

Nonlinear Nonconservative Systems

(i) Van der Pol Oscillator Dynamics

$$\ddot{x} - \alpha(1-x^2)\dot{x} + \omega^2x = 0$$

$$\dot{x} = y$$

$$\dot{y} = \alpha(1-x^2)y - \omega^2x$$

This can be simplified by letting

$$t' = \omega t$$

then the Van der Pol equation becomes

$$x'' - \epsilon(1-x^2)x' + x = 0$$

where $x' = \frac{dx}{dt'}$

$$\epsilon = \frac{\alpha}{\omega}$$

∴

$$x' = y$$

$$y' = \epsilon(1-x^2)y - x$$

♪ Singular Point at (0,0)

$$\tilde{A} = \begin{bmatrix} 0 & 1 \\ -1-2\epsilon xy & \epsilon - \epsilon x^2 \end{bmatrix}$$

At (0,0) $\Rightarrow \det(\tilde{A} - \lambda \tilde{I}) = 0$

$$\begin{vmatrix} -\hat{\lambda} & 1 \\ -1 & \epsilon - \hat{\lambda} \end{vmatrix} = 0$$

$$\hat{\lambda}^2 - \epsilon \hat{\lambda} + 1 = 0$$

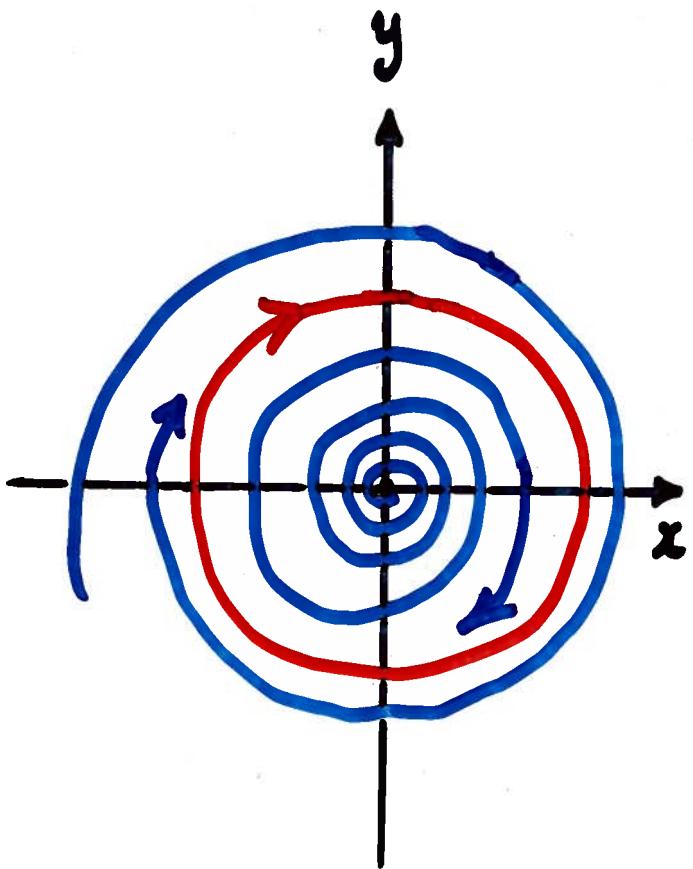
$$\hat{\lambda} = \frac{1}{2} [\epsilon \pm \sqrt{\epsilon^2 - 4}]$$

Real part of eigen values > 0 (for $\epsilon > 0$)

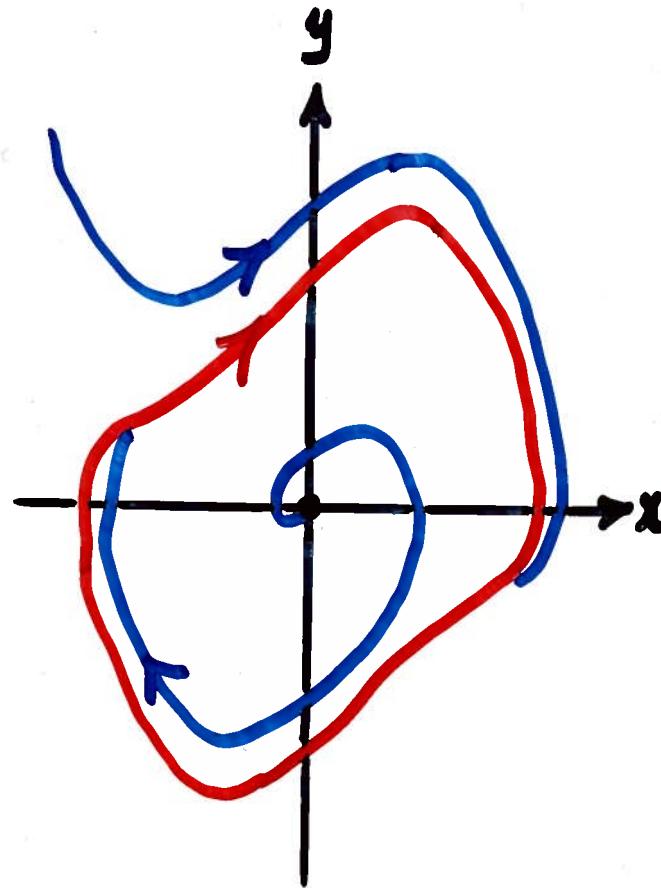
$0 < \epsilon < 2 \Rightarrow$ unstable focus

$\epsilon \geq 2 \Rightarrow$ unstable node

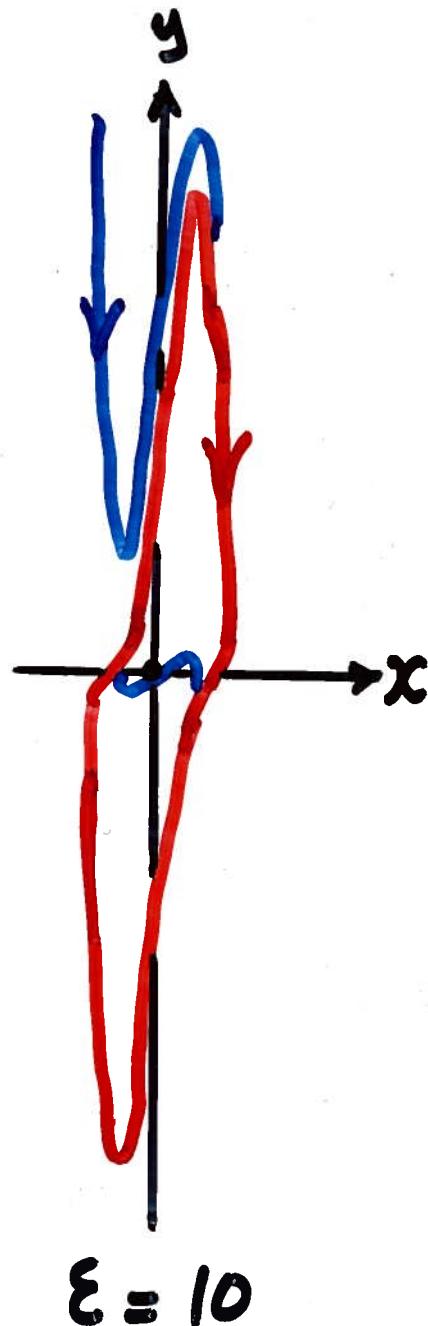
In the Phase Plane



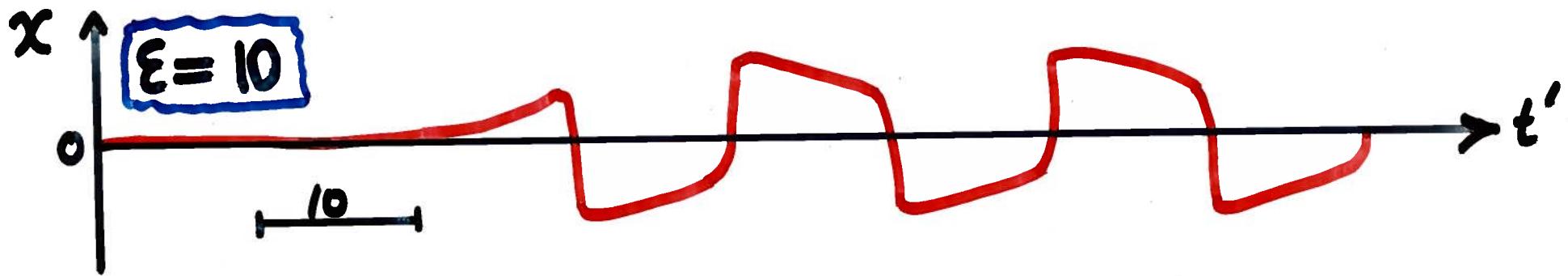
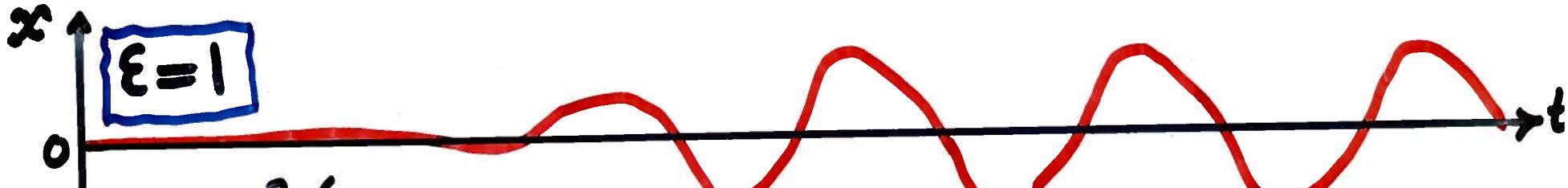
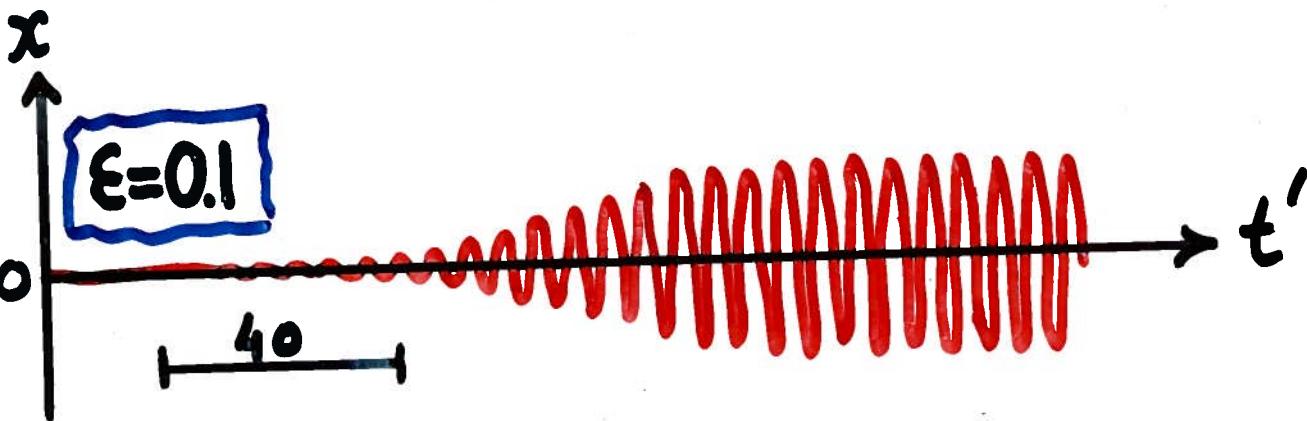
$$\epsilon = 0.1$$



$$\epsilon = 1$$



$$\epsilon = 10$$



The Van der Pol equation can be written as two first order simultaneous differential equations using the Lienard Transformation

