Feature Binding as Neuron Synchronization: Quantum Aspects

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Feature binding denotes how a large collection of coupled neurons combines external signals with internal memories into new coherent patterns of meaning. An external stimulus spreads over an assembly of coupled neurons, building up a corresponding collective state. Thus, the *synchronization* of spike trains of many individual neurons is the basis of a *coherent perception*.

Homoclinic chaos has been proposed as the most suitable way to code information in time by trains of equal spikes occurring at apparently erratic times; a new quantitative indicator, called *propensity*, is introduced to select the most appropriate neuron model.

In order to classify the set of different perceptions, the percept space is given a metric structure. The distance in percept space is conjugate to the duration of the perception in the sense that an uncertainty relation in percept space is associated with time limited perceptions. Thus coding of different percepts by synchronized spike trains entails fundamental *quantum* features with a quantum constant related to the details of the perceptual chain and very different from Planck's action.

I. FEATURE BINDING

A. Neuron synchronization

It is by now established that a holistic perception emerges, out of separate stimuli entering different receptive fields, by synchronizing the corresponding spike trains of neural action potentials [1, 2].

Action potentials play a crucial role for communication between neurons. They are steep variations in the electric potential across a cell's membrane, and they propagate in essentially constant shape from the soma (neuron's body) along axons toward synaptic connections with other neurons. At the synapses they release an amount of neurotransmitter molecules depending upon the temporal sequences of spikes, thus transforming the electrical into a chemical carrier.

As a fact, neural communication is based on a temporal code whereby different cortical areas which have to contribute to the same percept *P* synchronize their spikes. Limiting for convenience the discussion to the visual system, spike emission in a single neuron of the higher cortical regions results as a trade-off between bottom-up stimuli arriving through the LGN (lateral geniculate nucleus) from the retinal detectors and threshold modulation due to top-down signals sent as conjectures by the semantic memory. This is the core of ART (adaptive resonance theory [3]) or other computational models of perception [4] which assume that a stable cortical pattern is the result of a Darwinian competition among different percepts with different strength. The winning pattern must be confirmed by some matching procedure between bottom-up and top-down signals.

B. Perceptions, feature binding and Qualia

The role of elementary feature detectors has been extensively studied in the past decades [5]. By now we know that some neurons are specialized in detecting exclusively vertical or horizontal bars, or a specific luminance contrast, etc. However the problem arises: how elementary detectors con-tribute to a holistic (Gestalt) perception? A hint is provided by [2]. Suppose we are exposed to a visual field containing two separate objects. Both objects are made of the same visual elements, horizontal and vertical contour bars, different degrees of luminance, etc. What are then the neural correlates of the identification of the two objects? We have one million fibers connecting the retina to the visual cortex, through the LGN. Each fiber results from the merging of approximately 100 retinal detectors (rods and cones) and as a result it has its own receptive field. Each receptive field isolates a specific detail of an object (e.g. a vertical bar). We thus split an image into a mosaic of adjacent receptive fields.

Now the "feature binding" hypothesis consists of assuming that all the cortical neurons whose receptive fields are pointing to a specific object synchronize the corresponding spikes, and as a consequence the visual cortex organizes into separate neuron groups oscillating on two distinct spike trains for the two objects (Fig.1).

Direct experimental evidence of this synchronization is obtained by insertion of microelectrodes in the cortical tissue of animals just sensing the single neuron [2]. Indirect evidence of synchronization has been reached for human beings as well, by processing the EEG (electro-encephalo-gram) data [6].

Based on the neurodynamical facts reported above, we can understand how this occurs [3]. The higher cortical stages where synchronization takes place have two inputs. One (bottom-up) comes from the sensory detectors via the early stages which classify elementary features. This single input is insufficient, because it would provide the same signal for e.g. horizontal bars belonging indifferently to either one of the two objects. However, as we said already, each neuron is a nonlinear system passing close to a saddle point, and the application of a suitable perturbation can stretch or shrink the interval of time spent around the saddle, and thus lengthen or shorten the interspike interval. The perturbation consists of top-down signals corresponding to conjectures made by the semantic memory (Fig.2).

