Notes on The Phase reduction method with emphasis on weakly coupled oscillators as simple neural models.

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July 28, 2000

0.1 Introduction

In this colloqium, I'm trying to summarize some important points of this analytical paradigm invented more or less by Kuramoto (see his book [1]) and developed by Ermentrout and Kopell over the last two decades. When working with dynamical systems, such as coupled oscillators modelling e.g. a neuronal system, the idea is to only consider the relative phases between the different oscillators and not the dynamics of each individual oscillator itself. Thus, we are reducing a complicated dynamical system into one dimension per oscillator, and the claim is that we can still capture the qualitative ensemble dynamics.

This approach is applicable in many physical systems, but has recently gained popularity in chemistry, biology and neuroscience. First, the basic idea.

0.2 The perturbed oscillator

A dynamical system can be written as a set of ordinary differential equations (ODEs):

$$\frac{d\mathbf{X}}{dt} = \mathbf{F}(\mathbf{X}, \mu) \tag{1}$$

in the unperturbed non–interacting case where $\mathbf{X}$ contains all the phase space variables, $\mathbf{F}$ characterizes the dynamics and μ has all the different parameters. Let's assume that that the dynamics is periodic and it has a stable limitcycle in some region of phase space. Thus $\mathbf{X}(t) = \mathbf{X}(T + t)$. We choose to associate a scalar,$\phi(\mathbf{X})$,to every point, $\mathbf{X}$, on the limitcycle and we parameterize is so the value of ϕ increases constantly in time

$$\frac{d\phi}{dt} = \omega, \tag{2}$$

and ω is scaled so $\omega = \frac{2\pi}{T}$. Note that with this definition of the phase doesn't follow the point on the limitcycle in realtime, since the velocity along the trajectory generally is nonlinearly dependent on time – think about the Hudgkin–Huxley neuron or the Van der Pol oscillator which both have at least two different time scales on the trajectory.

Now, we can rewrite the ODE for ϕ in terms of X by a mathematical identity so

$$\frac{d\phi}{dt} = \sum_i \frac{\partial \phi}{\partial x_i}\frac{dx_i}{dt} = \nabla\phi \cdot \mathbf{F}(\mathbf{X}, \mu) \tag{3}$$

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Now, if we introduce a perturbation to the equation of motion, the point is no longer on the limit cycle and the situation is different.

$$\frac{d\mathbf{X}}{dt} = \mathbf{F}(\mathbf{X}, \mu) + \epsilon \mathbf{P}(\mathbf{X}, \mathbf{X}') \tag{4}$$

where the functional $\mathbf{P}$ represents the perturbation as a function of the position in phase space and other dynamical parameters $\mathbf{X}'$ e.g. if the perturbation comes from another oscillator. The scalar ϵ is the parameter we can set to zero to regain the unperturbed motion. Now, the previous definition of the phase is invalid, since the trajectory doesn't close on itself and therefore doesn't parametrize itself. However, we can define a small surface in phase space for which all the points give the same phase lag, when they again reach the limit cycle. These are referred to as *Isochrons*. The isochron will move around with the limit cycle in the same fasion as the phase of the unperturbed oscillator. Therefore, we can now extend the definition of phase to be a scalar for any point on an isochron, C:

$$\frac{d\phi(\mathbf{X})}{dt} = \omega, \ \mathbf{X} \ \epsilon \ C \tag{5}$$

With this extension in the definition of the phase we can now write up the equation including the perturbation and coordinates off limit cycle:

$$\frac{d\phi}{dt} = \nabla\phi \cdot \mathbf{F}(\mathbf{X}, \mu) + \epsilon \nabla\phi \cdot \mathbf{P}(\mathbf{X}, \mathbf{X}') \tag{6}$$

Since we assumed that the limit cycle is stable the perturbation will decay and eventually reach the initial dynamics. However, a pertubation will change the phase compared with the unperturbed motion. How much will the will the oscillator get shifted in phase given a perturbation? Now, the latter equation is still dependent on the exact location in phase space, $\mathbf{X}$, which can be very complicated to calculate. We wish to reduce the problem into an equation only dependent on ϕ and time (so the substitution $\phi(\mathbf{X}) \rightarrow \phi(t)$ is valid) and therefore we make the assumption that the perturbed point is *close* to the unperturbed trajectory. In fact, the perturbed point, denoted $\mathbf{X_p}$, is as close to the unperturbed, denoted $\mathbf{X_0}$, as long as the perturbation is small.

$$\exists \ \epsilon \ \forall \ \delta > 0, \ |\mathbf{X_p} - \mathbf{X_0}| < \delta \tag{7}$$

In this way, we can write the phase equation with the unperturbed intrinsic frequency ω_i

$$\frac{d\phi_i}{dt} = \omega_i + \epsilon \nabla\phi(\mathbf{X_0}) \cdot \mathbf{P}(\phi_i, \phi_j) \tag{8}$$

The term $\nabla\phi$ is called the phase sensitivity, which represent the direction in phase space that will change the phase the most given a perturbation in that direction. Let's call it $\mathbf{Z}(\phi)$.