$$y = \frac{\dot{x}}{\alpha} + \frac{x^3}{3} - x$$

to obtain

$$\dot{x} = \alpha \left(y + x - \frac{x^3}{3} \right)$$

$$\dot{y} = -\frac{\omega^2}{\alpha} x$$

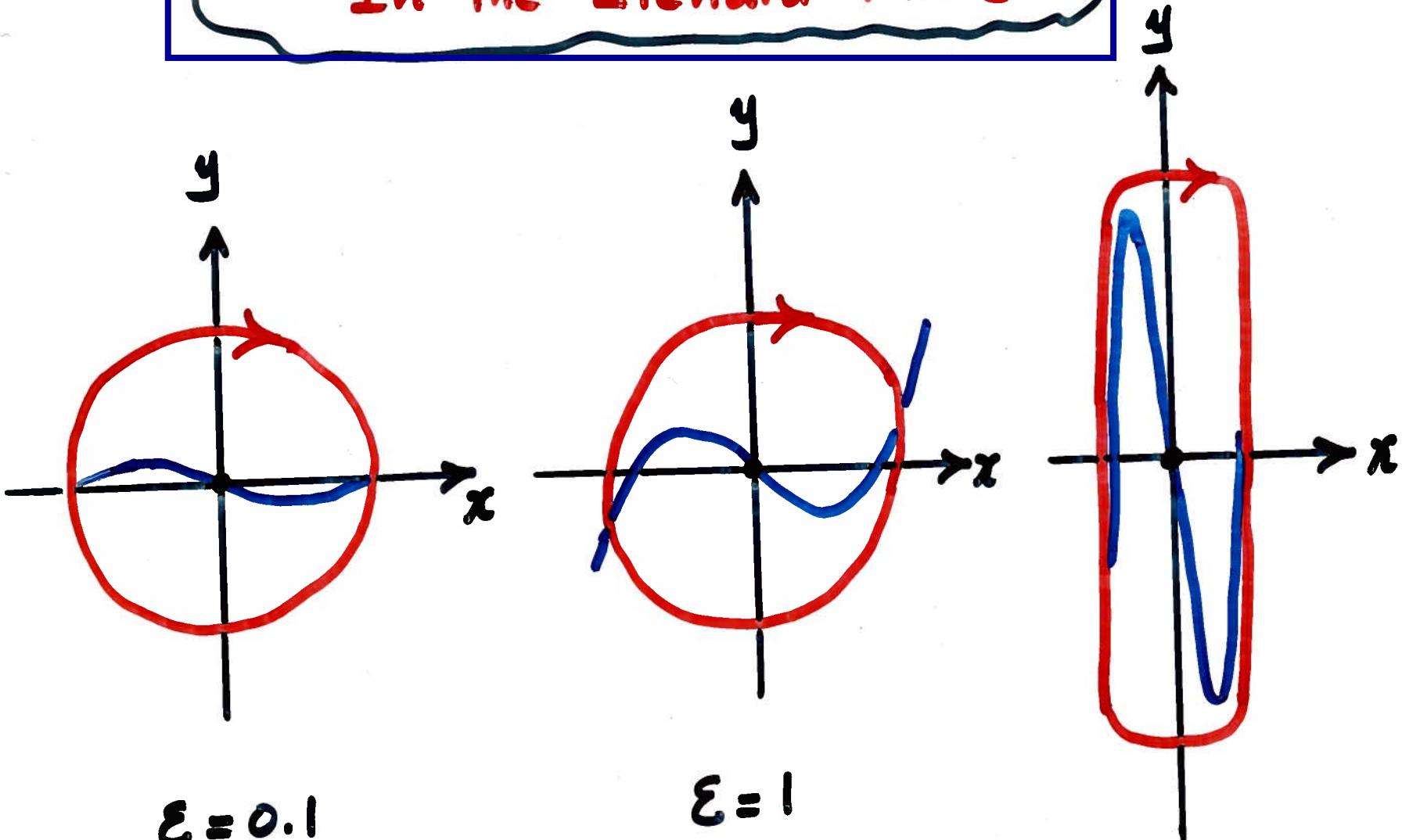
This can be simplified by letting

$$t' = \omega t \quad \text{and} \quad \varepsilon = \frac{\alpha}{\omega}$$

$$\therefore x' = \varepsilon \left(y - \frac{x^3}{3} + x \right)$$

$$y' = -\frac{1}{\varepsilon} x$$

In the Liénard Plane



$$\epsilon = 0.1$$

$$\epsilon = 1$$

$$\epsilon = 10$$

(ii) Bonhoeffer - Van der Pol (BVP) Oscillator Dynamics

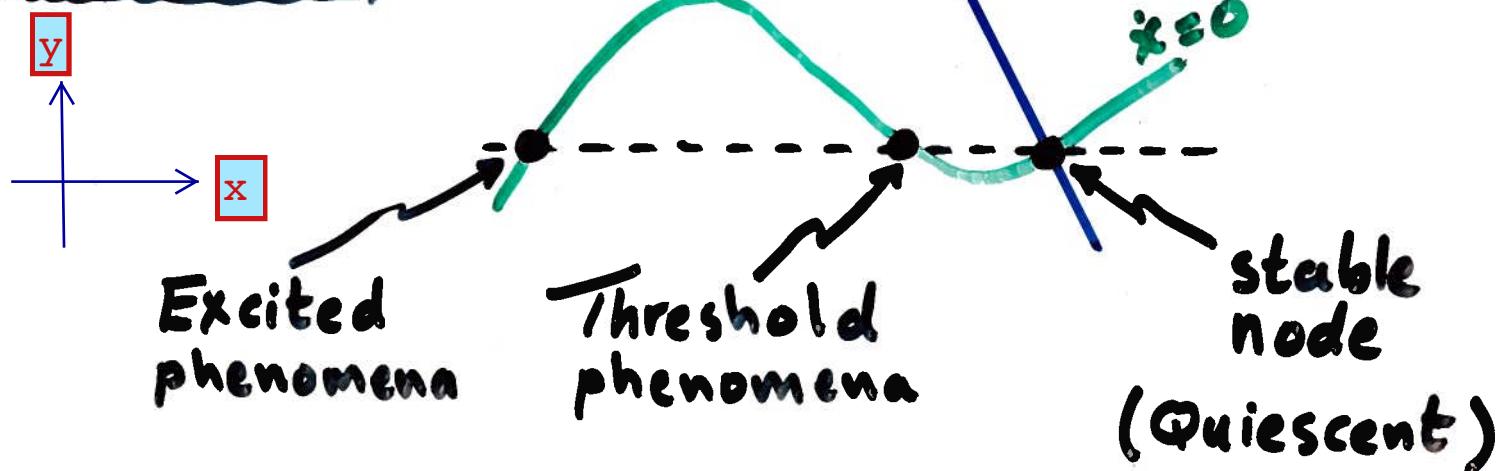
The Van der Pol equation (in the Liénard plane) can be generalized as follows

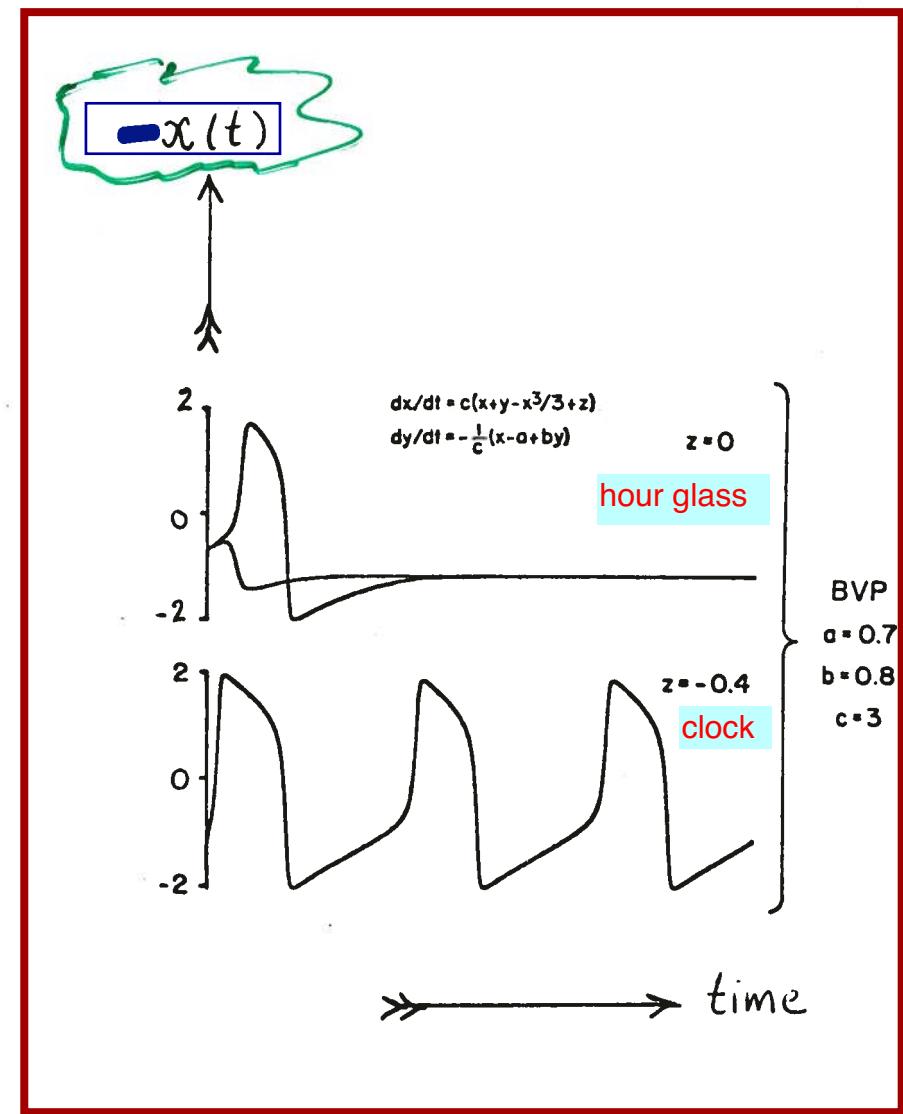
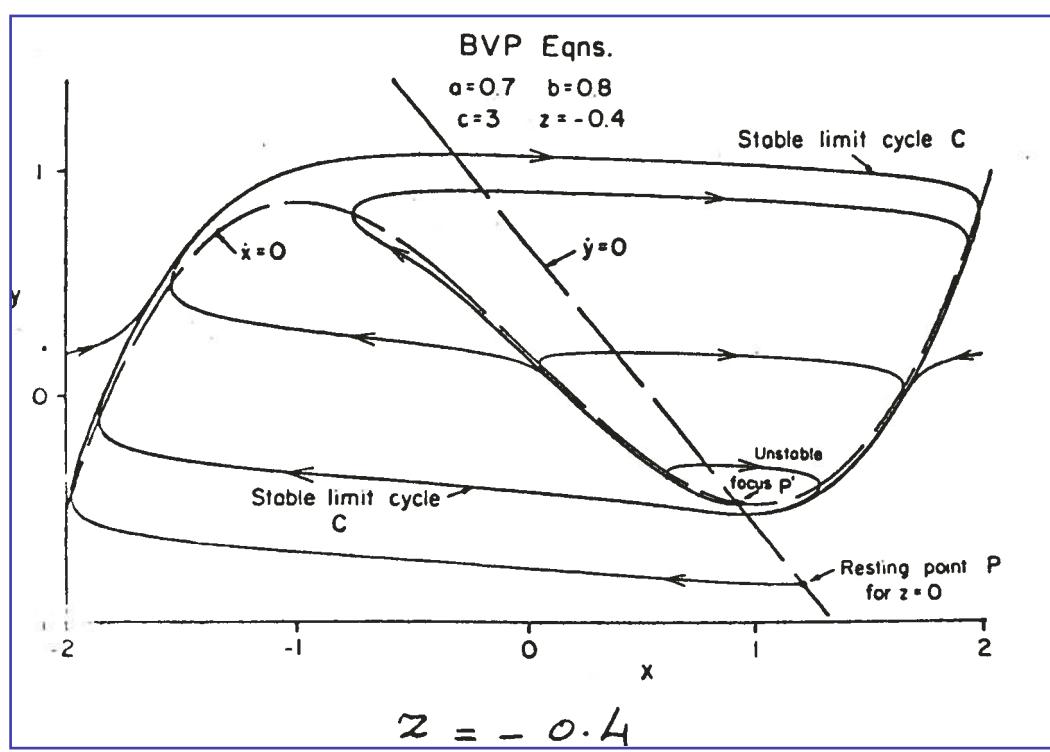
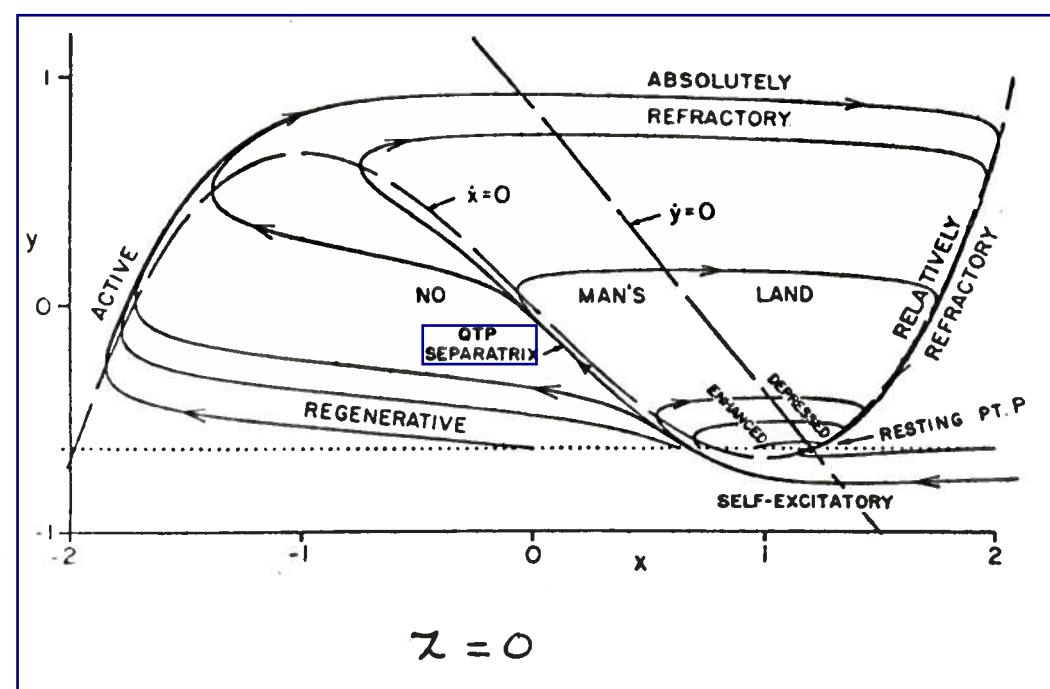
$$\begin{aligned}\dot{x} &= \alpha \left[y + x - \frac{x^3}{3} + z \right] \\ \dot{y} &= -\frac{1}{\alpha} \left[\omega^2 x - a + by \right]\end{aligned}$$