In other words, the perception process is not like the passive imprinting of a camera film, but it is an active process whereby the external stimuli are interpreted in terms of past
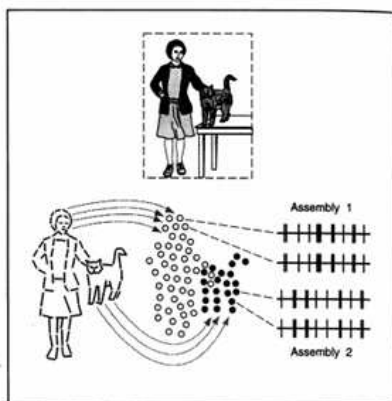
FIG. 1: *Feature binding*: the lady and the cat are respectively represented by the mosaic of empty and filled circles, each one representing the receptive field of a neuron group in the visual cortex. Within each circle the processing refers to a specific detail (e.g. contour orientation). The relations between details are coded by the temporal correlation among neurons, as shown by the same sequences of electrical pulses for two filled circles or two empty circles. Neurons referring to the same individual (e.g. the cat) have synchronous discharges, whereas their spikes are uncorrelated with those referring to another individual (the lady) (from [2]).
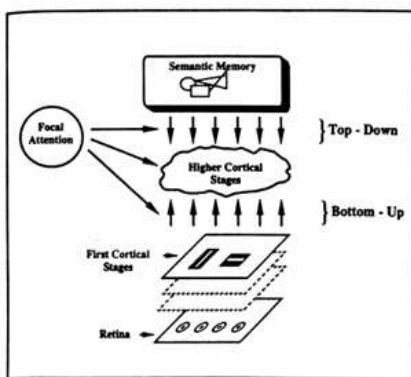

FIG. 2: *ART = Adaptive Resonance Theory*. Role of bottom-up stimuli from the early visual stages an top-down signals due to expectations formulated by the semantic memory. The focal attention assures the matching (resonance) between the two streams (after Julesz).

memories. A focal attention mechanism assures that a matching is eventually reached. This matching consists of resonant or coherent behavior between bottom-up and top-down signals. If matching does not occur, different memories are tried, until the matching is realized. In presence of a fully new image without memorized correlates, then the brain has to accept the fact that it is exposed to a new experience.

Notice the advantage of this time dependent use of neurons, which become available to be active in different perceptions at different times, as compared to the computer paradigm of fixed memory elements which store a specific object and are

not available for others (the so called "grandmother neuron" hypothesis).

II. HOMOCLINIC CHAOS, SYNCHRONIZATION AND PROPENSITY

Let us model the neurodynamics of spike formation As for the dynamics of the single neuron, a saddle point instability separates in parameter space an excitable region, where axons are silent, from a periodic region, where the spike train is periodic (equal interspike intervals). If a control parameter is tuned at the saddle point, the corresponding dynamical behavior (homoclinic chaos) consists of a frequent return to the instability [7]. This manifests as a train of geometrically identical spikes, which however occur at erratic times (chaotic interspike intervals). Around the saddle point the system displays a large susceptibility to an external stimulus, hence it is easily adjustable and prone to respond to an input, provided this is at sufficiently low frequencies; this means that such a system is robust against high frequency noise as discussed later.

Such a type of dynamics has been recently dealt with in a series of reports that here I recapitulate as the following chain of linked facts.

1. A single spike in a 3D dynamics corresponds to a quasi-homoclinic trajectory around a saddle focus SF (fixed point with 1 (2) stable direction and 2 (1) unstable ones); the trajectory leaves the saddle and returns to it (Fig.3).We say "quasi-homoclinic" because, in order to stabilize the trajectory away from SF, a second fixed point, namely a saddle node SN, is necessary to assure a *heteroclinic* connection. The experiment on a CO_2 laser confirms this behavior(Fig.4)

2. A train of spikes corresponds to the sequential return to, and escape from, the SF. A control parameter can be set at a value BC for which this return is erratic (chaotic interspike interval). As the control parameter is set above or below B_C, the system moves from *excitable* (single spike triggered by an input signal) to *periodic* (yielding a regular sequence of spikes without need for an input), with a frequency monotonically increasing with the separation ΔB from B_C [8].

3. Around SF, any tiny disturbance provides a large response. Thus the homoclinic spike trains can be synchronized by a periodic sequence of small disturbances (Fig. 5). However each disturbance has to be applied for a minimal time, below which it is no longer effective; this means that the system is insensitive to broadband noise, which is a random collection of fast positive and negative signals [9].

