Generalized memory associativity in a network model for the neuroses

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We review concepts introduced in earlier work, where a neural network mechanism describes some mental processes in neurotic pathology and psychoanalytic *working-through*, as associative memory functioning, according to the findings of Freud. We developed a complex network model, where modules corresponding to sensorial and symbolic memories interact, representing unconscious and conscious mental processes. The model illustrates Freud's idea that consciousness is related to symbolic and linguistic memory activity in the brain. We have introduced a generalization of the Boltzmann machine to model memory associativity. Model behavior is illustrated with simulations and some of its properties are analyzed with methods from statistical mechanics.
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A working hypothesis in neuroscience is that human memory is encoded in the neural net of the brain, and it has been assumed, since Freud, that part of this memory is not directly accessible through symbolic representations, but is repressed. It forms the unconscious mind, and as it cannot be expressed through language, it does so through other body response mechanisms in the form of neurotic symptoms. In the present work, we have reviewed and extended our work relating neuronal networks to memory functioning and the repressed. The model we employed to represent conscious and unconscious mental processes consists of modeling the underlying brain mechanisms as a bimodular network. In this description, the links between neurons pertaining to different modules are weaker than those belonging to the same module. Each minimum energy state of the global neural network is associated with a single memory trace, where only the part of memory which can trigger access to symbolic processing brain areas is directly accessible and consists of the conscious region, the remaining part forming the unconscious. Stimulating the unconscious sensorial part of a memory trace, through changes in the state of one of its neurons, may bring about a change in the symbolic region of the same memory pattern. In such a case, the connections between the sensorial and symbolic parts of memory are reinforced and unconscious processes may become conscious. This mechanism, which corresponds in psychoanalysis to the working-through process, is introduced in the model as a synaptic learning procedure. Memory retrieval, on the other hand, is described through a simulated annealing process. The behavior of the network under different assumptions on the way simulated annealing is performed on the model network is the main purpose of this work. We show that traditional approaches to memory modeling, such as the Boltzmann machine, may, at least in some cases, be advantageously superseded by more recent treatments inspired by nonextensive statistical mechanics, which may describe more appropriately brain mechanisms involved in thinking.

I. INTRODUCTION

Much of our recent work[1–3] regards the search for neuronal network mechanisms, whose emergent states underlie behavioral aspects traditionally studied in areas such as psychiatry, psychoanalysis, and neuroscience. Our motivations range from understanding psychopathologies, in the hope of contributing to the comprehension of methods of treatment, to investigations of basic mechanisms for the development of artificial intelligence and consciousness.

It is one of the early findings of psychoanalytic research regarding the *transference neuroses*, that traumatic and repressed memories are knowledge which is present in the subject but is symbolically inaccessible to him, i.e., momentarily or permanently inaccessible to his conscience. It is therefore considered *unconscious* knowledge.[4,5] They arise from events which give the mind a stimulus too powerful to be dealt with in the normal way, and thus result in permanent disturbances.

Freud[6,7] observed that neurotic patients systematically repeated symptoms in the form of ideas and impulses and called this tendency a *compulsion to repeat*. It is as if the patient repeats with "*…the intention of correcting a distressing portion of the past…* ."[4] The obsessional ideas and impulses and the perception of the performance of the neurotic actions are not themselves unconscious, but their psychical predeterminants are unconscious. It is the task of analytical treatment to infer these predeterminants and bring them to

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consciousness and provide, through their interpretation, the connections into which they are inserted.

He found that the process of "bringing what is unconscious into consciousness," which is part of the analytical treatment, involves a basic procedure of "*filling up the gaps in the patient's memories, to remove his amnesias*,"[4] and furthermore, that in neurotic patients these amnesias have an important connection to the origin of the symptoms, i.e., with the compulsion to repeat. Neurotic analysands have obtained relief and cure of painful symptoms through a mechanism called *working-through*. This technique aims at developing knowledge regarding the causes of symptoms by accessing unconscious memories and understanding and changing the analysand's compulsion to repeat.[5–7] It involves mainly analyzing free associative talking, symptoms, parapraxes (slips of the tongue and pen, misreading, forgetting, etc.), dreams, and also that which is acted out in transference.

We have described in Ref. 2 a schematic functional and computational model for some concepts associated with neurotic mental processes, as described by Freud.[4–10] In doing so, we expect that further understanding regarding psychopathology and the unconscious may also enhance our comprehension of basic mechanisms that underlie consciousness. An organization derived from a neurophysiological approach was proposed by Edelman.[11]

Based on the view of the brain as a parallel and distributed processing system,[12–14] we assume that human memory is encoded in the architecture of the neural net of the brain. By this we mean that we record information by reconfiguring the topology of our neural net, i.e., the set of active neurons and synapses that interconnect them to each other, along with the intensities and durations of these connections. We will refer to this reconfiguration process as *learning*, in accordance with the terminology used in neural network modeling. Mental states are the result of the distributed neural activity in the brain,[11,12,15,16] where the emergence of a global state generates a bodily response, called an *act*.

Since there is no clear consensus on the relevance of quantum effects in the macroscopic phenomena that underlie molecular and cellular activity in the brain, we take a classical approximation of these phenomena. In particular, we disregard the possibility of the occurrence of nondeterministic events in brain activity caused by quantum effects. We thus attribute any unpredictability in mental behavior to the sensitivity of the nonlinear complex neural networks to initial states and to internal and external quantities, which cannot be determined exactly.

Finally, we assume that each brain state (global state of the neural network) represents only one mental state. This is equivalent to affirming, in linguistic terms, that each symbol is associated with only one significance (meaning), so that we have a one-to-one functional mapping. We suggest that the symbol is represented physiologically by a minimal energy state of the neural net configuration, which encodes the memorized symbolic information.