where

α, ω^2, a, b are model parameters
 z is stimulus intensity

For $z=0$



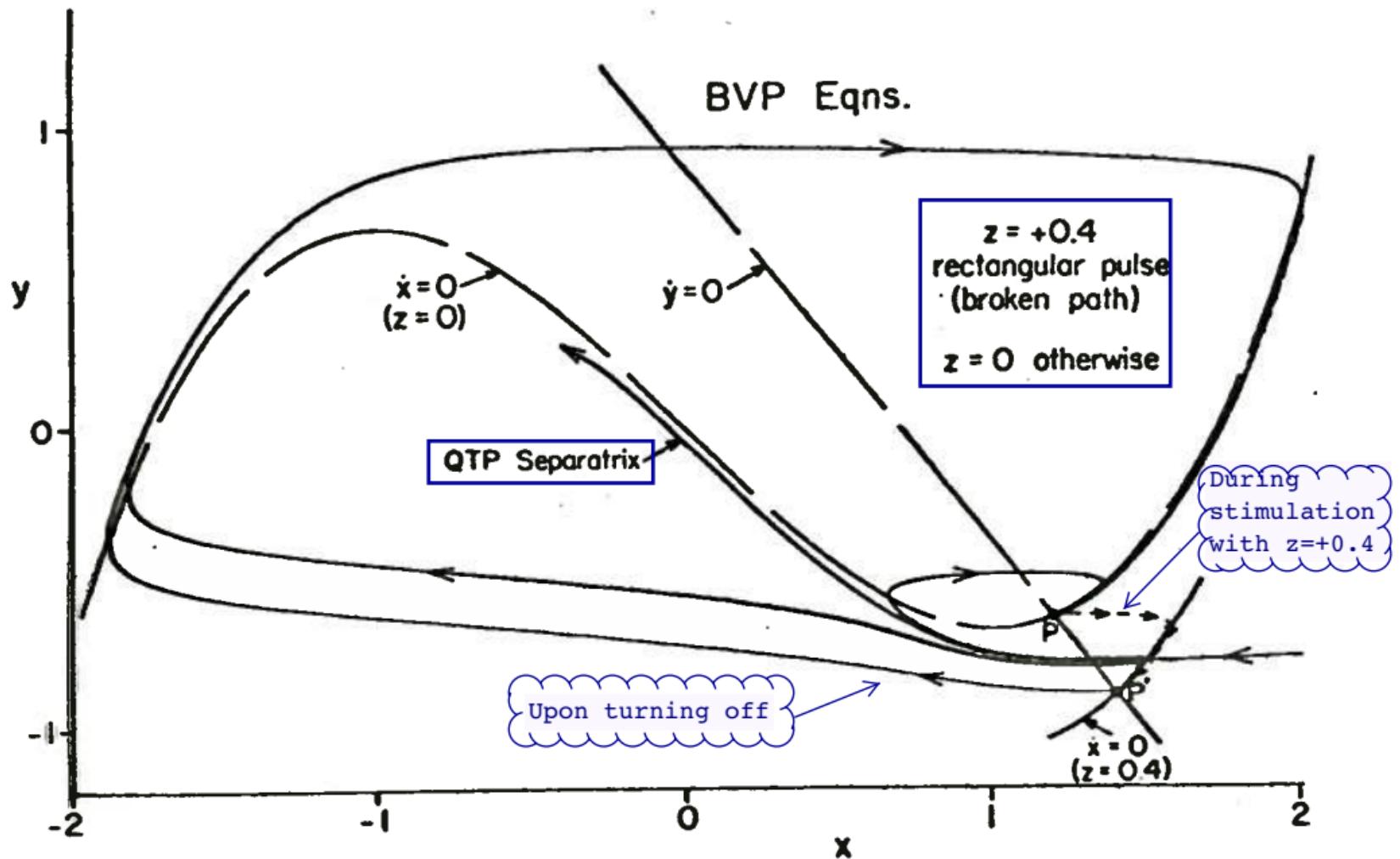


* The Quasi Threshold Plateau (QTP)
Separatrix permits intermediate responses between the all and none responses (hence it is quasi).

* x is the excitability variable
 y is the refractoriness variable

>If the stimulus intensity "z" is of short duration, then it will move the state point along the x -direction but will not change the state plane portrait. On the other hand, long duration pulses alter the topology of the state plane portrait.

* Along the $y=0$ line, the singular point moves upward for $z < 0$ and downward for $z > 0$. (For Long duration pulses).



x The classical phenomenon of anodal break excitation :

A constant anodal current (positive z) moves the $\dot{x}=0$ curve downward which moves the singular point downward. When z jumps to zero, the state point will be below the separatrix, and an impulse results.

筠 For BVP oscillator dynamics, there is one singular point which can be either a stable node or an unstable focus.

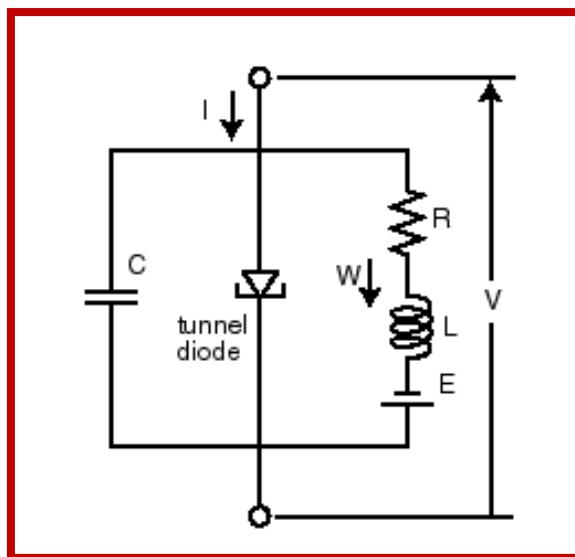
The FitzHugh-Nagumo Model:

$$\begin{aligned}\dot{V} &= V - V^3/3 - W + I \\ \dot{W} &= .08(V + .7 - .8W)\end{aligned}$$

is a two-dimensional simplification of the [Hodgkin-Huxley model](#) of [spike](#) generation in squid giant [axons](#). Here,

- V is the [membrane potential](#),
- W is a recovery variable,
- I is the magnitude of stimulus current.

This system was suggested by [FitzHugh \(1961\)](#), who called it "Bonhoeffer-van der Pol model", and the equivalent circuit by [Nagumo et al. \(1962\)](#)



Circuit diagram of the tunnel-diode nerve model of Nagumo et al. (1962).

The [motivation](#) for the FitzHugh-Nagumo model was to isolate conceptually the essentially mathematical properties of [excitation](#) and propagation from the electrochemical properties of [sodium](#) and [potassium](#) ion [flow](#). The model consists of

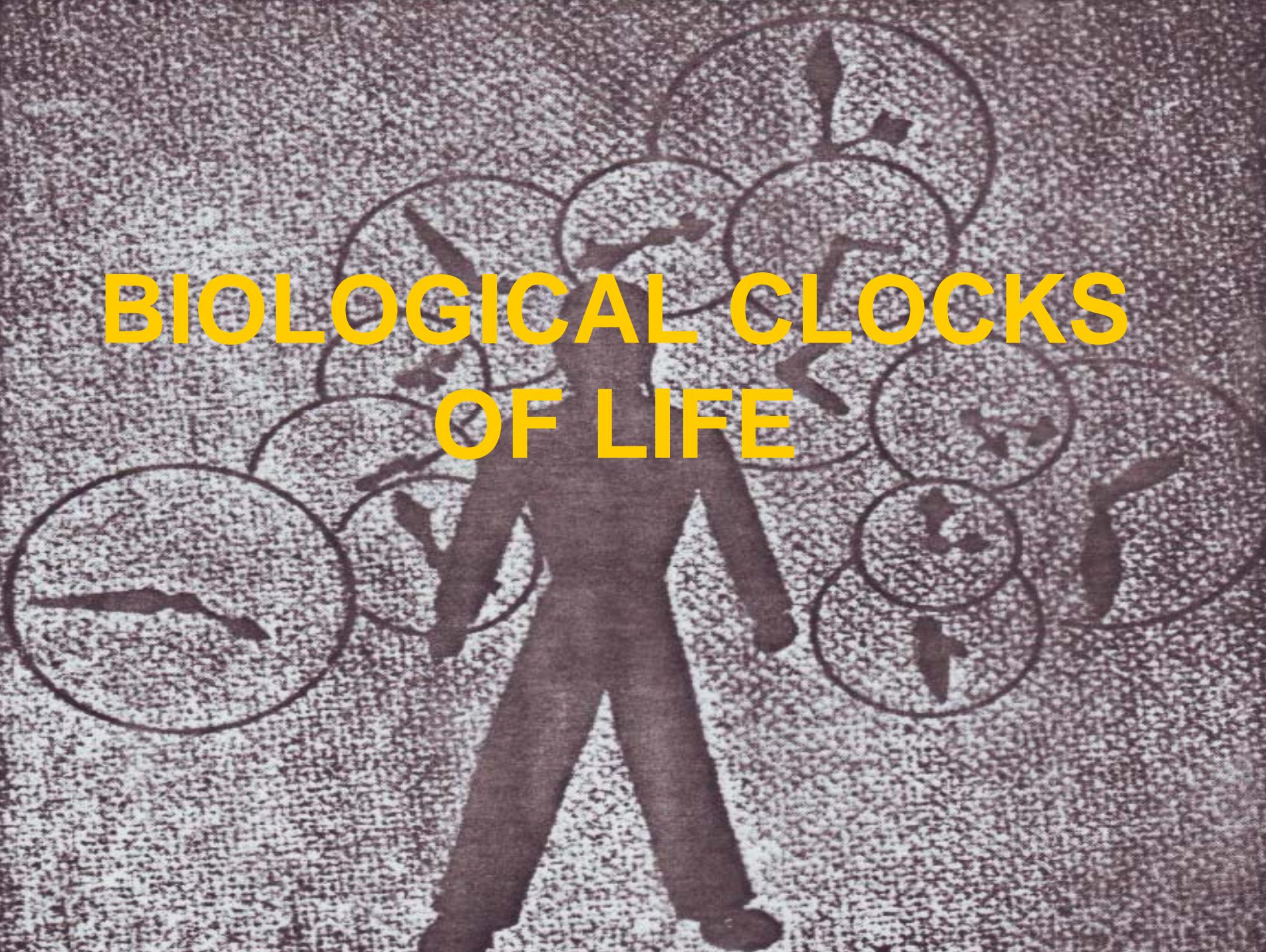
- a *voltage-like variable* having cubic nonlinearity that allows regenerative self-excitation via a [positive feedback](#), and
- a *recovery variable* having a linear dynamics that provides a slower [negative feedback](#).

The model is sometimes written in the abstract form

$$\begin{aligned}\dot{V} &= f(V) - W + I \\ \dot{W} &= a(bV - cW)\end{aligned}$$

Where

$f(V)$ is a [polynomial](#) of third degree, and a , b , and c are constant parameters.