4. The above considerations lay the floor for the use of mutual synchronization as the most convenient way to let different neurons respond coherently to the same stimulus, organizing as a space pattern. In the case of a
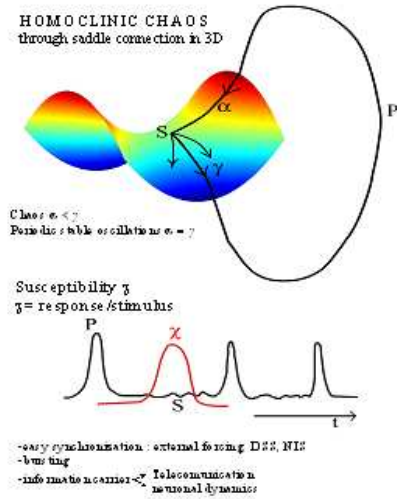
FIG. 3: Schematic view of the phase space trajectory approaching the saddle S and escaping from it. Chaos is due to the shorter or longer permanence around S; from a geometrical point of view most of the orbit P provides a regular spike.
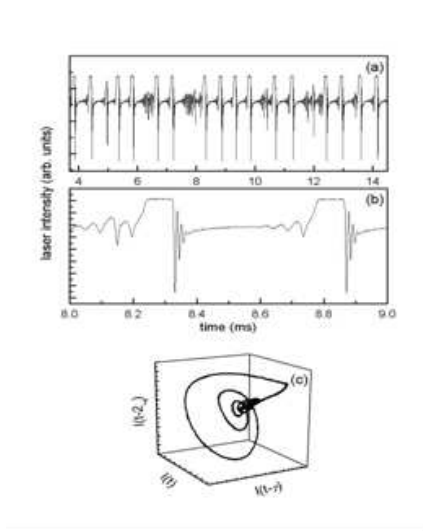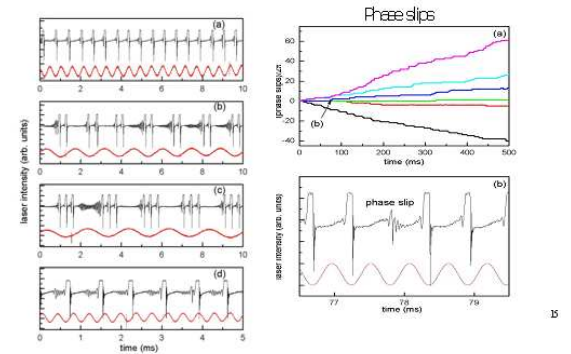

FIG. 5: Left: experimental time series for different synchronization ratios induced by periodic changes of the control parameter. (a) 1:1 locking, (b) 1:2, (c) 1:3, (d) 2:1 . Right: when the system is not able to spike for each period of the driver, a phase slip (one spike less or more) occurs, it is a jump of +/- 2, if the interspike interval is normalized to 2. The rate of +/- phase slips increases with the offset of the driving frequency from the natural frequency (associated with the average interspike interval of the free system).

FIG. 4: Experimental time series of the laser intensity for a CO_2 laser with feedback in the regime of homoclinic chaos. (b) Time expansion of a single orbit. (c) Phase space trajectory built by an embedding technique with appropriate delays (from [7]).
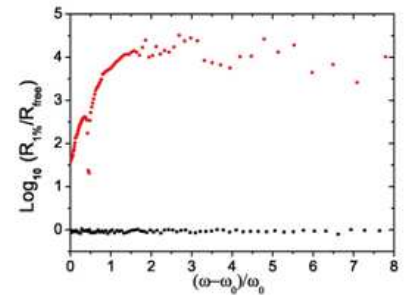

FIG. 6: The coherence parameter R is defined as the ratio between the average ISI (interspike interval) and its r.m.s. fluctuation. R is unity for a fully chaotic system and tends to infinity for a periodic system. Here we plot in log scale the ratio between R for a driving periodic disturbance of 1% to a control parameter and Rfree for the free system, at different frequencies ω away from the natural one ω_0 (average of the chaotic spiking in the unperturbed system); for HC (circles) the ratio is 30 at ω_0 and it goes up to 104 for higher frequencies; for the Lorenz system(squares) the ratio stays flat to 1. We thus take this ratio as a quantitative indicator of the *propensity to synchronization*

single dynamical system, it can be fed back by its own delayed signal. As the delay is long enough the system is decorrelated with itself and this is equivalent to feeding an independent system. This process allows to store meaningful sequences of spikes as necessary for a short term memory [10].

5. Several neuron models (integrate-and-fire, Hodgkin-Huxley, FitzHugh-Nagumo, Hindmarsh-Rose) have

been used by different investigators. We have introduced the *propensity to synchronization* as a quantitative indicator of how easy is for a chaotic system to recognize an external input (Fig. 6) [11].