The artificial neural network we developed to illustrate the functional model for the mental processes, which we are describing, has interesting properties that can be studied within the context of complex networks.[17–21] We have used some concepts commonly used in the approach of complex networks, such as node degree distributions, clustering coefficients, and other statistical mechanical quantities and tried to relate network structure and topology to system dynamics.

We have also used methods from statistical mechanics to study the dynamics of memory access.[23] The power-law and generalized q-exponential behaviors we have found for the node degree distributions in our model are a common feature of many biological systems and indicate that they may not be well described by Boltzmann–Gibbs (BG) statistical mechanics but rather by nonextensive statistical mechanics.[20,22,23] We have therefore modeled memory by a generalization of the Boltzmann machine (BM), called generalized simulated annealing (GSA),[23] derived from the nonextensive formalism. In GSA, the probability distribution of the system's microscopic configurations is not the BG distribution, assumed in the BM, and this should affect the chain of associations of ideas which we are modeling.

In this paper, we review the model where brain mechanisms involved in neurosis are represented as a complex system, based on a neural network composed of hierarchically clustered memory modules. We then present simulation illustrations of the model and analyze them with methods from statistical mechanics. We show some macroscopic properties of the network's structure, which suggest the generalization of the memory retrieval mechanism that affects associativity. A review conducted by Taylor of recent developments in the scientific understanding of consciousness, as well as a model for attention as a basic function related to conscious activity, may be found in Refs. 24 and 25. Kinsbourne[26] discussed how Freud's attempt at proposing a neural substrate for mental processes[27] can be viewed in light of modern developments in neuroscience, such as the understanding of forebrain functioning. Aleksander and Morton reviewed their Axiomatic Consciousness theory in Ref. 28, which addresses phenomenology in neuroscience (relating symbolic representation to subjective experience), and apply it to modeling of visual phenomenology. The Global Workspace theory[29] is based on the computational science concept of global workspace, where material needed to be worked on by a number of processors is held, and gives a useful view on certain aspects of consciousness.

Another contribution for modeling neurotic phenomena with neural networks can be found in Ref. 30. In their paper, the authors developed a ten-node neural network to represent one of Freud's case studies of a patient he named Lucy R. The authors called their architecture Lucynet and each node of the network corresponds to important actors and events in Lucy R.'s saga. A learning algorithm was proposed to change connectivity in Lucynet to simulate the emergence of the trauma and the associations among the elements of the story. Our work is similar to these ideas, in assuming that the subject's history in psychopathology is stored in the connectivity structure of the neural network. In Lucynet, the associations are among the different elements of the story (e.g., children +love). We differ in proposing also a generic neural network model, based on an associative memory mechanism, where stimuli different than the repressed unconscious, although similar, recall the repressed memory to simulate the compul-

sion to repeat. Our model contemplates the type of association present in Lucynet (which we represented by long-range synapses) and also similarity associations such as kitten → cat.

In order to develop the model, we have used many concepts and methods from statistical mechanics and the recent research area of complex networks. Some examples of these works which have strongly influenced us can be found in Refs. 12, 13, 18, 19, and 31–33. A review on synchronization in complex networks can be found in Ref. 21. From the area of neurophysiology some examples can be found in Refs. 11, 15, and 34.

Our main contribution with respect to the current work regarding machine models of consciousness is to propose a neuronal associative memory mechanism that describes conscious and unconscious memory activities involved in neurosis. The unconscious compulsion to repeat is explained as an associative memory mechanism, where an input stimulus of any kind associates with a pattern in sensorial memory, which cannot activate symbolic brain processing areas. Neurotic (unconscious) acts are isolated from symbolic representation and association (similar to reflexes). With our network model, we illustrate how Freud's ideas regarding the unconscious show that symbolic processing, language, and meaning are essential for consciousness. The neuronal model we have proposed presents interesting structural properties which can be studied from the perspective of complex networks, and we also show some interesting results in this area.

Although biologically plausible, in accordance with many aspects described by psychodynamic and psychoanalytic clinical experience, and experimentally based on simulations, the model is very schematic. It presents a metaphorical view of facets of mental phenomena, for which we seek a neuronal substratum, and suggests directions of search. We do not sustain or prove that this is the actual mechanism that occurs in the human brain. Our investigations strongly indicate the importance of the connection of symbolic processing, meaning, and language for consciousness.

In Sec. II, we present the neuronal model for the conscious-unconscious processes involved in neuroses, with a description of the algorithms. Section III presents results from experiments with computer simulations of the model. In Sec. IV, we present our conclusions and perspectives for future work.

II. A MODEL FOR CONSCIOUS AND UNCONSCIOUS PROCESSES IN NEUROSIS

In this section, we review the model described in Ref. 2, where we proposed that the neuroses manifest themselves as an associative memory process. An associative memory is a mechanism where the network returns a given stored pattern when it is shown another input pattern sufficiently similar to the stored one.[12–14] The compulsion to repeat neurotic symptoms was modeled by supposing that such a symptom is acted when the subject is presented with a stimulus which resembles a repressed or traumatic (unconscious) memory trace. The stimulus causes a stabilization of the neural net onto a minimal energy state, corresponding to the memory trace that synthesizes the original repressed experience, which, in turn, generates a neurotic response (an act). In neurotic behavior, associated with a stimulus, the act is not a result of the stimulus as a new situation but a response to the original repressed memory trace.

The original repression can be accounted for by a mechanism which inhibits the formation of certain synaptic connections. The inhibition may be externally imposed, for example, by cultural stimulation or the relation with the parents, and internalized so that the subject inhibitively stimulates the regions associated with the memory traces, not allowing the establishment and enforcement of certain synaptic connections.

The symbolic, associative process involved in psychoanalytic working-through was mapped onto a process of reinforcing synapses among memory traces in the brain. These connections should involve symbolic memory, leading to at least partial transformation of repressed memory to consciousness. This is related to the importance of language in psychoanalysis and the idea that unconscious memories are those that cannot be expressed symbolically. As the analysand symbolically elaborates manifestations of unconscious material through transference in psychoanalytic sessions, he reconfigures the topology of his neural net, by creating new connections and reinforcing or inhibiting older ones, among the subnetworks that store the repressed memory traces. The network topology that results from this reconfiguration will stabilize onto new energy minima, associated with new acts.