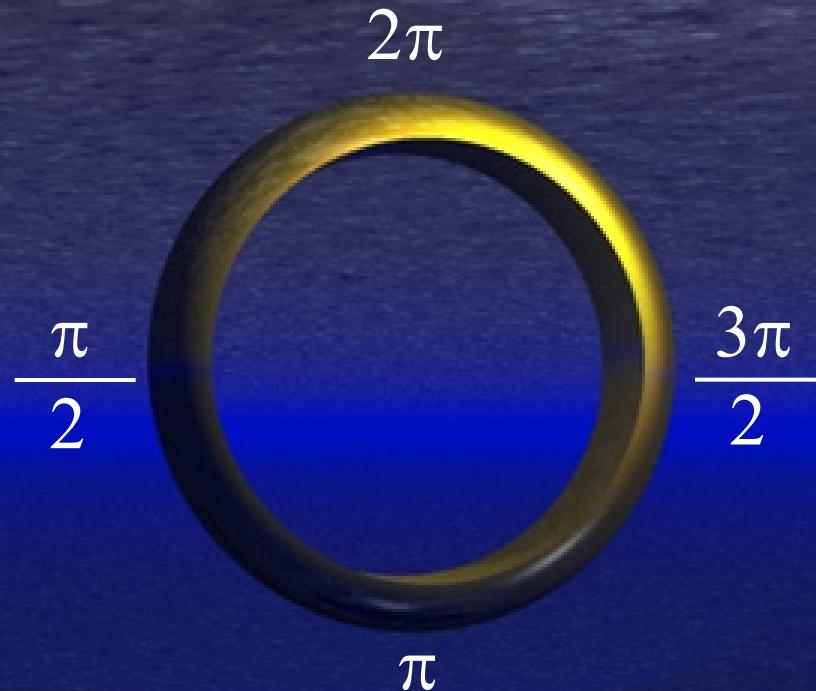
BIOLOGICAL CLOCKS OF LIFE

"In living systems, as in much of mankind's energy-handling machinery, rhythmic return through a cycle of change is an ubiquitous principle of organization".



A. T. Winfree

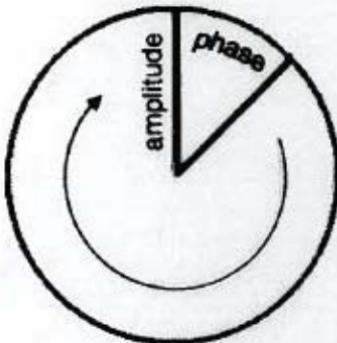
Ring Devices



A continuum of states is represented as real numbers $(0, 2\pi]$

Arthur Winfree proposed **ring devices** to describe amplitude and phase dynamics to characterize intrinsic biological rhythms.

Circle of life

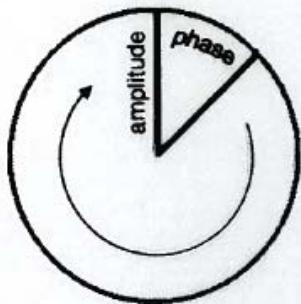


"The circle is the first, the most-simple, and the most perfect figure."

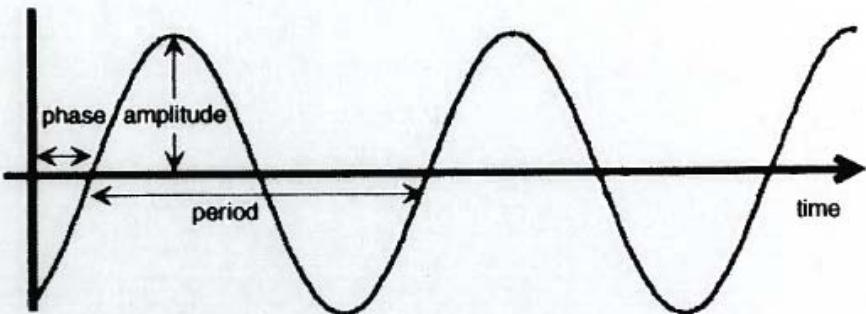
Proclus

Commentary on the first book of Euclid's Elements

Circle of life



Pantha rei



Oscillations illustrate the orthogonal relationship between frequency and time and space and time. An event can repeat over and over, giving the impression of no change (e.g., circle of life). Alternatively, the event evolves over time (*pantha rei*). The forward order of succession is a main argument for causality. One period (right) corresponds to the perimeter of the circle (left).

old

Pantha rei \triangleq everything flows

old

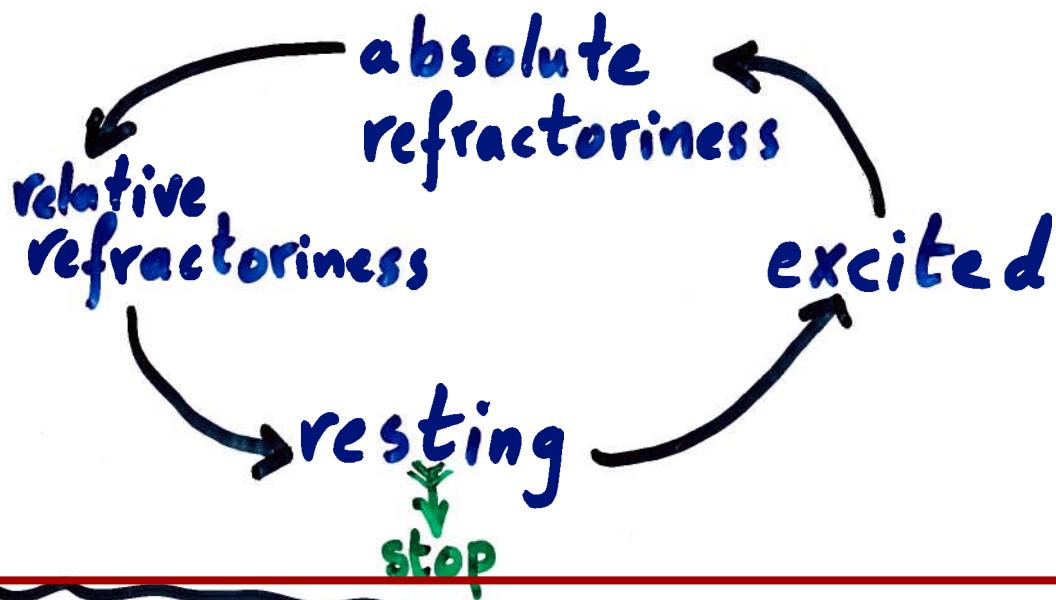
Heraclitus's doctrine: Change is central to the universe.

"You cannot step twice into the same river".

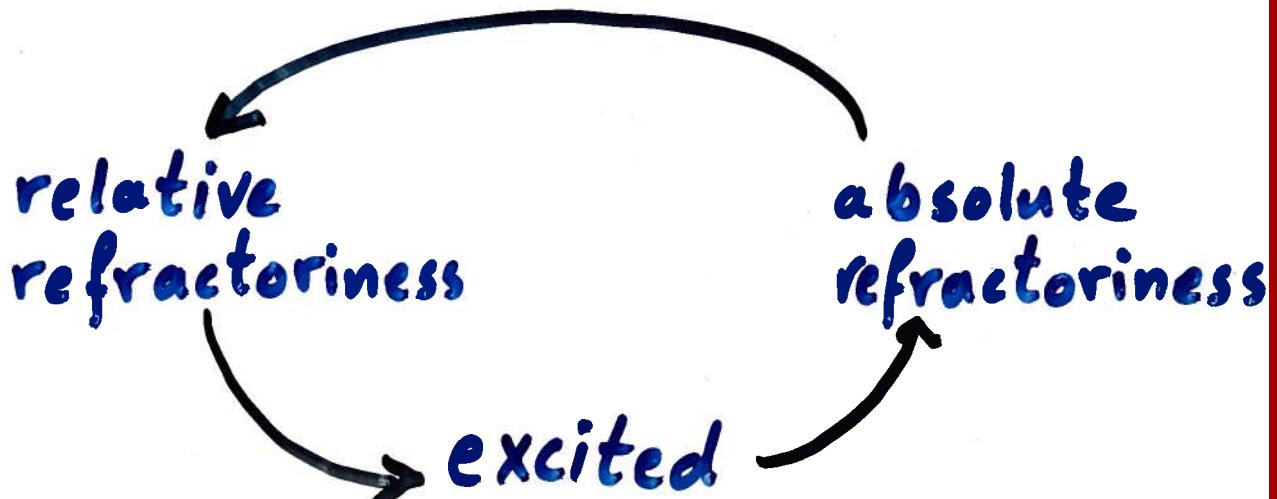
RING DEVICES

I. Hourglasses

For example , nerve cells



II. Clocks



* The states of an hourglass or a clock constitute a **ring**. An hourglass needs to be "turned over" to "tick" since the first and last state is a resting state. A clock "turns itself over" since the first and last state, in a cycle, is not a resting state.

* A ring device runs on a fixed cycle.

* Ring devices can be either hourglasses or clocks according to external conditions. For example, a nerve cell may linger in a resting state until a current pulse induces it to fire an action potential, but if "continually" biased, it fires spontaneously and rhythmically.

* A ring device's rate of advance through its cycle is conditioned by an external influence.

$$\dot{\Phi} = v(\Phi, I)$$

instantaneous
phase

intensity of
external influence

Consider a simple model

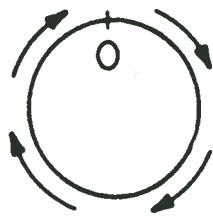
$$v(\Phi, I) = 1 + I \cos 2\pi \Phi$$

then the ring device is a clock
for $v > 0$ and an hourglass
for $v \leq 0$.

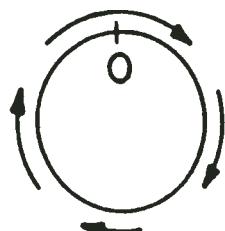
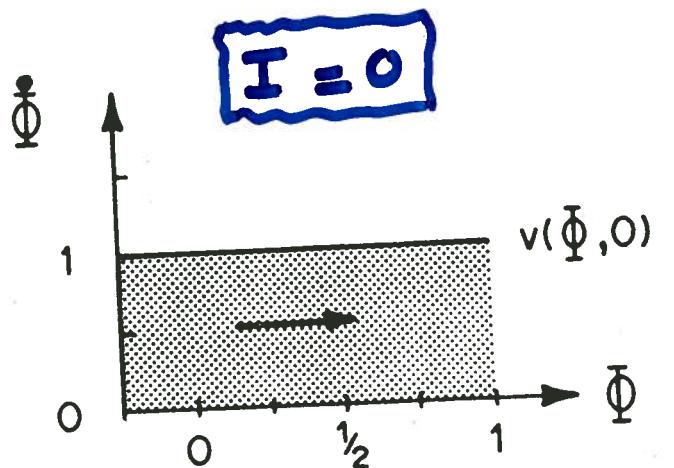
For $-1 < I < 1 \Rightarrow$ clock

otherwise \Rightarrow hourglass

$$\dot{\Phi} = v(\Phi, I) = 1 + I \cos 2\pi \Phi$$

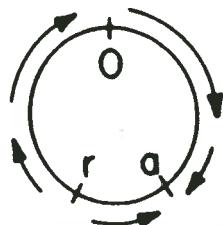
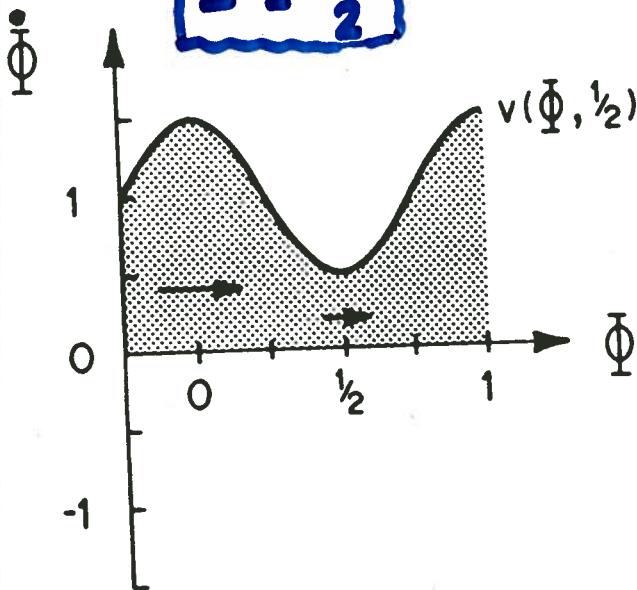