6. In presence of localized stimuli over a few neurons, the corresponding disturbances propagate by inter-neuron coupling (either excitatory or inhibitory); a synchronized pattern is uniquely associated with each stimulus; the degree of mutual synchronization is measured by

the disappearance of phase slips, or defects in a space-time fabric [12].

These facts have been established experimentally and confirmed by a convenient model in the case of a class B laser with a feedback loop which readjusts the amount of losses depending on the value of the light intensity output [13].

I here recall the classification widely accepted in laser physics. Class A lasers are ruled by a single order parameter, the amplitude of the laser field, which obeys a closed dynamical equation; all the other variables having much faster decay rate, thus adjusting almost instantly to the local field value. Class B lasers are ruled by two order parameters, the laser field and the material energy storage providing gain; the two degrees of freedom having comparable characteristic times and behaving as activator and inhibitor in chemical dynamics [14].

The above listed facts hold in general for any dynamical system which has a 3-dimensional sub-manifold separating a region of excitability from a region of periodic oscillations: indeed, this separatrix has to be a saddle focus.

III. TIME CODE IN NEURAL INFORMATION EXCHANGE

How does a synchronized pattern of neuronal action potentials become a relevant perception? Not only the different receptive fields of the visual system, but also other sensory channels as auditory, olfactory, etc. integrate via feature binding into a holistic perception. Its meaning is "decided" in the PFC (pre-frontal cortex) which is a kind of arrival station from the sensory areas and departure for signals going to the motor areas. On the basis of the perceived information, motor actions are started, including linguistic utterances [6].

Sticking to the neurodynamical level, and leaving to psychophysics the investigation of what goes on at higher levels of organization, we stress here a fundamental temporal limitation.

Taking into account that each spike lasts about 1 msec, that the minimal interspike separation is 3 msec, and that the decision time at the PCF level is estimated to be $\overline{T} \approx 200$ ms, we can split $\overline{T}$ into $200/3 \sim 66$ bins of 3 msec duration, which are designated by 1 or 0 depending on whether they have a spike or not. Thus a priori total number of different messages which can be transmitted is:

$$2^{66} \approx 6 \cdot 10^{19} \ .$$

However we must account also for the average rate at which spikes proceed in our brain, which is $r = 40$ Hz (so called γ band, $< ISI >$ (average ISI) = 25 ms). When we account for this rate we can evaluate a reduction factor $\alpha = \frac{S}{T} = 0.54$ where S is an entropy [15], thus there are roughly $2^S \approx 10^{11}$ words with significant probability. Even though this number is large, we are still within a finitistic realm. Provided we have time enough to ascertain which one of the different messages we are dealing with, we can classify it with the accuracy of a digital processor, without residual error.

But suppose we expose the cognitive agent to fast changing scenes, for instance by presenting in sequence unrelated video frames with a time separation less than 200 msec. While small gradual changes induce the sense of motion as in movies, big differences imply completely different subsequent spike trains. Here any spike train gets *interrupted* after a duration ΔT less than the canonical $\overline{T}$. This means that the brain cannot decide among *all* coded perceptions having the same structure up to ΔT, but different afterwards.

Whenever we stop the perceptual task at ΔT shorter than the total time, then the bin stretch $T - \Delta T$ (we measure the times in bin units) is not explored. This means that all stimuli which provide equal spike sequences up to ΔT, and differ afterwards by *at least one* spike will cover an uncertainty region ΔP whose size is given by:

$$\Delta P = 2^{\alpha T} 2^{-\alpha \Delta T} = P_M e^{-\alpha \Delta T \ln 2} \ , \tag{1}$$

where $P_M \approx 10^{11}$ is the maximum perceptual size available with the chosen $T \sim 66.6$ bins per perceptual session and rate $r = 40$ Hz. Relation (1) is very different from the standard uncertainty relation,

$$\Delta P \cdot \Delta T = C \ , \tag{2}$$

that we would expect in a word-bin space ruled by Fourier transform relations.