In our model, it is clear why repetition in psychoanalysis is specially important. Neuroscience has established that memory traces are formed by repeatedly reinforcing, through stimulation, the appropriate synaptic connections. This corresponds to the learning process in a neural network model and accounts for the long durations of psychoanalytic processes. Much time will be needed to overcome resistances in order to access and interpret repressed material, and even more to repeat and reconfigure the net in a learning process.

We propose a memory organization, where neurons belong to two hierarchically structured modules corresponding to *sensorial* and *symbolic* memories (Fig. 1). Traces stored in sensorial memory represent mental images of stimuli received by sensory receptors (located in eyes, ears, skin, and other parts of the body) from the environment and the body itself, including information regarding affects and emotion. Sensorial memory represents brain structures such as the amygdala, cerebellum, reflex pathways, hippocampus, and prefrontal, limbic, and parieto-occipital-temporal cortices that synthesize visual, auditory, and somatic information. Symbolic memory stores representations of traces in sensorial memory, i.e., *symbols*, and refers to a higher level of representation. It represents structures such as areas of the medial temporal lobe, the hippocampus, Wernicke's and Broca's areas, and other areas of the frontal cortex. These latter areas are associated with language and, because of them, we can associate a description by words or maybe a painting with the visual sensation of seeing a beautiful view. Sensorial and symbolic memories interact, producing unconscious and conscious mental activity. Attentional mechanisms,[24,25]

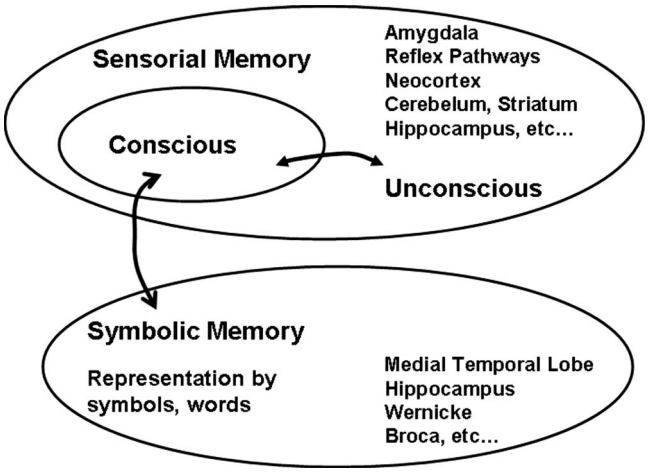

FIG. 1. Memory modules that represent storage of sensorial input and symbolic representations, and also memory traces that can or cannot become conscious.

which we did not model, select stimuli to sensorial memory and allow them to become conscious, if associated with a trace in symbolic memory.

We partially follow an idea from cognitive psychology,[15] where memory is categorized as *implicit* (or *nondeclarative*) and *explicit* (*declarative*). Implicit memory refers to information about how to perform something and is recalled unconsciously. It is rigid, tightly connected to the original stimulus conditions under which the learning occurred, and is typically involved in training reflexive motor or perceptual skills. Explicit memory refers to factual knowledge of people, places, and things and what these facts mean, and is recalled by a deliberate, conscious effort. It is highly flexible and involves the association of multiple bits of information. Kandel[15] explained that "…all explicit memories can be concisely expressed in declarative statements… ." Sensorial memory resembles implicit memory with mental images of all sensorial input, including information regarding affects and emotion (feeling sad, happy, etc…). Symbolic memory associates symbols with sensorial information.

The compulsion to repeat in neuroses[4,6] is thus explained here as a bodily response (an act) to an access to sensorial memory, which does not activate symbolic memory, as in a reflex. This accounts for the fact that neurotics say they cannot explain their neurotic acts. If the retrieval of a sensorial memory trace can activate retrieval of a pattern in symbolic memory, it can become conscious, the output is not as in reflexive behavior and there is another level of processing. We include here symbolic representations of emotions such as in "I felt nostalgic when I remembered the happiness I experienced last summer." Sensorial information which cannot associate with a symbol remains unconscious. This mechanism is similar to ideas of Edelman[11] and Changeux[34] and strongly reflects Freud's concepts of conscious and unconscious mental processes and the role of language in psychoanalysis.[4,6]

A. Algorithm neurosis

Memory functioning was initially modeled by a BM[13,14] with N nodes, which are connected symmetrically by weights

Algorithm *Boltzmann_Simulated_Annealing*

$S := S_0$;
$T := T_0$;
while $T \geq T_f$ **do**
begin
for $l := 1$ **to** $L(T)$ **do**
begin
Generate S';
if $H(S') \leq H(S)$ **then**
$S := S'$;
else
Assign S' to S with probablility $P_{BG}(S \rightarrow S')$;
end;
$T := r(T)$;
end

FIG. 2. Basic simulated annealing procedure (Ref. 14). There are variations of this basic procedure with different choices of T_0, T_f, $r(T)$, $L(T)$, and the stopping criterion. In our simulations, $r(T)=\alpha T$.

$w_{ij}=w_{ji}$. The states S_i of the units n_i take output values in $\{0,1\}$. Because of the symmetry of the connections, there is an energy functional

$$H(\{S_i\}) = -\frac{1}{2}\sum_{ij} w_{ij}S_iS_j, \tag{1}$$

which allows us to define the BG distribution function for network states

$$P_{\mathrm{BG}}(\{S_i\}) = \exp\left[-\frac{H(\{S_i\})}{T}\right] \Bigg/ \sum_{\{S_i\}} \exp\left[-\frac{H(\{S_i\})}{T}\right], \tag{2}$$

where T is the network temperature parameter. The corresponding transition probability (acceptance probability) from state $S \equiv \{S_i\}$ to S', if $H(S') \geq H(S)$, is given by

$$P_{\mathrm{BG}}(S \rightarrow S') = \exp\left[\frac{H(S) - H(S')}{T}\right]. \tag{3}$$

In the BM, pattern retrieval on the net is achieved by a standard simulated annealing process, in which the network temperature T is gradually lowered by a factor α. Figure 2 presents the basic procedure.[14] A detailed treatment of the BM may be found in Refs. 13 and 14.