clock



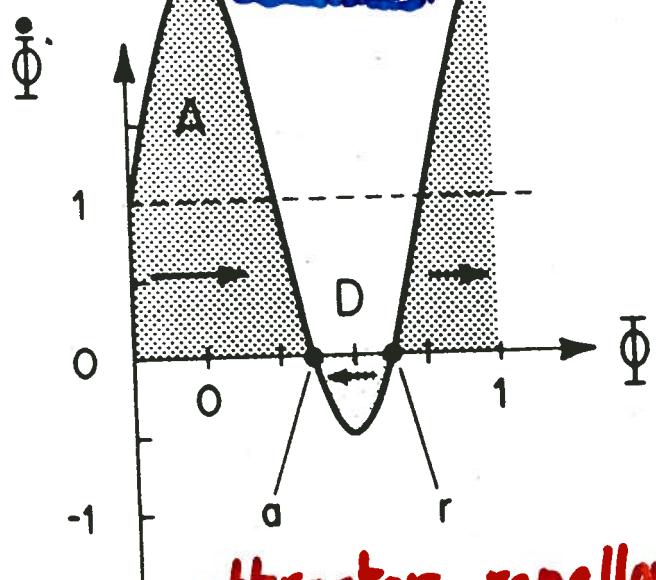
clock

$$I = \frac{1}{2}$$



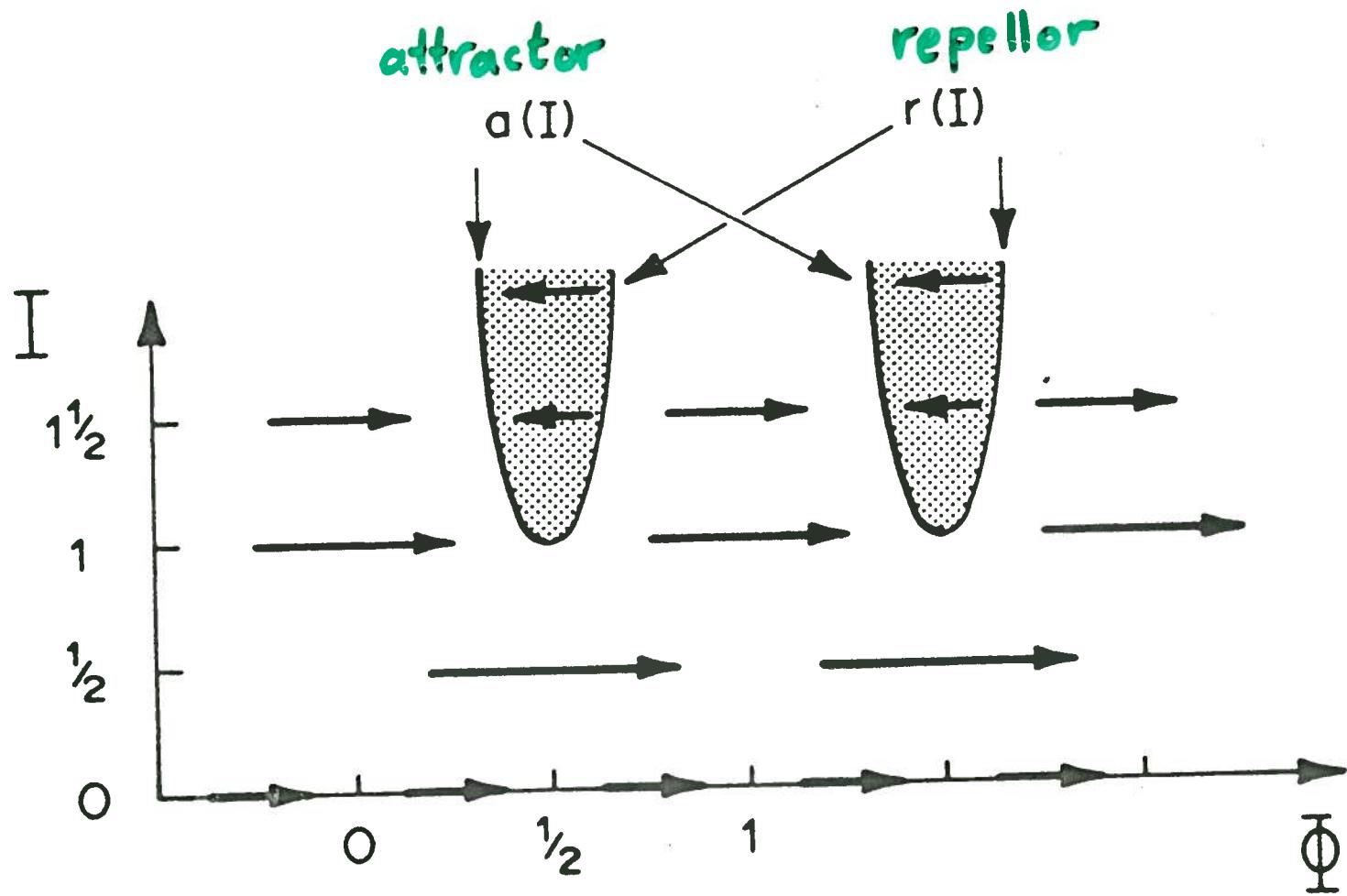
hourglass

$$I = 1 \frac{1}{2}$$



attractor-repellor
pair

The zones of negative angular velocity " ν "



🎵 The phase will "stick" on the any island of negative " ν ".

attractor (or upwind) shore of

Phase - Resetting Curves

$$\dot{\Phi} = v(\Phi, I)$$

$$\int_{\Phi}^{\Phi'} \frac{d\Phi}{v(\Phi, I)} = \int_0^M dt \quad (\text{for } I \neq 0)$$

$$\Phi' = f(\Phi, M, I)$$

final phase

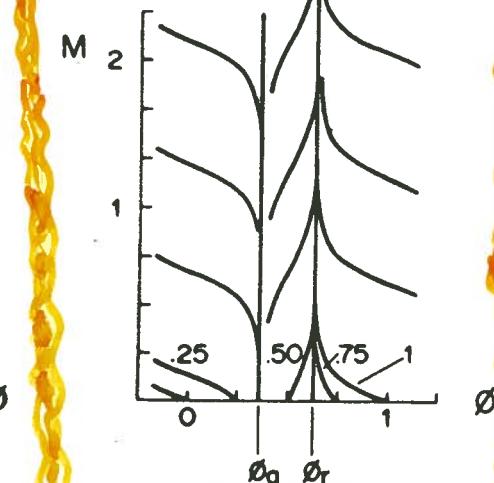
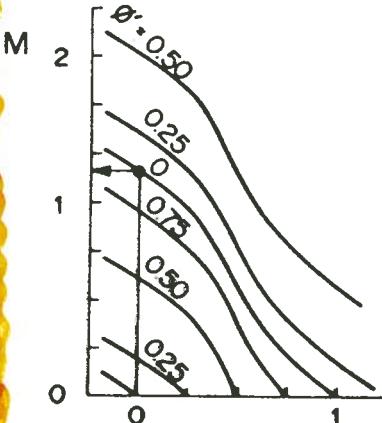
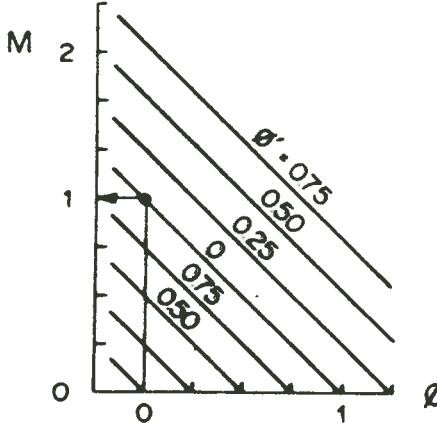
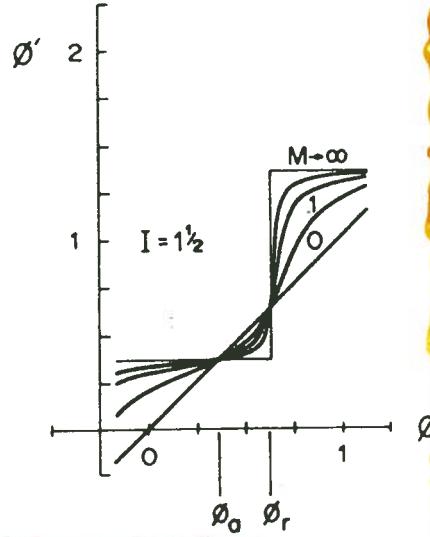
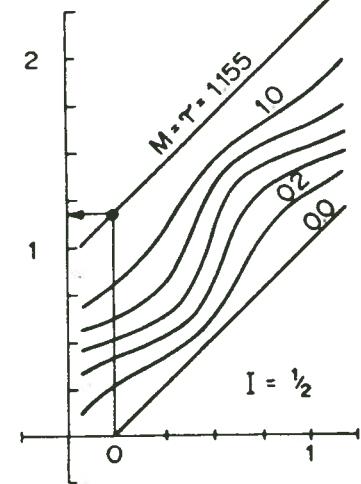
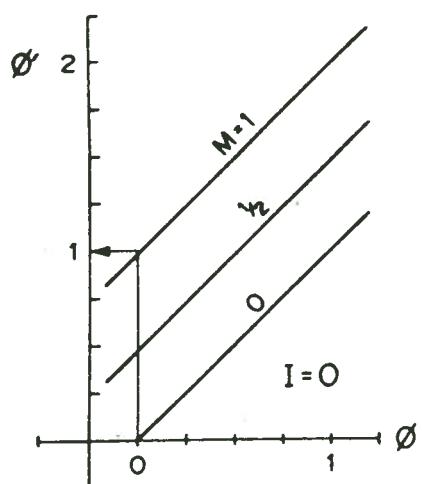
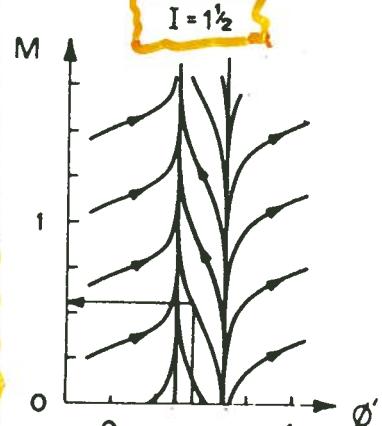
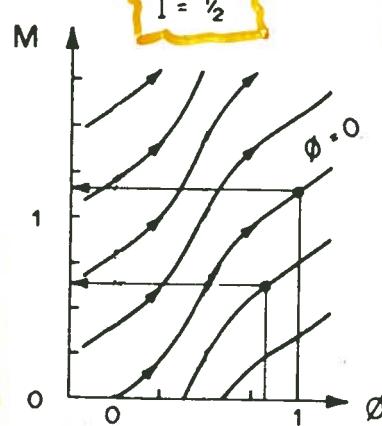
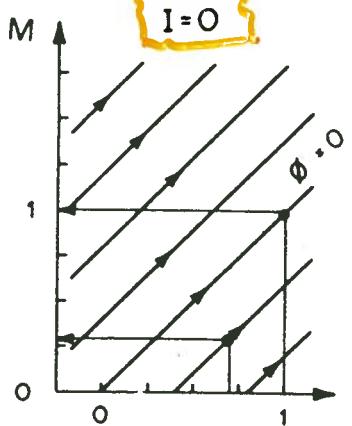
initial phase

stimulus duration

stimulus intensity

Consider $v(\Phi, I) = 1 + I \cos 2\pi \Phi$

- ⇒ I represents deviations from standard conditions.
- ⇒ M, ϕ, ϕ' are measured as fractions of the unperturbed cycle duration.
- ⇒ For each setting of I, contour maps of ϕ, M , and ϕ' can be drawn.



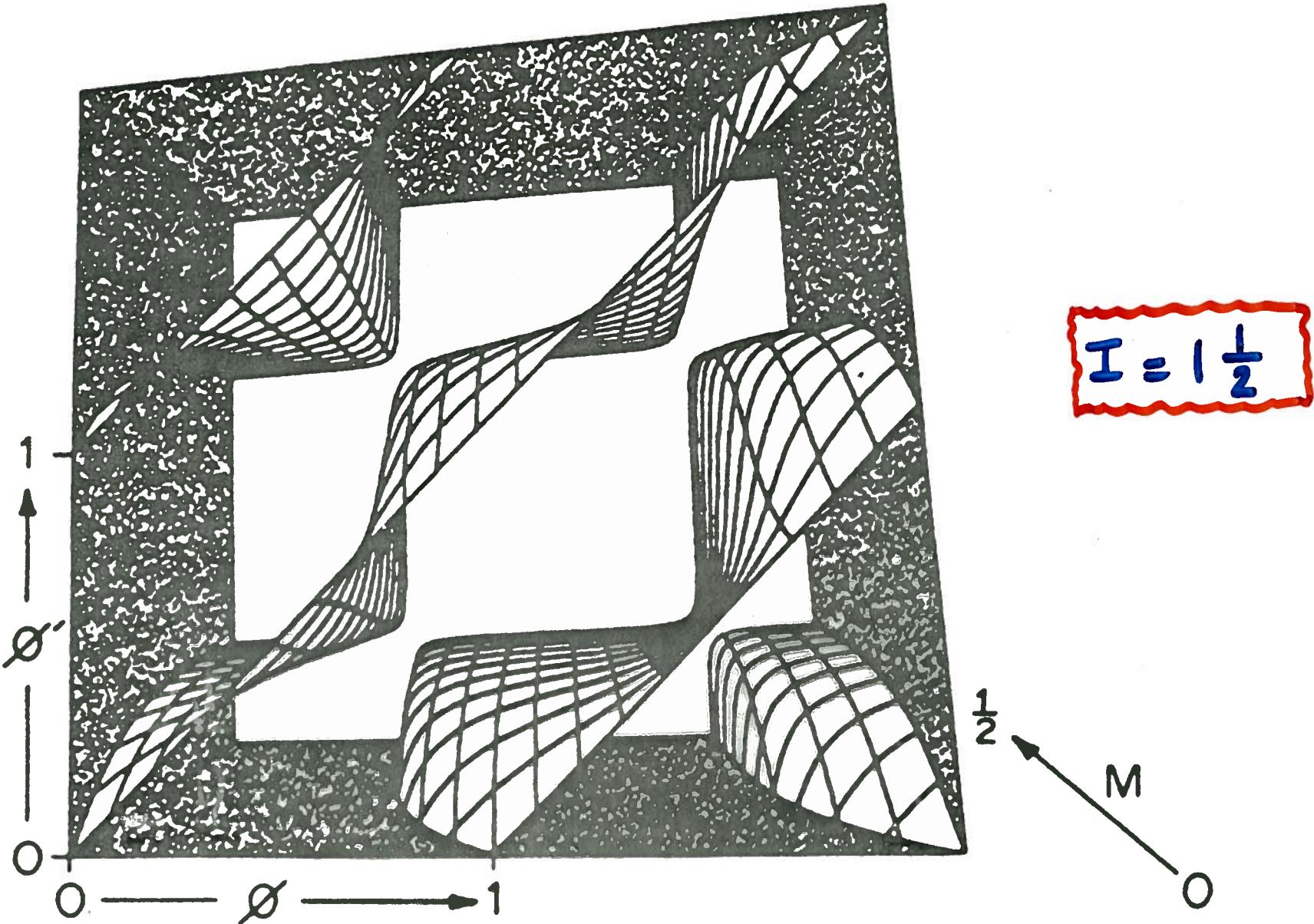
(a)