Indeed, the trascendental equation (1) is more rapidly converging at short and long ΔT than the hyperbola (2). We fit (1) by (2) in the neighborhood of a small uncertainty $\Delta P = 10 \, words$, which corresponds to $\Delta T = 62 \, bins$. Around $\Delta T = 62 \, bins$ the local uncertainty (2) yields a quantum constant:

$$C = 10 \cdot 62 = 620 \, words \cdot bins \ . \tag{3}$$

To convert C into Js as Planck's $\hbar$, consider that:

1. $1 \, bin = 3 \, ms$

2. in order to jump from an attractor corresponding to one perception to a nearby one, a *minimal amount* of energy is needed, corresponding to one spike; but one spike requires the energy corresponding to about 10^7 transitions ATP $\rightarrow$ ADP + P [16] each one taking 0.3 eV; thus the total energy quantum is about 10^{-14} joules. The conversion factor is then:

$$C \approx 10^{-14} Js \approx 10^{20} \hbar \ .$$

Quantum limitations were also put forward by Penrose [17] but on a completely different basis. In his proposal, the quantum character was attributed to the physical behavior of the "microtubules" which are microscopic components of the neurons playing a central role in the synaptic activity. However, speaking of quantum coherence at the $\hbar$ level in biological processes is not plausible, if one accounts for the extreme vulnerability of any quantum system to *decoherence* processes, which make quantum superposition effects observable only in extremely controlled laboratory situations, and at

sub-picosecond time ranges ,not relevant for synchronization purposes in the $10-100$ msec range.

Our tenet is that the quantum C-level in a living being emerges from the limited time available in order to take vital decisions ;it is logically based on a non-commutative set of relevant variables and hence it requires the logical machinery built for the $\hbar$ quantum description of the microscopic world where non-commutativity emerges from use of variables coming from macroscopic experience ,as coordinate and momenta, to account for new facts.

A more precise consideration consists in classifying the spike trains. Precisely, if we have a sequence of identical spikes of unit area localized at erratic time positions τ_l then the whole sequence is represented by:

$$f(t) = \sum_l \delta(t - \tau_l) \ , \qquad (4)$$

where τ_l is the set of position of the spikes. A temporal code, based on the mutual position of successive spikes, depends on the moments of the interspike interval distributions:

$$(ISI)_l = (\tau_l - \tau_{l-1}) \ . \qquad (5)$$

Different *ISIs* encode different sensory information.

A time ordering within the sequence (3) is established by comparing the overlap of two signals as (3) mutually shifted in time. Weighting all shifts with a phase factor and summing up, this amounts to constructing a Wigner function [18]:

$$W(t, \omega) = \int_{-\infty}^{+\infty} f(t + \frac{\tau}{2}) \cdot f(t - \frac{\tau}{2}) \exp(i\omega\tau) d\tau \ . \qquad (6)$$

If now f is the sum of two packets $f = f_1 + f_2$ as in Fig. 8, the frequency-time plot displays an intermediate interference. Eq. (5) would provide interference whatever is the time separation between f_1 and f_2. In fact, we know that the decision time $\overline{T}$ truncates a perceptual task, thus we must introduce a cutoff function $g(\tau) = \exp(\frac{-\tau^2}{-T^2})$ which transforms the Wigner function as:

$$W(t, \omega) = \int_{-\infty}^{+\infty} f(t + \frac{\tau}{2}) \cdot f(t - \frac{\tau}{2}) \exp(i\omega\tau) g(\tau) d\tau \ . \qquad (7)$$

In the quantum jargon, the brain physiology breaks the quantum interference for $t > \overline{T}$ (*decoherence* phenomenon).

IV. THE ROLE OF THE WIGNER FUNCTION IN BRAIN OPERATIONS.

We have seen that feature binding in perceptual tasks implies the mutual synchronization of axonal spike trains in neurons which can be even far away and yet contribute to a well defined perception by sharing the same pattern of spike sequence. The synchronization conjecture was given experimental evidence by inserting several micro-electrodes probing each one single neuron in the cortex of cats and then studying the temporal correlation in response to specific visual inputs

[2]. In the human case, indirect evidence is acquired by exposing a subject to transient patterns and reporting the time-frequency plots of the EEG signals [6]. Even though the space resolution is poor, phase relations among EEG signals coming from different cerebral areas at different times provide an indirect evidence of the synchronization mechanism.

The dynamics of homoclinic chaos (HC) was motivated by phenomena observed in lasers and then explored in its mathematical aspects, which display strong analogies with the dynamics of many biological clocks ,in particular that of a model . HC provides almost equal spikes occurring at variable time positions and presents a region of high sensitivity to external stimuli; perturbations arriving within the sensitivity window induce easily a synchronization, either to an external stimulus or to each other (mutual synchronization) in the case of an array of coupled HC individuals (from now on called neurons).

