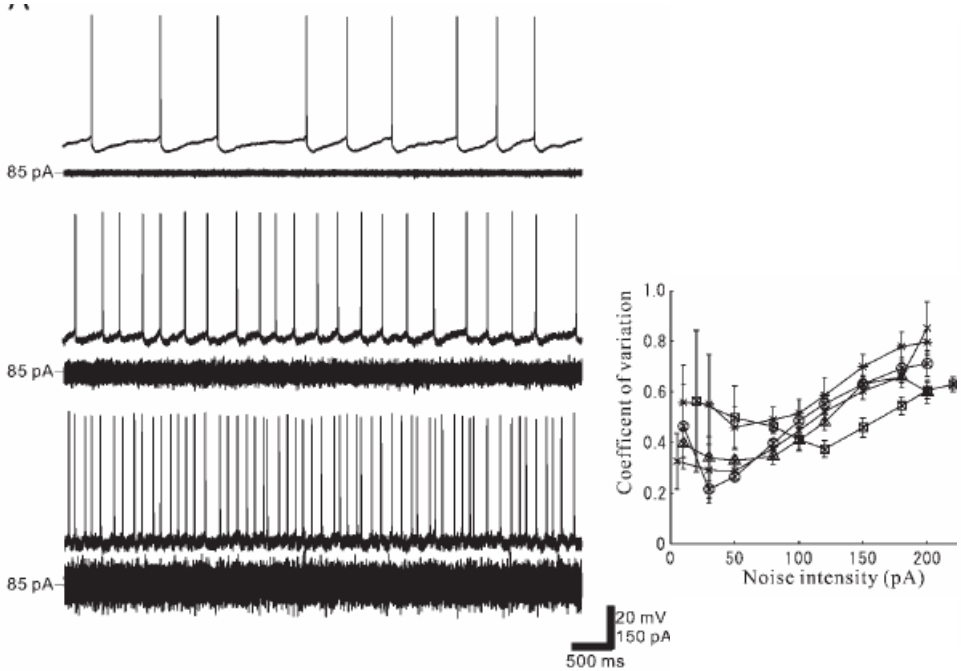


to 30 Hz. Very close to threshold, FS cells switch irregularly between bursts of periodic firing and subthreshold oscillations. These characteristics mean that the dynamics of RS neurons are well suited to encoding inputs into low-frequency firing rates, whereas the dynamics of FS neurons are suited to maintaining and quickly synchronizing to gamma and higher-frequency input.

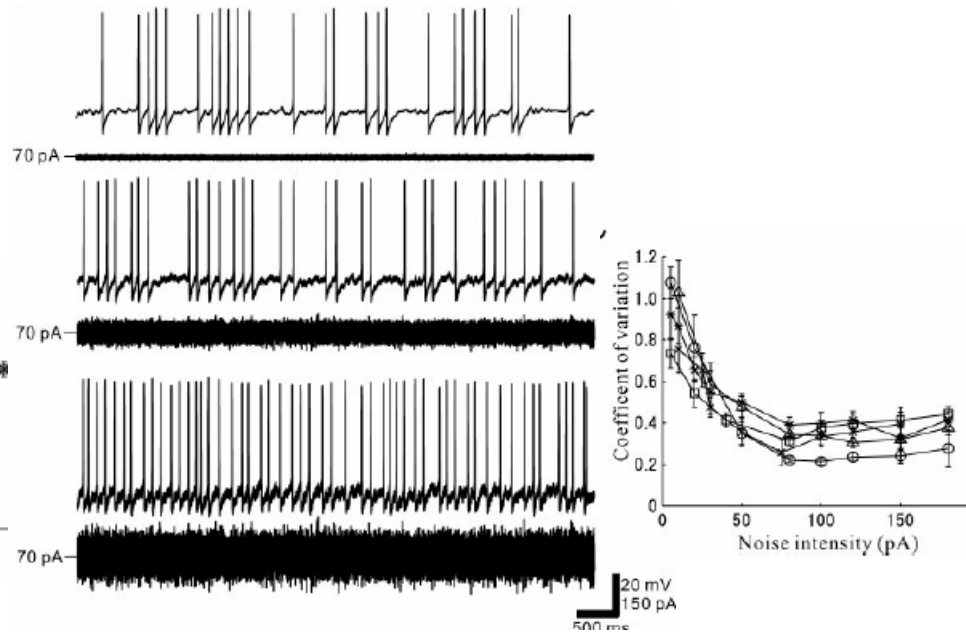
FS cell
near threshold



RS cell, w/ noise



FS cell, w/ noise



Take Home Message

Excitability/Oscillations : fast autocatalysis + slower negative feedback

Value of reduced models

Time scales and dynamics

Phase space geometry

Different dynamic states – “Bifurcations”

XPP software:http://www.pitt.edu/~phase/ (Bard Ermentrout’s home page)