In our simulations, initially, we take random connection weights w_{ij}. Links that connect neurons between sensorial and symbolic memories are weaker, representing the weaker conscious and unconscious connections in neurosis. This is done by multiplying the connections between the two subsets by a real number ζ in the interval $(0,1]$.

Once the network is initialized, we find the stored patterns by presenting many random patterns to the BM, with an annealing schedule α that allows stabilizing onto the many local minimum states of the network energy function. These

initially stored patterns, associated as they are with two weakly linked subnetworks, represent the neurotic memory states. In Ref. 1, Carvalho *et al.* proposed a neurocomputational model to describe how the original memory traces are formed in cortical maps.

In order to simulate the working-through process, one should stimulate the net by means of a change in a randomly chosen node n_i belonging to the sensorial, "unconscious" section of a neurotic memory pattern. This stimulus is then presented to the network and, if the BM retrieves a pattern with conscious configuration different from that of the neurotic pattern, we interpret this as a new conscious association, and enhance all weights from n_i to the changed nodes in the symbolic (conscious) module. The increment values are given by

$$\Delta w_{ij} = \beta S_i S_j w_{\max}, \tag{4}$$

where β is the learning parameter chosen in (0,1) and $w_{\max}$ the maximum absolute value of the synaptic strengths. We note that new knowledge is learned only when the stimulus from the analyst is not similar to the neurotic memory trace.[9]

This procedure must be repeated for various reinforcement iterations in an adaptive learning process, and also each set of reinforcement iterations is repeated for various initial annealing temperature values. The new set of synaptic weights will define a new network configuration.

B. Hierarchical clustering algorithm

In order to model the topological structure of each of the two memories, we consider the following microscopic biological mechanisms, which inspired the model we have proposed in Ref. 35. Brain cells in many animals have a structure called *on-center/off-surround*, in which a neuron is in cooperation, through excitatory synapses, with other neurons in its immediate neighborhood, whereas it is in competition with neurons that lie outside these surroundings. Competition and cooperation are found statically hardwired and also as part of many neuronal dynamical processes, where neurons compete for certain chemicals.[15,36] In synaptogenesis, for example, substances generically called neural growth factors are released by stimulated neurons and, spreading through diffusion, reach neighboring cells, promoting synaptic growth. Cells that receive neural growth factors make synapses and live, while cells that have no contact with these substances die.[15,37] A neuron that releases neural growth factors guides the process of synaptic formation in its tridimensional neighborhood, becoming a center of synaptic convergence. When neighboring neurons release different neural growth factors in different amounts, many synaptic convergence centers are generated and a competition is established between them through the synapses of their surroundings. A signaling network is thus established to control development and plasticity of neuronal circuits. Since this competition is started and controlled by environmental stimulation, it is possible to have an idea of the way environment represents itself in the brain.

Based on these microscopic mechanisms, we developed the following *clustering algorithm* to model the self-organizing process which controls synaptic plasticity, resulting in a structured topology of each of the two memory modules.

- Step 1. Neurons are uniformly distributed in a square bidimensional sheet.
- Step 2. To avoid the unnecessary and time-consuming numerical solution of the diffusion equation of the neural growth factors for simulation of synaptic growth, we assume a Gaussian solution. Therefore, a synapse is allocated to connect a neuron n_i to a neuron n_j according to a Gaussian probability, given by

  $$P_{ij} = \exp(-(\mathbf{r_j} - \mathbf{r_i})^2/(2\sigma^2))/\sqrt{2\pi\sigma^2}, \tag{5}$$

  where $\mathbf{r_j}$ and $\mathbf{r_i}$ are the positions of n_j and n_i in the bidimensional sheet and σ is the standard deviation of the distribution and is considered here a model parameter. If a synapse is allocated to connect n_i and n_j, its strength is proportional to P_{ij}.
- Step 3. We verified in Ref. 1 that cortical maps representing different stimuli are formed such that each stimulus activates a group of neurons spatially close to each other, and that these groups are uniformly distributed along the sheet of neurons representing memory. We thus now randomly choose m neurons which will each be a center of the representation of a stimulus. The value of m should be chosen considering the storage capacity of the BM.[13]
- Step 4. For each of the m centers chosen in step 3, reinforce adjacent synapses according to the following criteria. If n_i is a center, define $\text{sum}_{n_i} = \Sigma_j |w_{ij}|$, where w_{ij} is the weight of the synapse connecting n_j to n_i. For each n_j adjacent to n_i, increase $|w_{ij}|$ by Δw_{ij}, with probability $\text{Prob}_{n_j} = |w_{ij}|/\text{sum}_{n_i}$, where $\Delta w_{ij} = \eta\, \text{Prob}_{n_j}$ and $\eta \in \Re$ is a model parameter chosen in [0,1]. After incrementing $|w_{ij}|$, decrement Δw_{ij} from the weights of all the other neighbors of n_i, according to: $\forall k \neq j, |w_{ik}| = |w_{ik}| - \Delta w_{ik}$, where $\Delta w_{ik} = (1 - |w_{ik}|/\Sigma_{k\neq j}|w_{ik}|)\Delta w_{ij}$.
- Step 5. Repeat step 4 until a clustering criterion is met.