But who reads temporal information contained across synchronized and oscillatory spike trains? [19].

In view of the above facts, we can model the encoding of external information on a sensory cortical area (e.g. V1 in the visual case) as a particular spike train assumed by an input neuron directly exposed to the arriving signal and then propagated by coupling through the array. As shown by the experiments on feature binding [2], we must transform the local time information provided by Eqs.(3) and (4) into a spatial information which tells the amount of a cortical area which is synchronized. If many sites have synchronized by mutual coupling, then the read-out problem consists in tracking the pattern of values (3), one for each site. Let us take for simplicity a continuous site coordinate r. In case of two or more different signals applied at different sites a competition starts and we conjecture that the winning information (that is, the one channeled to a decision center) corresponds to a *"majority rule"*. Precisely, if the encoding layer is a 1-dimensional chain of N coupled sites activated by external stimuli at the two ends ($i = 1$ and $i = N$),the majority rule says that the prevailing signal is that which has synchronized more sites.

The crucial question is then :who reads that information in order to decide upon? We can not recur to some *homunculus* who reads the synchronization state. Indeed, in order to be made of physical components, the homunculus itself should have some interpreter which would be a new homunculus, and so on with a *"regressio ad infinitum"*.

On the other hand, it is well known that ,as we map the interconnections in the vision system,$V1$ exits through the Vertical stream and the Dorsal stream toward the *Inferotemporal Cortex* and *Parietal Cortex* respectively. The two streams contain a series of intermediate layers characterized by increasing receptive fields ; hence they are cascades of layers where each one receives converging signals from two or more neurons of the previous layer. Let us take for the time being this feed-forward architecture as a network enabled to extract relevant information upon which to drive consequent actions. We show how this cascade of layers can localize the interface between two domains corresponding to different synchronization. It is well known that ON/OFF cells with a center-surround configuration perform a *first and second space derivative* [5]. Suppose this operation was done at certain layer . At the suc-

cessive one, as the converging process goes on, two signals will converge on a simple cells which then performs a *higher order derivative*, and so on. This way ,we build a power series of space derivatives. A translated function as $f(r+\xi)$ is then reconstructed by adding up many layers, as can be checked by a Taylor expansion. Notice that the alternative of exploring different neighborhoods ξ of r by varying ξ would imply a *moving pointer* to be set sequentially at different positions, and there is nothing like that in our physiology.

The next step consists in comparing the function $f(r+\xi)$ with a suitable standard, to decide upon its value. Since there are no metrological standards embedded in a living brain, such a comparison must be done by comparing f with a shifted version of itself, something like the product:

$$f(r+\xi) \cdot f(r-\xi) \ .$$

Such a product can be naturally be performed by converging the two signals $f(r \pm \xi)$ onto the same neuron, exploiting the nonlinear (*Hebbian*) response characteristic limited to the lowest quadratic nonlinearity, thus taking the square of the sum of the two converging inputs and isolating the double product. This operation is completed by summing up the different contributions corresponding to different, with a kernel which keeps track of the scanning over different ξ, keeping information on different domain sizes. If this kernel were just a constant, then we would retrieve a trivial average which cancels the ξ information.

Without loosing in generality, we adopt a Fourier kernel $\exp(ik\xi)$ and hence have built the quantity:

$$W(r,k) = \int_{-\infty}^{+\infty} f(r-\frac{\xi}{2}) \cdot f(r+\frac{\xi}{2})\exp(ik\xi)d\xi \ . \quad (8)$$

It contains information on both the space position r around which we are exploring the local behavior, as well as the frequency k which is associated with the space resolution. As well known, it contains the most complete information compatible with the Fourier uncertainty:

$$\Delta x \Delta k \approx 1 \ . \quad (9)$$

Notice that building a joint information on locality (x) and resolution (k) by physical measuring operations implies such an intrinsic limitation.

In summary, it appears that the Wigner function is the best read-out of a synchronized layer that can be done by exploiting natural machinery, rather than recurring to a homunculus. The local value of the Wigner function represents a decision to be sent to motor areas triggering a suitable action.