(b)

(c)

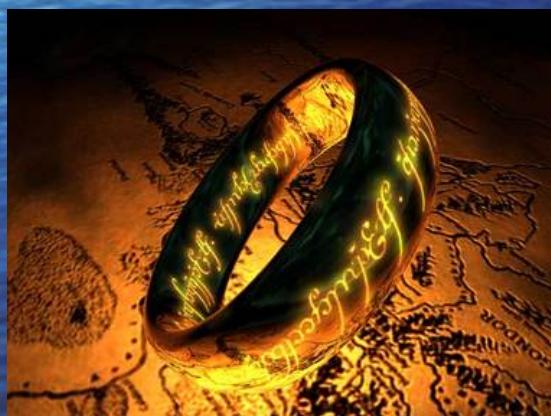
Contour maps of ϕ , M and ϕ'
for different settings of I .

Phase-Resetting Surfaces

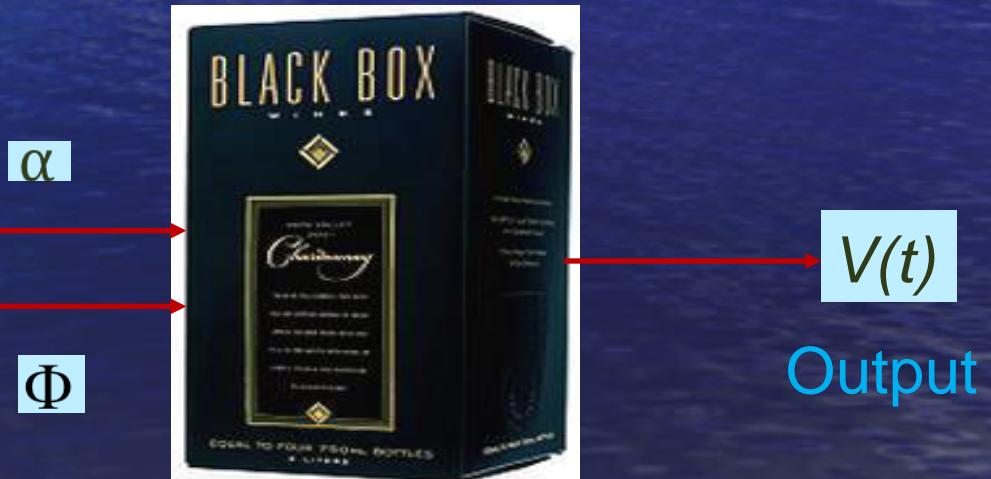


Rhythm Generators

- The dynamics of Ring Devices are described in terms of phase and amplitude.
- Changes in phase or amplitude reflect changes in state.
- The instantaneous state can be ‘mapped’ to a physical quantity, such as voltage, using a **static nonlinearity**.



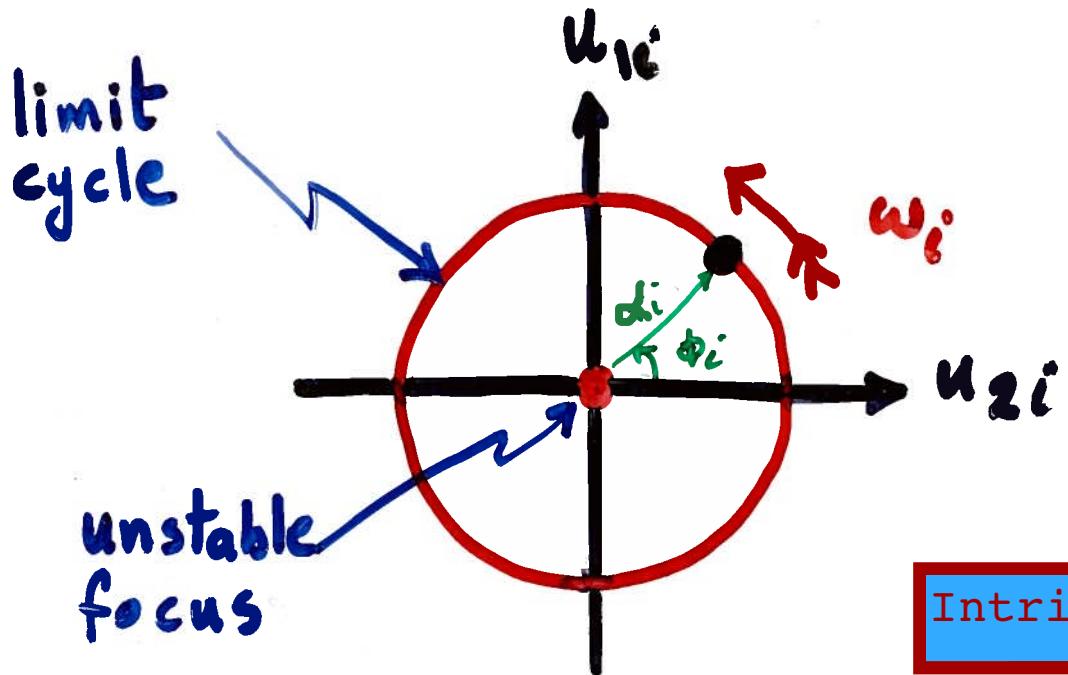
Ring Device
(dynamic ring of states)



Mapper

$V(t)$
Output

Synthesis of Nonlinear Oscillator Dynamics



Intrinsic Clock

$$\dot{\alpha}_i = \omega_i \alpha_i (1 - \alpha_i^2)$$

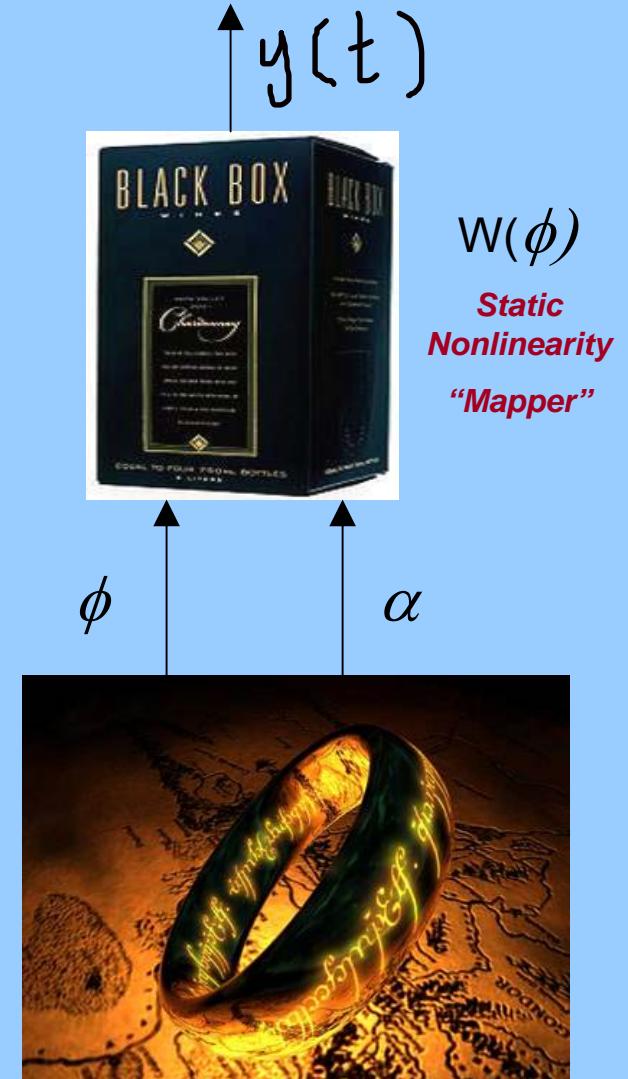
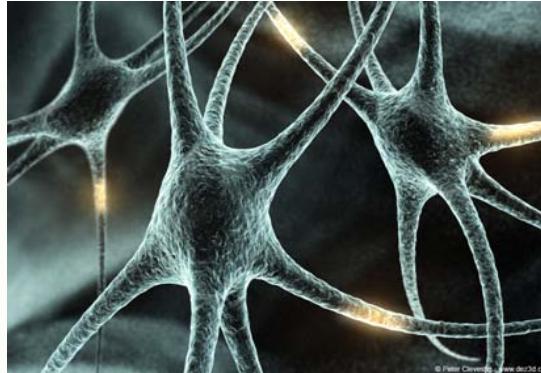
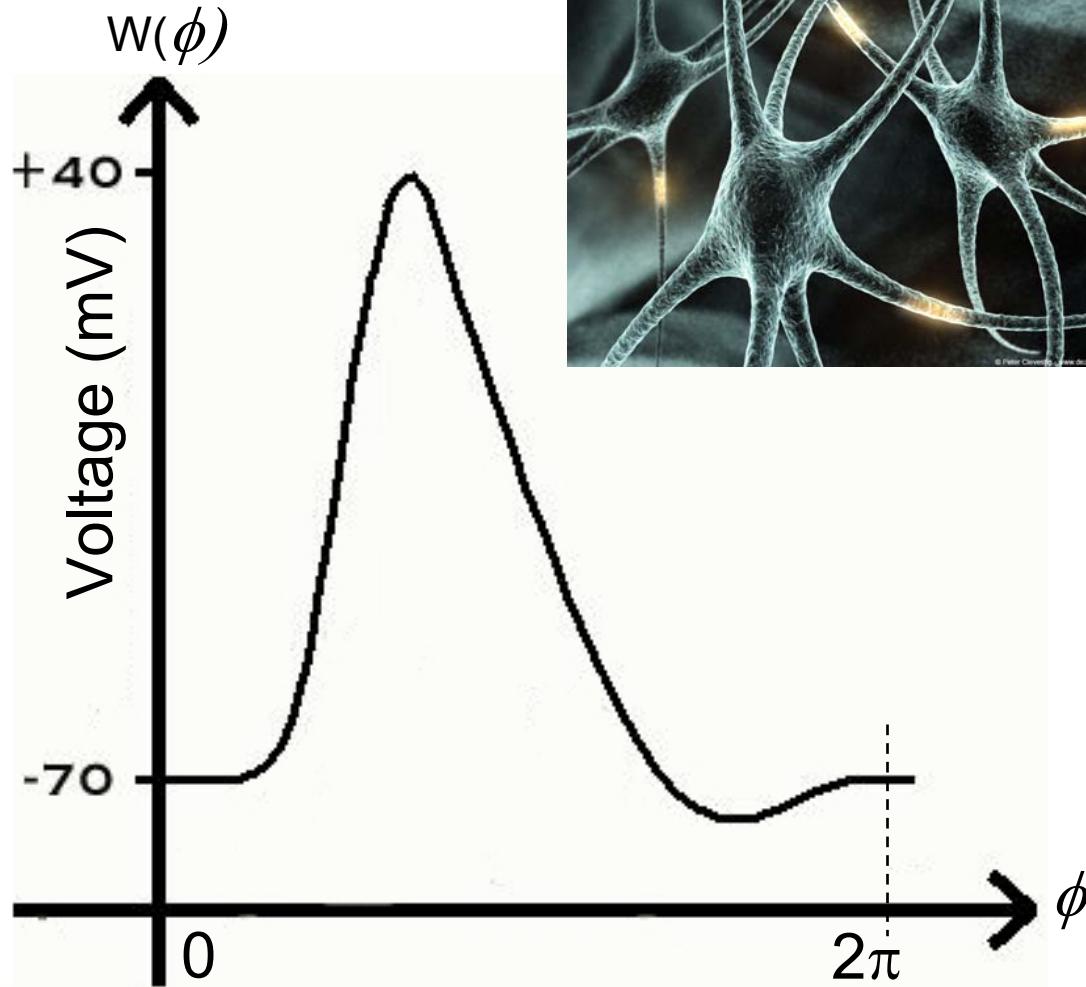
$$\dot{\Phi}_i = \omega_i$$

$$\dot{\alpha}_i = \begin{cases} < 0 & ; \alpha_i > 1 \\ = 0 & ; \alpha_i = 0, 1 \\ > 0 & ; 0 < \alpha_i < 1 \end{cases}$$

Excluding $\alpha_i = 0$ all other solutions approach $\alpha_i = 1$ as a limit. Hence unit circle is a limit cycle and the origin is unstable focus.