0.3 Two weakly interacting oscillators

If we assume that the perturbations come from other oscillators with weakly coupling the change is small over one cycle and we can substitute with the average:

$$\frac{d\phi_i}{dt} = \omega_i + \frac{\epsilon}{T} \int_0^T dt \mathbf{Z}(\phi_\mathbf{i}) \cdot \mathbf{P}(\phi_i, \phi_j) = \omega_i + \Gamma(\phi_i, \phi_j), \tag{9}$$

$$\Gamma(\phi_i, \phi_j) = \frac{\epsilon}{2\pi} \int_0^{2\pi} d\theta \mathbf{Z}(\phi_i) \cdot \mathbf{P}(\phi_i, \phi_j) \tag{10}$$

where Γ represents the interaction as a function of the relative phases of the two oscillators. Γ turns out to be the convolution between the phase sensitivity and the perturbation. The perturbation is considered known, and the sensitivity function is to be determined as a characteristics of the oscillator.

By perturbing with a delta–function at different parts of the phase we can determine $\mathbf{Z}(\phi)$. Now, if we introduce a small deviation in phase, ψ_1 for oscillator 1 and ψ_2 for 2, so that

$\phi_1 = \omega t + \psi_1$

$\phi_2 = \omega t + \psi_2$

assuming that the oscillators are identical. The equation of motion is now

$\frac{d\psi_1}{dt} = \frac{\epsilon}{2\pi}\int_0^{2\pi} d\theta \mathbf{Z}(\theta)\cdot\mathbf{P}(\theta, \theta-(\psi_1-\psi_2)) = \Gamma(\psi_1-\psi_2)$

$\frac{d\psi_2}{dt} = \frac{\epsilon}{2\pi}\int_0^{2\pi} d\theta \mathbf{Z}(\theta)\cdot\mathbf{P}(\theta, \theta-(\psi_2-\psi_1)) = \Gamma(\psi_2-\psi_1)$

with $\theta' = \omega t$ and the substitution $\theta = \theta' + \psi_1$. In this way we can also write the original equations as

$\frac{d\phi_1}{dt} = \omega_1 + \Gamma(\phi_1 - \phi_2)$

$\frac{d\phi_2}{dt} = \omega_2 + \Gamma(\phi_2 - \phi_1)$

And generally for many coupled oscillators:

$$\frac{d\phi_i}{dt} = \omega_i + \sum_{i\neq j}\Gamma(\phi_i - \phi_j) \tag{11}$$

0.4 Phaselocking, fixpoints and stability for two–oscillators

The Γ–function is periodic and can therefore be divided into an even part and an odd part denoted Γ^+ and Γ^- respectively:

$$\Gamma(\psi) = \frac{1}{2}(\Gamma^+(\psi) + \Gamma^-(\psi)) \tag{12}$$

This is useful when looking for fixpoints in the phaselocking between oscillators. If we look at the small deviations ψ_1 and ψ_2 from previous section we are interested in the difference between the deviations of the two oscillators phases, $\psi \equiv \psi_2 - \psi_1$, and the phaselocking condition reads [2]:

$$\frac{d\psi}{dt} = \omega_2 - \omega_1 + \Gamma(\psi) - \Gamma(-\psi) = \Delta\omega + \Gamma^-(\psi) = 0 \tag{13}$$

which only has a solution if the difference in intrinsic frequencies is smaller than the coupling strength. If we assume for now that the oscillators are identical, the fixpoints are at the ψ^*:

$$\Gamma^-(\psi^*) = 0,\ \psi^* = \frac{T}{2}n,\ n = 0, 1, 2, ... \tag{14}$$

with alternating stable and unstable fixpoints. The stability at the points are analyzed by Taylor expansions. The linear stability criterion reads:

$$\frac{\partial\Gamma^-(\psi^*)}{\partial\psi} < 0 \tag{15}$$

In this way, the phase dynamics and locking in or out of phase depends entirely on the shape of the coupling function. The phase sensitivity function (and therefore the coupling function) can be measured experimentally, as described earlier, or calculated from the original set of equations.