In order to have a suitable description of the feature binding mechanism in terms of a Wigner function in time (at a single site) and space (over an array of sites), we need an evolution equation. But which are the physical objects whose evolution describes the phenomenology? I conjecture that the transition from decoupled to fully synchronized neurons is controlled by the dynamics of defects (see Fig. 7). A preliminary treatment of defects as harmonic oscillators is summarized in Fig. 9. The total Hamiltonian then rules the evolution equation for
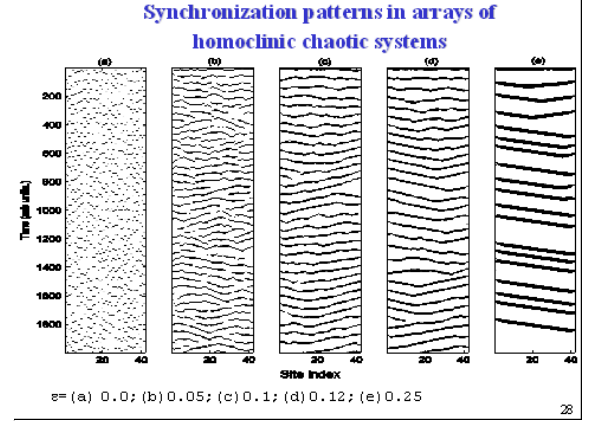

FIG. 7: Space-time representation of spike positions for a linear array of 40 neurons and for different amounts of nearest neighbor coupling. At $\varepsilon = 0.25$ percolation has been reached, in the sense that spikes at all sites are connected (besides a mutual time lag to the transfer operation); at $\varepsilon = 0$, no correlation at all among different sites; in between, a partial synchronization with the evidence of defects as phase slips (one spike more, or less, with respect to the neighbor site)
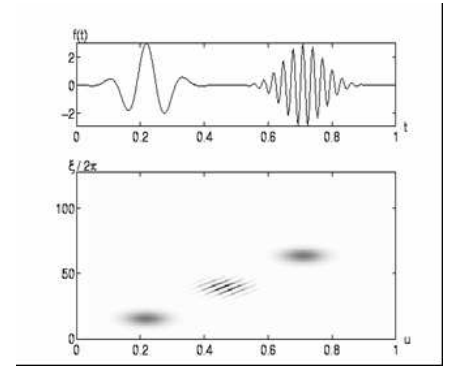

FIG. 8: Wigner distribution of two localized sinusoidal packets shown at the top. Bottom :frequency-time representation of the levels of the (real but not always positive!) Wigner function. The oscillating interference is centered at the middle time-frequency location.

the Wigner function and should provide a complete quantum description.

The oscillating interference of Fig. 8 is crucial for *quantum computation*. The reason why we don't see it in ordinary life is *"decoherence"*. Let us explain what we mean. If the system under observation is affected by the *environment*, then the two states of the superposition have a finite lifetime; however, even worse, the interference decays on a much shorter time (the smaller, the bigger is the separation between the two states): so while the separation is still manageable for the two polarization states of a photon, it becomes too big for the two states of a macroscopic quantum system. Precisely if we call τ_i the intrinsic decay of each one of the two states of the superposition, then the mutual interference in the Wigner function
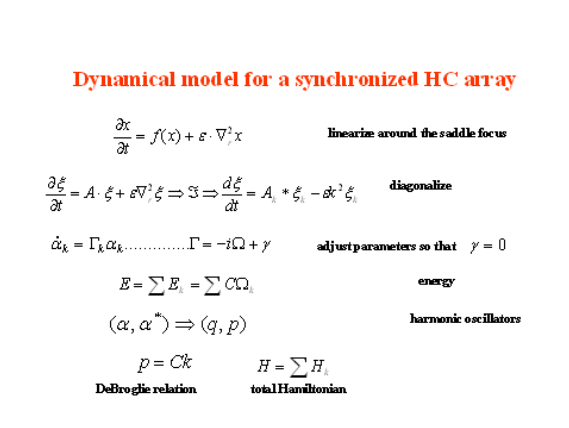
FIG. 9: A summary of the strategy to characterize the quantum aspects of the time code. Here x is an N-dimensional (N=6 for HC) dynamical field depending on position r. Its local dynamics is given by the equation $f(x)$. Furthermore, the space coupling of strength ε is given by the Laplacian. Linearizing the equation at parameter values below the percolation threshold, Fourier transforming, and diagonalizing the linearized equation, we have harmonic oscillators. Their coordinate and momenta do not commute, because of the fundamental energy-time uncertainty with the quantum constant C. From the $k-$dependence of the oscillator amplitudes one reconstructs the space features of the defects.