In the above clustering algorithm, steps 2 and 3 are justified in the algorithm's description. Step 4 regulates synaptic intensities, i.e., *plasticity*, by strengthening synapses within a cluster and reducing synaptic strength between clusters (disconnects clusters). By cluster, we mean a group of neurons that are spatially close, with higher probability of being adjacent by stronger synapses. This step represents a kind of preferential attachment criterion with some conservation of energy (neurosubstances) among neurons, controlling synaptic plasticity. Neurons that have received stronger sensorial stimulation and are therefore more strongly connected will stimulate their neighborhoods and promote still stronger connections. This is in agreement with the microscopic biological mechanisms we mentioned above.

The growth of long-range synapses is energetically more costly than short-range synaptic growth, and, therefore, in the brain the former is less frequent than the latter. For allocating long-range synapses that connect clusters, we should consider the basic learning mechanism proposed by Hebb,[11,13,38] based on the fact that synaptic growth among two neurons is promoted by simultaneous stimulation of the pair.

Structural concepts such as combinatorial syntax and semantics, involving compositionality and systematicity, are important in the study of language and thought and have been studied in depth by classical cognitive theories.[39] For example, these features account for the fact that a person who understands the sentence "John loves the girl" can also understand "the girl loves John" without having an independent representation for each of the sentences that can be constructed with a language. It seems not to be clear whether a purely connectionist theory can reproduce such structure.[39] However, both classical and connectionist theorists agree that symbol structures in a cognitive model should correspond to physical structures in the brain and "the combinatorial structure of a representation should have a counterpart in structural relations among physical properties of the brain."[39] Combinatorial syntactic and semantic structures should be amenable to mapping to a neuronal network.[12,39]

It would thus be interesting if a model such as ours could be expanded to treat such issues, which we have not yet considered. One could associate clusters connected by short-range synapses with atomic mental representations and use long-range synapses to implement combinatorial structure. This suggests a possibility of constructing a mechanism, whereby the external world, culture, and language[40] would be reflected onto brain topology. Memory traces stored by configurations of states of neuronal groups which receive simultaneous stimuli should enhance synaptic growth among these groups, allowing association among traces. Since memory traces represent both sensorial information and concepts (symbolic memory), we also represent association of ideas or symbols by long-range synapses.

We have begun to study these processes and, since we are still not aware of the synaptic distributions that result in such topologies, as a first approximation, we have allocated synapses randomly among clusters. Within a cluster C, a neuron n_i is chosen to receive a connection with probability $P_i=\Sigma_j|w_{ij}|/\Sigma_{n_j\in C}\Sigma_k|w_{jk}|$. If the synapse connects clusters in different memory sheets (sensorial and symbolic memories), its randomly chosen weight is multiplied by a real number ζ in the interval (0,1], reflecting the fact that, in neurotic patterns, sensorial information is weakly accessible to consciousness, i.e., repressed.

Mechanisms of memory storage and retrieval by the BM and simulation of the working-through psychoanalytical process are then carried on as reviewed in Sec. II A and described in Ref. 2.

C. Generalized simulated annealing

In neural network modeling, temperature is inspired by the fact that real neurons fire with variable strength, and that there are delays in synapses, random fluctuations from the release of neurotransmitters, and so on. These are effects that we can loosely think of as noise,[12,13,15] and thus we may consider that temperature in BMs controls noise. In our model, temperature allows associativity among memory configurations, lowering synaptic inhibition, in an analogy with the idea that freely talking in analytic sessions and stimulation from the analyst lower resistances and allow greater associativity.

The BM differs from a gradient descent minimization scheme, in that it allows the system to change state with an increase in energy, depending on the temperature value, according to Eq. (3). The Boltzmann distribution function favors changes in states with small increases in energy, so that the machine will strongly prefer visiting state space in a nearby energy neighborhood from the starting point.

The topologies we have generated with the algorithm presented in Sec. II B are hierarchically clustered, containing synapses that connect neurons that are nearest neighbors in spatial coordinates, and also long-range synapses. Furthermore, the node degree distribution curves for these topologies (see Fig. 5) show that, asymptotically, the power-law and generalized q-exponential fits are appropriate. This is a common feature of many biological systems and indicates that they may not be well described by BG statistical mechanics.[32]

There is no theoretical indication of the exact relation between network topology and memory dynamics. There have been some indications that complex systems which present a power-law behavior, i.e., which are asymptotically scale invariant, may be better described by the nonextensive statistical mechanics formalism.[20,23,32,33,41] Since the neural systems we are studying do not have only local interactions and present the scale-free topology characteristic, we have begun to investigate memory dynamics with a generalized acceptance probability distribution function[23] for a transition from state S to S', if $H(S')\geq H(S)$, given by

$$P_{\mathrm{GSA}}(S\rightarrow S')=\frac{1}{[1+(q_A-1)(H(S')-H(S))/T]^{1/(q_A-1)}}, \qquad (6)$$

where q_A is a model parameter and other variables and parameters are the same as defined in Sec. II A.

If one substitutes Eq. (6) for Eq. (3) in the algorithm in Fig. 2, the resulting procedure is called GSA.[23] The GSA procedure presented in Ref. 23 also proposes a *visiting distribution function* for generating the possible state S', which we have not studied yet. As it is well known, in the $q_A\rightarrow 1$ limit, GSA recovers the BM.