Neural Rhythms

The action potential as a cycle



Stimulated Clock

$$\dot{\alpha} = \omega_i \propto (1 + Z_\alpha - \alpha^2)$$
$$\dot{\phi} = \omega_i (1 + Z_\phi)$$

where Z_α & Z_ϕ are input stimuli

Output of Mapper

$$y(t) = y_0 + \alpha W(\phi)$$

$$y_0 = S_y$$

The Refractory Function

$$R = \frac{1}{\sqrt{1 + \left[\frac{2\pi \hat{r}}{\phi} \right]^{2N}}}$$

High pass phase filter

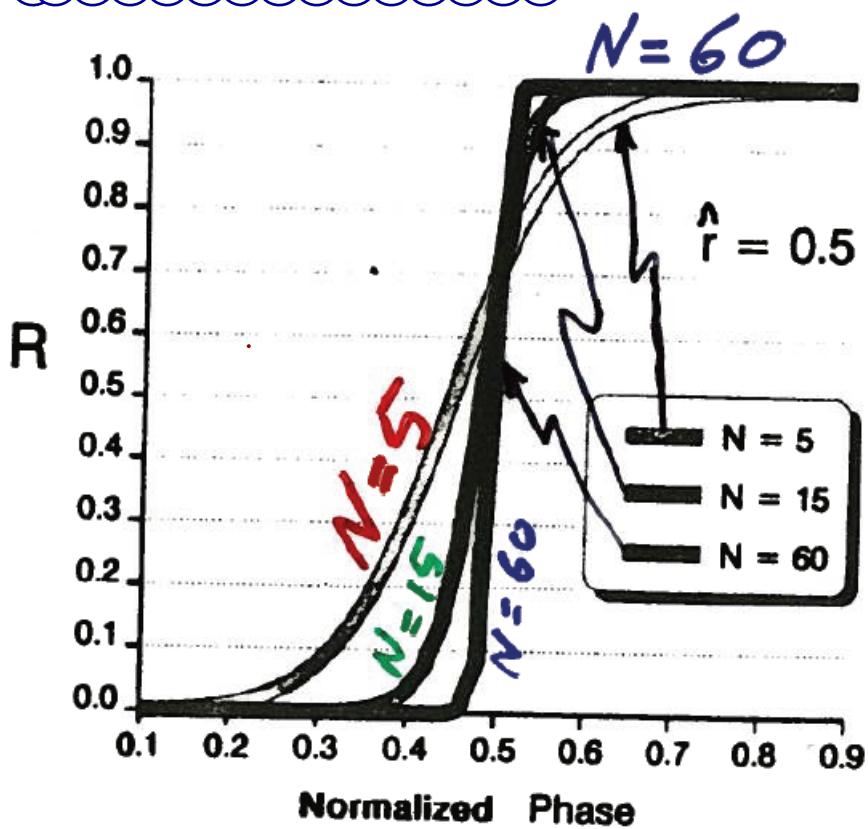
could be a Butterworth or sigmoidal function of phase.

Effective Stimuli:

$$Z_\alpha = R S_\alpha ; Z_\phi = R S_\phi$$

Input
Stimuli

S_α
 S_ϕ
 S_y



clock

$$\frac{d\alpha}{dt} = k_\alpha \alpha (1 + R_\alpha(\phi) S_\alpha - \alpha^2),$$

labile clock

$$\frac{d\alpha}{dt} = k_\alpha \alpha^{1/3} (R_\alpha(\phi) V(S_\alpha) - \alpha),$$

$$\frac{d\phi}{dt} = \omega (1 + R_\phi(\phi) S_\phi)$$

$R_\alpha(\cdot)$ is a refractory function

$R_\phi(\cdot)$ is a refractory function

$V(\cdot)$ is a threshold function controlling activation
of the labile clock

The implicit ring device functions $R_\phi(\cdot)$, $R_\alpha(\cdot)$ and $V(\cdot)$ are all sigmoids of the form

$$s(u) = \frac{1}{1 + \exp(-c_1(u - c_2))}$$

mapper

$$y = S_y + \alpha W(\phi)$$

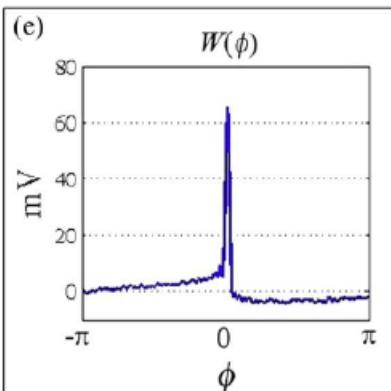
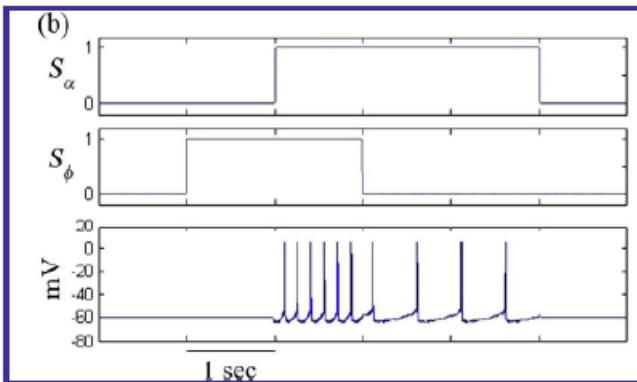
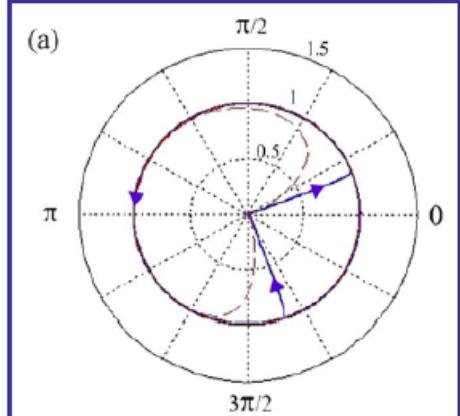
Ring Device

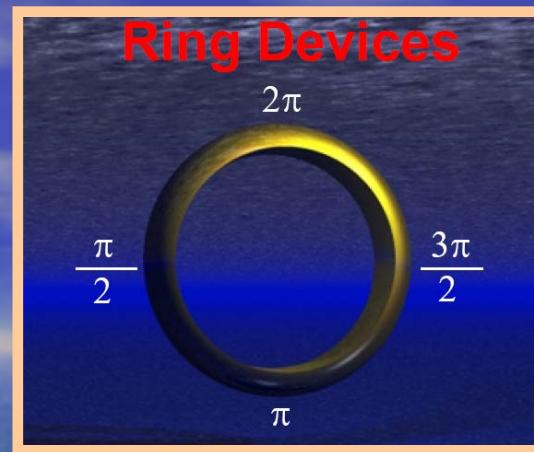
Labile Clock

(a) Labile clock ring dynamics: a suprathreshold alpha stimulus (red segmented line) forces the state trajectory (blue solid line) away from the origin and toward its active limit cycle (the threshold is 0.5). The radius of the limit cycle is not determined by the stimulus magnitude but by the threshold function. When the stimulus is removed, the state dot returns to its orbit near the origin.

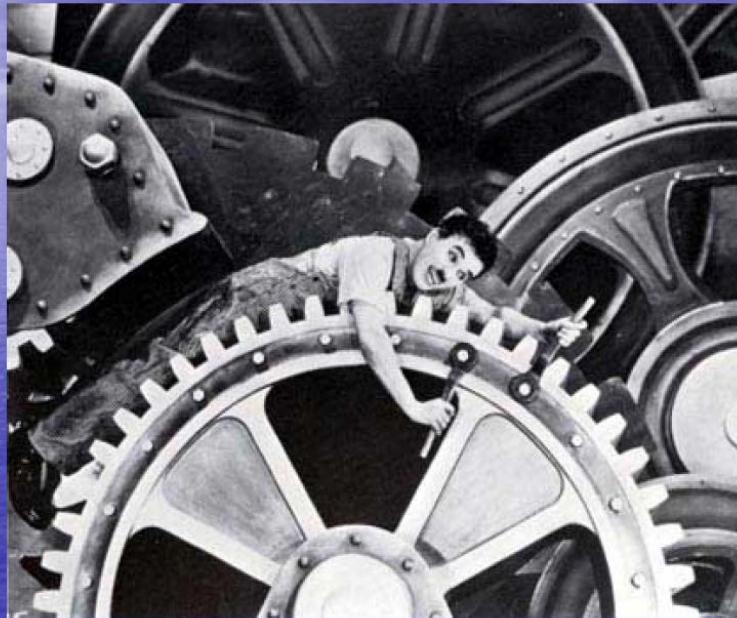
(b) Stimuli S_{α} and S_{ϕ} are administered in staggered fashion. The frequency-modifying effect of the phase stimulus is not registered unless the labile clock is in its active state, which is brought about by the suprathreshold amplitude step.

(e) The intrinsic waveform of the static nonlinearity is the recorded biological source waveform normalized in phase over one cycle.