decays with the so-called decoherence time:

$$\tau_{dec} = \frac{\tau_i}{D^2} \; , \tag{10}$$

where D^2 is the square of the separation D in phase space between the centers of the Wigner functions of the two separate states. Notice that, in microscopic physics, D^2 *is measured in units of* $\hbar$. Usually $\frac{D^2}{\hbar}$ is much bigger than unity for a macroscopic system and hence τ_{dec} is so short that any reasonable observation time is too long to detect a coherent superposition. Instead, in neurodynamics, we perform measurement operations which are limited by the intrinsic sizes of space and time resolutions peculiar of brain processes. The associated uncertainty constant C is such that it is very easy to have a relation as (9) with D^2/C comparable to unity, and hence superposition lifetimes comparable to times of standard neural processes. This implies the conjecture that for short times or close cortical domains a massive parallelism typical of quantum computation should be possible. The threshold readjustment due to expectations arising from past memory acts as an environmental disturbance, which orthogonalizes different neural states, destroying parallelism.

[1] C. Von der Malsburg, *"The correlation theory of brain function"*, reprinted in: E. Domani, J.L. Van Hemmen and K. Schulten (Eds.), *"Models of neural networks II"* (Springer, Berlin, 1981).

[2] W. Singer and C.M. Gray, *"Visual feature integration and the temporal correlation hypothesis"*, Annu. Rev. Neurosci. **18**, 555 (1995).

[3] S. Grossberg, *"The attentive brain"*, The American Scientist, **83**, 439 (1995).

[4] G.M. Edelman and G. Tononi, *"Neural Darwinism: The brain as a selectional system"*, in: "Nature's Imagination: The frontiers of scientific vision", J. Cornwell (ed.) (Oxford University Press, New York, 1995), pp.78-100.

[5] D.H. Hubel, *"Eye, brain and vision"*, Scientific American Library, n. 22 (W.H. Freeman, New York, 1995).

[6] E. Rodriguez, N. George, J.P. Lachaux, J. Martinerie, B. Renault, and F. Varela, *"Perception's shadow:Long-distance synchronization in the human brain"*, Nature **397**, 340 (1999).

[7] E. Allaria, F.T. Arecchi, A. Di Garbo, and R. Meucci, *"Synchronization of homoclinic chaos"*, Phys. Rev. Lett. **86**, 791 (2001).

[8] R. Meucci, A. Di Garbo, E. Allaria, and F.T. Arecchi, *"Autonomous Bursting in a Homoclinic System"*, Phys. Rev. Lett. **88**, 144101 (2002).

[9] C.S. Zhou, E. Allaria, S. Boccaletti, R. Meucci, J. Kurths, and F.T. Arecchi, *"Noise induced synchronization and coherence resonance of homoclinic chaos"*, Phys. Rev. E **67**, 015205 (2003).

[10] F.T. Arecchi, R. Meucci, E. Allaria, A. Di Garbo, and L.S.

Tsimring, *"Delayed self-synchronization in homoclinic chaos"*, Phys. Rev. E **65**, 046237 (2002).

[11] F.T. Arecchi, E. Allaria, and I. Leyva, *"A propensity criterion for networking in an array of coupled chaotic systems"*, Phys. Rev. Lett. **91**, 234101 (2003).

[12] I. Leyva, E. Allaria, S. Boccaletti, and F. T. Arecchi, Phys. Rev. E **68**, 066209 (2003).

[13] F.T. Arecchi, R. Meucci, and W. Gadomski, *"Laser dynamics with competing instabilities"*, Phys. Rev. Lett. **58**, 2205 (1987).

[14] F.T. Arecchi, *"Instabilities and chaos in single mode homogeneous line lasers"*, in: Instabilities and chaos in quantum optics, F.T. Arecchi and R.G. Harrison (eds.), Springer Series Synergetics, Vol. 34 (1987), pp. 9-48.

[15] F. Rieke, D. Warland, R. de Ruyter van Steveninck and W. Bialek, *"Spikes: Exploring the neural code"* (MIT Press, Cambridge, Mass., 1996).

[16] S.B. Laughlin, R. de Ruyter van Steveninck and J. Anderson, *"The metabolic cost of neural information"*, Nature Neuroscience **1**, 36 (1998).

[17] R. Penrose, *"Shadows of the Mind"* (Oxford University Press, New York, 1994).

[18] E. Wigner, *"On the Quantum Correction For Thermodynamic Equilibrium"* Phys. Rev. **70**, 749 (1932).

[19] K. MacLeod, A. Backer and G. Laurent, *"Who reads temporal information contained across synchronized and oscillatory spike trains?"*, Nature **395**, 693 (1998).