The acceptance probability distribution given by Eq. (6) should allow more associativity among memory states, which correspond to more distant minima in the energy functional H than Eq. (3). This implies that the GSA machine will tend to make many local associations and, more often than the BM, will also make looser, more distant associations. This should correspond to a more flexible and creative memory dynamics in the brain.[42]

III. SIMULATIONS AND NETWORK BEHAVIOR

In the following illustrative simulation experiments, N is the number of neurons in the network, such that N_{sens} and N_{symb} of them belong to the sensorial and symbolic memory sets, respectively. We are interested, in this first approach, in illustrating the basic concepts and mechanisms at a semantic level. For this reason and since simulation processing times

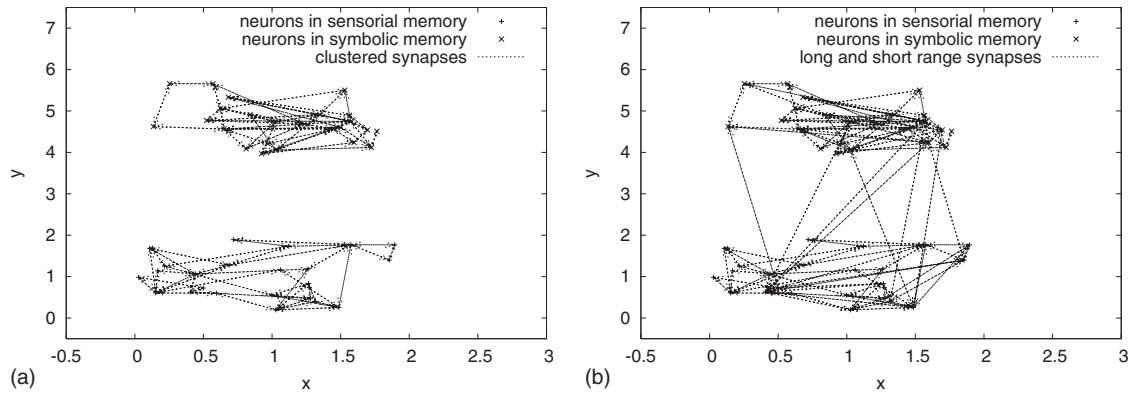

FIG. 3. (a) Network topology after clustering for $N=50$. (b) Network topology with long-range synapses.

are large, we consider small values of N in our experiments. Such values are small for realistic brain studies. However, since the short-range microscopic biological mechanisms in our algorithms are scalable, we expect that this semantic level should be amenable to mapping to a biological substratum. We focus our model on the neuronal mechanisms that represent neurosis as a pathology related to memory associativity. Our description concentrates on the interactions between and within the sensorial and symbolic modules, to explain how the possibility of memory traces to become conscious is sensitive to the intensities of these connections, and how they can be reconfigured in a learning process to enhance or inhibit associativity. In the real brain, these modules are not symmetric, and neurons and synapses function differently in different brain regions. Simulations of most system configurations are very time consuming on a sequential processor, even for the small systems that we have shown here. We plan to parallelize these algorithms in order to simulate significantly larger systems.

In Fig. 3, we show topologies generated for a network with $N=50$ and $N_{sens}=N_{symb}=25$. Other parameter values are $\sigma=0.58$ and $\eta=0.1$, and memory sheets have size of 1.9×1.9. To simulate the weak connectivity of neurotic traces to symbolic processing brain areas, synapses connecting different memory modules are multiplied by $\zeta=0.5$, defining the patterns initially stored by this network as neurotic. Figure 3(a) shows the topology after executing only the clustering algorithm, and Fig. 3(b) shows the corresponding topology after long-range synaptic generation, illustrating the self-organization of the network in a clustered, hierarchical manner. Of the memory patterns stored by the initial neurotic topology in Fig. 3(b), 30% remained after working-through, showing that the network adapts with the working-through simulation, freeing itself from some of the "neurotic" states. For smaller values of ζ, the network has difficulties in associating unconscious traces with symbolic memory, i.e., learning. Values of ζ that are too large allow excessive associativity, which may lead to unacceptable associations.

We generated 10 000 different neurotic topologies, for different values of N and spatial dimensions, from the same initial parameter values specified above and measured the average node degree (k) distribution, for these complex network structures. In Figs. 4 and 5, the discrete symbols represent the values found in our simulations. Figures 4(a) and 4(b) show distributions for $N=50$ and the curve is a fit by a Poisson distribution $P_\lambda(k)=\lambda^k \exp(-\lambda)/k!$. It is known that random graphs follow the Poisson distribution of node degrees.[17,19,20] Our networks are not random, but the spatially homogeneous Gaussian allocation of synapses in step 2 of the clustering algorithm shows a Poisson distribution of average node degrees in Fig. 4(a). Figure 4(b) shows the average node degree distribution after all the steps of the clustering algorithm, and the deviation from Poisson distri-

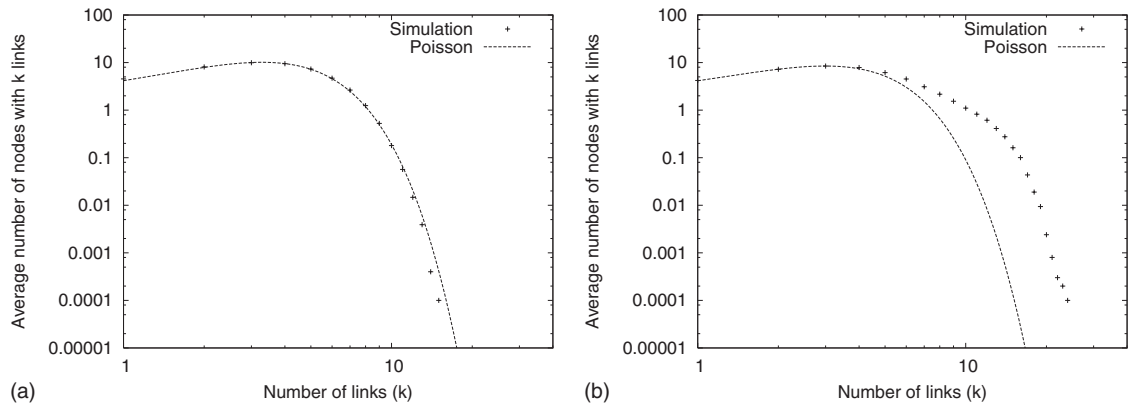

FIG. 4. (a) Average node degree distribution before clustering (after step 2), for $N=50$. The curve is a Poisson fit corresponding to $N=50$, with $\lambda=3.8$. (b) Same distribution after clustering. The Poisson curve corresponds to $N=39$ and $\lambda=3.5$.