0.5 Two integrate–and–fire oscillators

The simplest simplest integrate–and–fire model is the Lapicque model [2] [4]

$$\frac{dV}{dt} = -\frac{V}{\tau} + I_{ext} + I_{syn}(t),\ 0 < V < \theta \tag{16}$$

where the $V(t)$ is supposed to model the membrane potential difference of a neuron and V will get reset to zero if it reaches θ. τ is the time constant of the rise, I_{syn} is the *synaptic input* from other neurons, for example. This term plays the role as the pertubation (when multiplied by the driving force $[V(\phi) - V_{syn}]$) and is commonly modelled as an α–function:

$$\alpha(t) = \frac{t}{\tau_0} e^{-\frac{t}{\tau_0}} \tag{17}$$

so that the synaptic input is the sum over all incomming spikes: $I_{syn} = \sum_{spikes} g\alpha(t - t_{spike})$, where the spikes are the resetting procedure and g is the synaptic conductance. Since the α–function is positive at any point we can make a statement about the stability of in–phase locking.

$$\Gamma(\psi_i - \psi_j) = \frac{\epsilon}{2\pi} \int_0^{2\pi} d\theta Z(\theta + \psi_i) I_{syn}(\theta + \psi_i, \theta + \psi_j) \tag{18}$$

where the Z and P in this case are scalar functions and $I_{syn}(\phi_1, \phi_2) = -g\alpha(\phi_2)[V(\phi_1) - V_{syn}]$. The driving force doesn't change considerably in the non–spiking period so we can consider that constant. Thus, the stability condition in phase ($\psi^* = 0$) boils down to

$$\frac{\partial \Gamma^-(\psi^*)}{\partial \psi} = -\frac{\epsilon g c}{2\pi} \int_0^{2\pi} d\theta Z(\theta) \alpha'(\theta) \tag{19}$$

c and g are positive constants. Now, the we can calculate the derivative of the α–function and we know that the sensitivity function grows monotonically untill it resets. If the oscillators are identical it will only reset once per cycle. In this way, we can split up the integral in the first part where the slope of the α–function is positive and the second part where it's negative.

$$\Gamma'(\psi^* = 0) = -\frac{\epsilon g c}{2\pi} \int_0^{\phi_{max}} d\theta Z(\theta) \alpha'(\theta) - \frac{\epsilon g c}{2\pi} \int_{\phi_{max}}^{2\pi} d\theta Z(\theta) \alpha'(\theta) \tag{20}$$

where the first term is negative and the second term is positive, the two competing to make the point stable or unstable. The phase ϕ_{max} is the peak of the α–function. Since the α–function is periodic we can write

$\int_0^{\phi_{max}} d\theta \alpha'(\theta) = -\int_{\phi_{max}}^{2\pi} d\theta \alpha'(\theta)$

The mean value theorem ensures that there is some phase in the first interval, ϕ' so

$$\frac{\int_0^{\phi_{max}} d\theta \alpha'(\theta) Z(\theta)}{\int_0^{\phi_{max}} d\theta \alpha'(\theta)} = Z(\phi') \tag{21}$$

and ϕ''similarly for the second interval

$$\frac{\int_{\phi_{max}}^{2\pi} d\theta \alpha'(\theta) Z(\theta)}{\int_{\phi_{max}}^{2\pi} d\theta \alpha'(\theta)} = Z(\phi'') \tag{22}$$

Since Z is a monotonically increasing function, $Z(\phi') < Z(\phi'')$ is always true. In this way

$$\frac{\epsilon g c}{2\pi} \int_0^{\phi_{max}} d\theta Z(\theta) \alpha'(\theta) < -\frac{\epsilon g c}{2\pi} \int_{\phi_{max}}^{2\pi} d\theta Z(\theta) \alpha'(\theta) \tag{23}$$

and the in–phase locking is an unstable fixpoint.

This is an interesting result that also applies to other neuronal models. This says that excitatory coupling will tend to desynchronize the activity and inhibitory will tend to synchronize, at least in the two–neuron case. Experimentally, it is seen that whole networks of all inhibitory coupling will tend to synchronize [6]. The strong thalamo–cortical oscillation during sleep is generated in reticular nucleus around thalamus, which only has inhibitory connections [5]. This oscillation is so strong because a large number of neurons fire in synchrony.

The picture is similar when we look at *type 1* neurons i.e. neurons that have only positive sensitivity function, such as motoneurons for example. Type 2 neurons have a slightly negative Z in the first part of the phase, correspnding to the delayed rectifier K^+ current, which will slow down the next spike. This is the case of the Hodgekin–huxley neuron and many other neurons in your brain.

Another interesting result is the work of Hansel and collegues [3] that study the excitatory coupling and phase locking of two Hudgekin–Huxley neurons, and the average frequency is actually lower than if they are not connected. This is counter–intuitive, since excitation should speed up the frequency, right? The result comes from the even part of the coupling function, which is the slope in a plot of frequency versus coupling strength in the steady state case. The general argument goes that when the two neurons lock the frequencies of the two are equal $f = f_1 = f_2 = \frac{d\phi}{dt}$. The odd part of the coupling function is zero, $\Gamma^-(\phi^*) = 0$:

$f(g) = f(0)(1 + g\Gamma^+(\psi^*))$

where the Γ^+–function is determined to be negative, in that study. However, this is a property of the type 2 neuron.

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