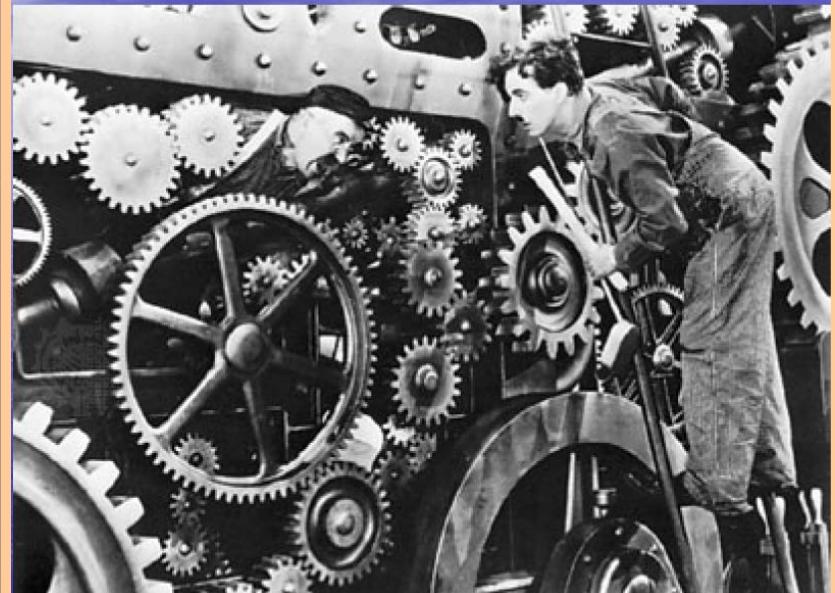


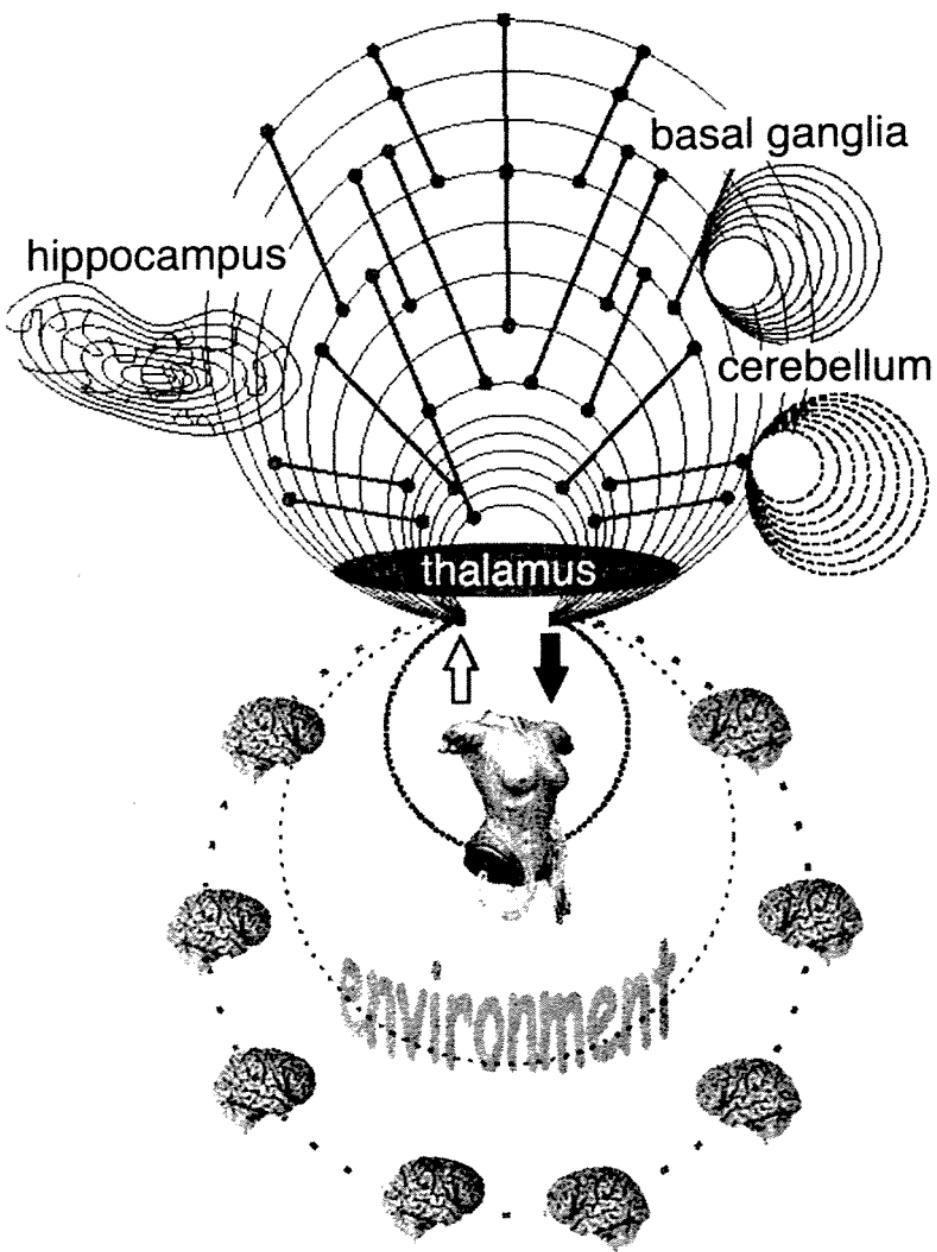


Ring Parameters can be adjusted



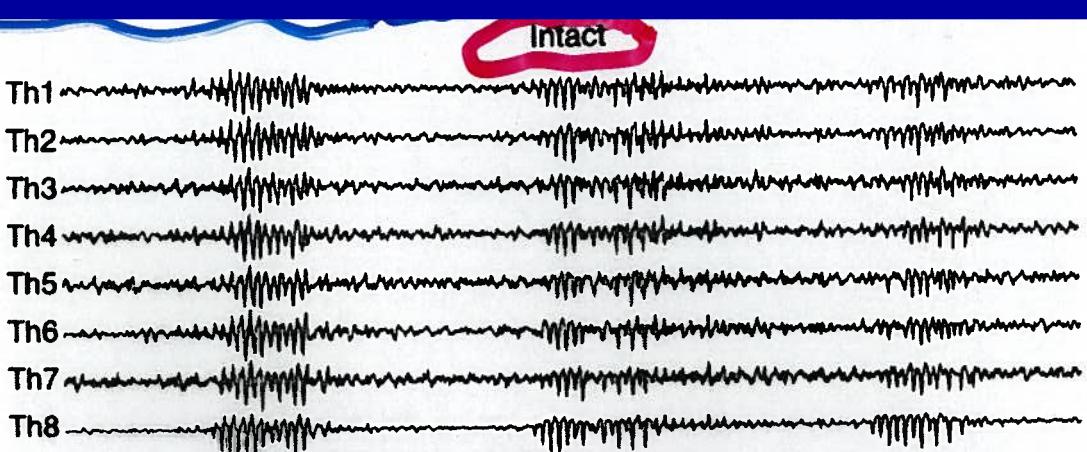
Rings can be coupled



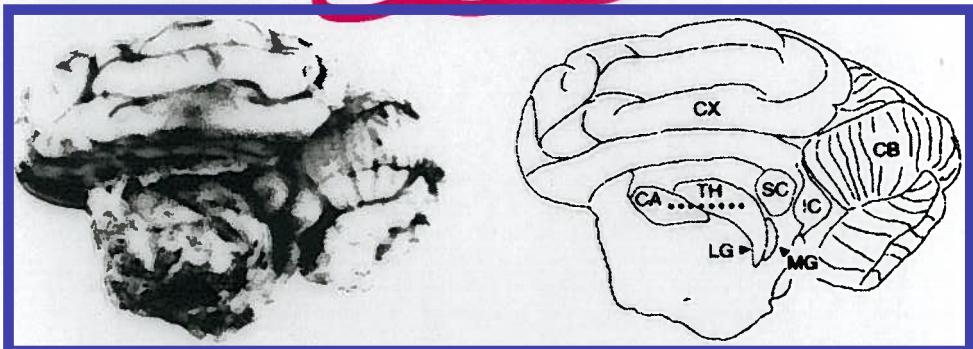


Barbiturate-induced spindles in the thalamus of the cat brain

Entrained

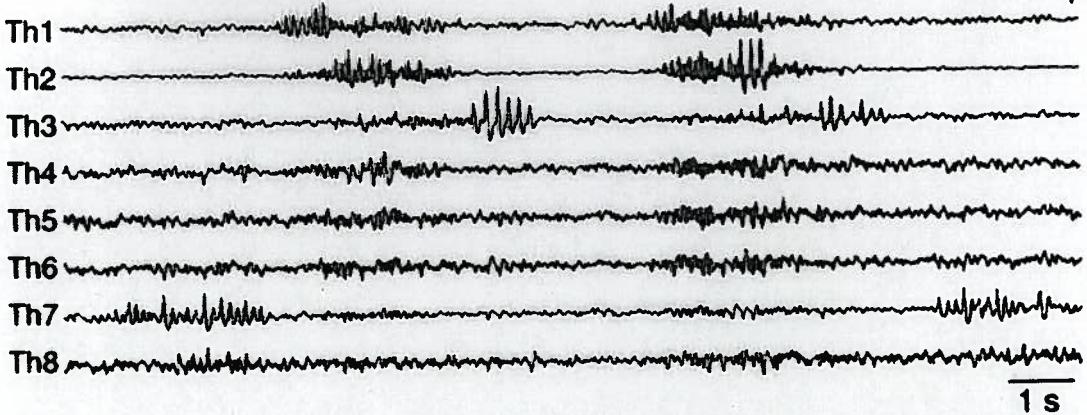


Decorticated



200 μ V |

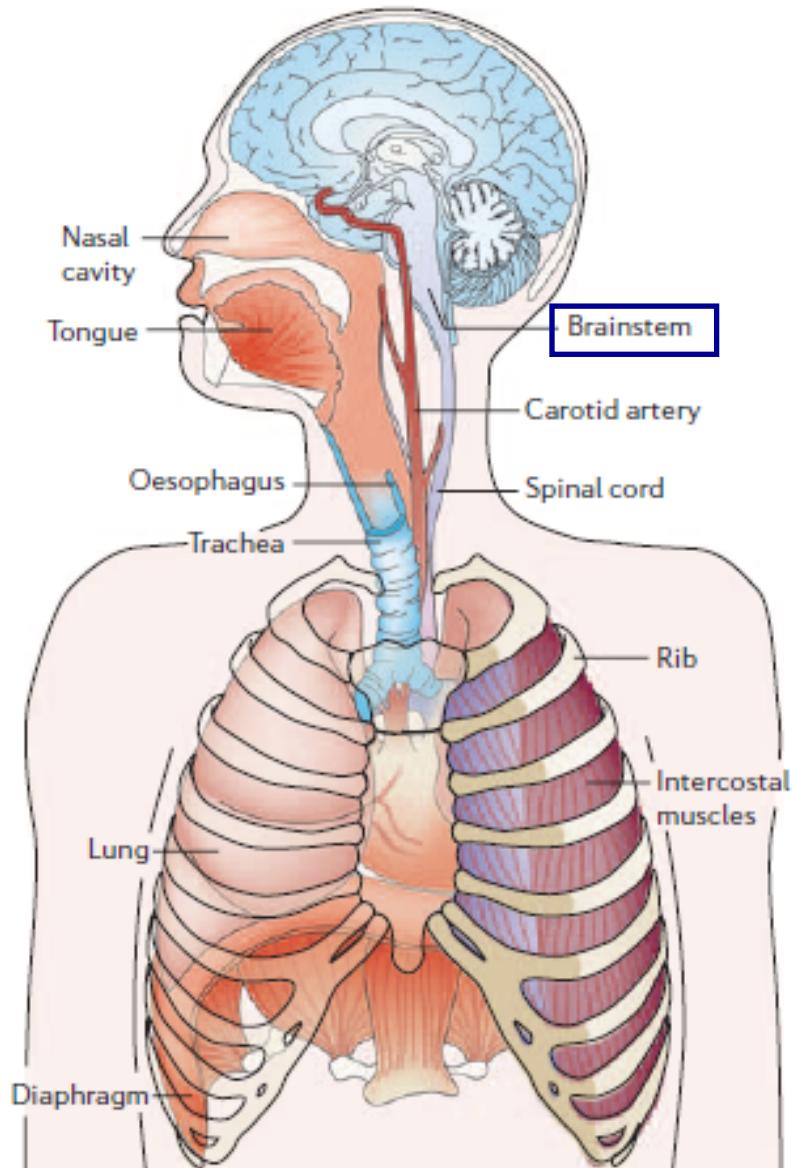
Not
entrained



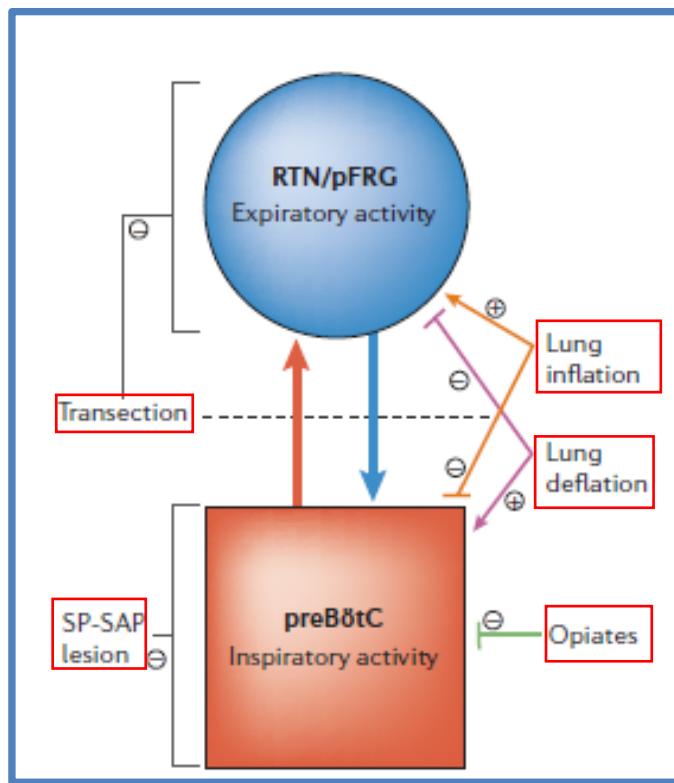
Cortical long-range connections synchronize thalamic activity. Top: Barbiturate-induced spindles (8–9 hertz, lasting for 1–3 seconds) are synchronous at multiple site in the thalamus (black dots in inset indicate recording sites Th1–Th8). Bottom: Following decortication, spindling continued to occur at each electrode site, but their temporal synchrony was disrupted. Reprinted, with permission, from Contreras et al. (1996).

Spindles ≡ Brain wave bursts

Anatomy and physiology of the respiratory system



The Respiratory Rhythmogenesis in the Mammalian Brainstem



There are two oscillators that are differentially affected by various inputs, such as opiates and lung inflation and deflation. The more rostral oscillator is located in the region of the **retrotrapezoid nucleus/parafacial respiratory group (RTN/pFRG)** and appears to drive active expiratory activity. It might not be rhythmic in mammals at rest, when there is little or no active expiration. The more caudal oscillator is located in the **preBötziinger Complex (preBötC)** and appears to drive inspiratory activity. Substance P-saporin (SP-SAP) lesion of preBötC neurokinin1 receptor (NK1R) neurons disrupts breathing. Transections between the two oscillators disrupt expiratory motor outflow, while inspiratory activity continues unabated. Lung inflation enhances the activity of the expiratory oscillator and depresses the inspiratory oscillator, which serves, ultimately, to reduce lung volume, whereas lung deflation has the opposite effect on the inspiratory and expiratory oscillators.

Fellowship of the Rings