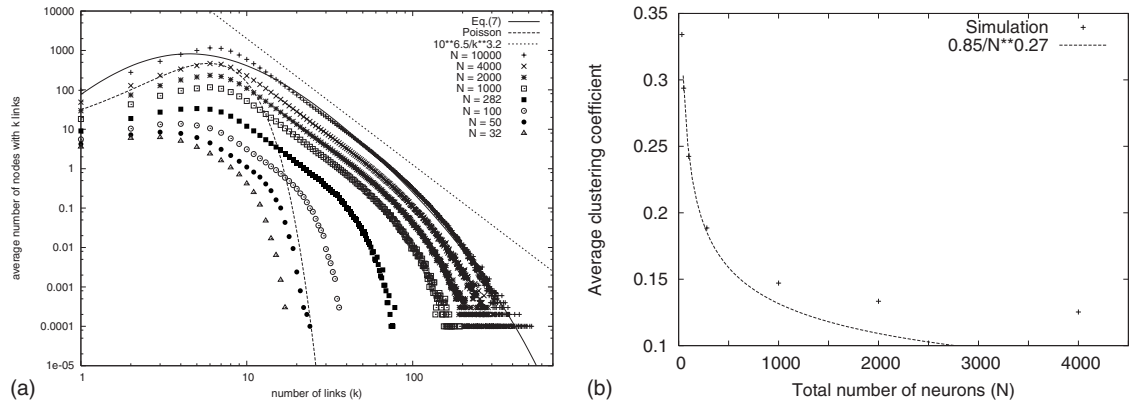

FIG. 5. (a) Average node degree distributions for various N. The fit by Eq. (7) corresponds to $q=1.113$, $p_0=610$, $\delta=4.82$, $\tau=2.34$. and $\mu=0.014$. The Poisson fit corresponds to $N=2950$ and $\lambda=6.4$. For large k, there is an exponential finite size effect. (b) Average C_{coef} for different N.

bution for higher values of k may be attributed to the cooperative-competitive biological mechanisms described in Sec. II. These mechanisms introduce structure, and the deviations from Poisson confirm that the resulting network has a nonrandom topology.

For larger values of N, Fig. 5(a) shows a power-law distribution for large k, with exponent of $\gamma \approx -3.2$. This behavior is characteristic of many biological systems and indicates scale independence.[20,31] For $N=4000$, the deviation from a Poisson distribution for $k>10$ is quite evident, as shown by the fit. Smaller values of k correspond to neurons that did not participate significantly in the competition-cooperation process, and therefore, the distributions for small k values are well fitted by Poisson forms.

In Fig. 5(b) crosses depict the calculated average clustering coefficient C_{coef} values, for the network configurations, as a function of N. As it is well known, C_{coef} is negligible in the case of an Erdös–Rényi random network. We notice that the dependence for larger N is much weaker than that for a network in which linkage is performed through the preferential attachment criterion, for which $C_{\text{coef}} \propto 1/N$.[43] To illustrate this, we show as a full line the function $1/N^{0.27}$. Although it follows the main trend of C_{coef} obtained in our simulations, this function overpredicts the drop for large N. The clustering algorithm presented in this work thus appears to lead to networks, for which the biological mechanisms establish some different topological properties than the traditional algorithms, studied in the complex network framework.

Figure 5(a) also shows an approximate fit by a generalization of the q-exponential function[20,44] given by

$$P_q(k) = p_0 k^{\delta} \frac{1}{\left[1 - \frac{\tau}{\mu} + \frac{\tau}{\mu} e^{(q-1)\mu k}\right]^{1/(q-1)}}, \tag{7}$$

where p_0, δ, τ, μ, and q are additional adjustable parameters. The curves indicate that asymptotically, the power-law and generalized q-exponential fits are appropriate, with $q \approx 1.113$. This is a common feature of many biological systems and indicates that they may not be well described by the traditional BG statistical mechanics but rather by the more recently proposed nonextensive statistical mechanics.[20,22,23]

We have explored this approach and have proceeded to model the memory function with GSA,[23] derived from the just mentioned nonextensive formalism. The method constructed in this way is a generalization of the BM employed in our previous work. In GSA, the probability distribution of the system's microscopic configurations is not the BG distribution, assumed in the BM, and this should affect the chain of associations of ideas which we are modeling. To illustrate this, we compare the energies of the patterns accessed by the BM and GSA at two different initial temperatures. Since we are searching for local minima, we use lower initial temperature values and higher values of the annealing schedule α.

Simulation of memory access is very time consuming and thus, in the following simulations, we have analyzed smaller networks with $N=32$, $N_{\text{sens}}=N_{\text{symb}}=16$. Memory sheets have size of 1.5×1.5 and $\sigma=0.58$, $\eta=0.1$, and $\zeta=0.5$, as before. The simulation experiment followed was to perform up to 10 000 minimization procedures, starting each one from a different random network configuration. When a new pattern is found, it is stored and the procedure is repeated from other random starting configurations, otherwise the search stops. We note in Figs. 6(a) and 6(b) that, for $T=0.2$, there are patterns found by GSA that are not found using the BM, while the opposite takes place at $T=0.1$ [Figs. 6(c) and 6(d)]. For the procedure described above, GSA appears to visit state space more loosely at higher temperatures, while the traditional BM visits state space more uniformly at lower temperatures. For lower temperatures, the BM functions more like a gradient descent method, and randomly generated patterns will stabilize at the closest local minima.

In order to understand the features of GSA that led to the results presented in Fig. 6, we compare in Fig. 7 the frequency with which the different minimum energy states corresponding to patterns are found, with the BM and with GSA, for $q_A=1.3$. Both calculations were performed at $T=0.2$, which corresponds to Figs. 6(a) and 6(b). We notice that, in the case of GSA, the frequency with which the hardest to detect patterns are found is much larger than the corresponding ones in the BM. In particular, several patterns that are not found by the BM are detected employing GSA. This corresponds to the gaps encountered in the spectrum shown in Fig. 6(a). One should remark that, obviously, if the

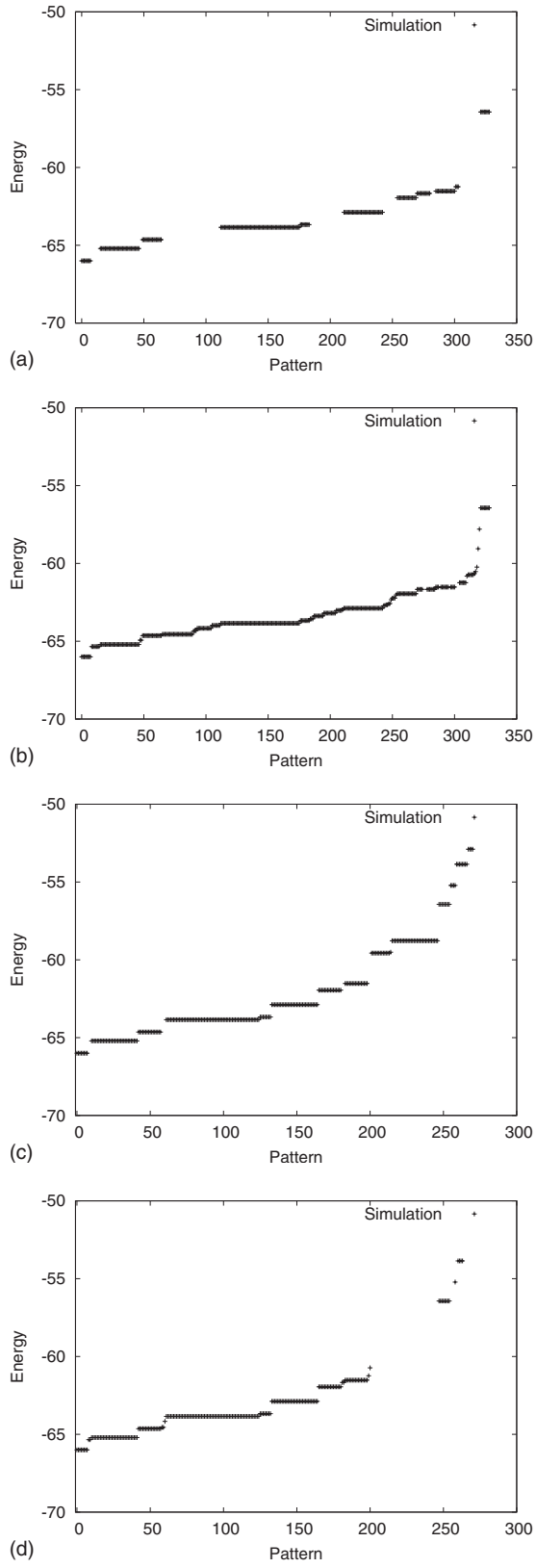

FIG. 6. The numbers taken as abscissas in (a) and (b), and in (c) and (d), identify the same patterns. (a) Energy of stored patterns visited by the BM for $T=0.2$. (b) Energy of stored patterns visited by GSA for $q_A=1.3$ and $T=0.2$. (c) Similar to (a) for $T=0.1$. (d) Similar to (c) for $q_A=1.3$ and $T=0.1$.

number of iterations allowed in the simulation experiment was increased, the patterns detected through both procedures should eventually coincide, and the gaps disappear, but our intention is to find minima without an exhaustive search procedure, but guided by the probability distribution function for network states. GSA tends to prefer the lower energy states, but will also find, with low probability, higher energy states. One can observe a power-law upper limit for the frequency of visits, as a function of energy for GSA. The BM tends to visit states with a more uniform distribution of frequencies, as is expected from the characteristic of the locality of visits of state space, which we mentioned in the end of Sec. II.

IV. CONCLUSIONS

We have proposed a neural network model, based on known biological brain mechanisms, which describes conscious and unconscious memory activities involved in neurotic behavior. The model emphasizes that symbolic processing, language, and meaning are important for consciousness. Although biologically plausible, in accordance with many aspects described by psychoanalytic theory and clinical experience, and based on simulations, the model is very schematic and we do not sustain or prove that this is the actual mechanism that occurs in the human brain. It nevertheless seems to be a good metaphorical view of facets of mental phenomena, for which we seek a neuronal substratum and suggests directions of search.

Temperature and noise in the simulated annealing process that occurs in the model for memory activity should be related to associativity. Very high temperatures allow the production of logically disorganized thought because they allow associations of excessively distant, usually uncorrelated ideas. This is common in the low signal-to-noise ratio, characteristic of low dopamine neurotransmitter levels in the brain, associated with hallucinations and psychotic states.[45,46] In the model we have presented, temperature regulates associativity among memory configurations, lowering synaptic inhibition, in an analogy with the idea that freely talking in analytic sessions and stimulation from the analyst lower resistances and allow greater associativity.

The model analysis, which we have presented here, indicates that BG statistical mechanics may not be most appropriate to describe biological systems, such as the complex networks with cooperative and competitive neuronal mechanisms that govern organization of topology, as we have introduced in our model. These mechanisms are very characteristic of much of the brain's functioning. A description according to nonextensive statistical mechanics seems to be more adequate and suggests the use of a GSA algorithm, under certain conditions, to model memory functioning and the way we associate ideas in thought. The study of network quantities such as node degree distributions and clustering coefficients may indicate possible experiments, that would validate models such as the one presented here.

We are continuing the systematic study of the parameter dependency of the model and also expanding and generalizing it to treat new features and phenomena. If possible, we will try an interpretation of these parameter dependencies as

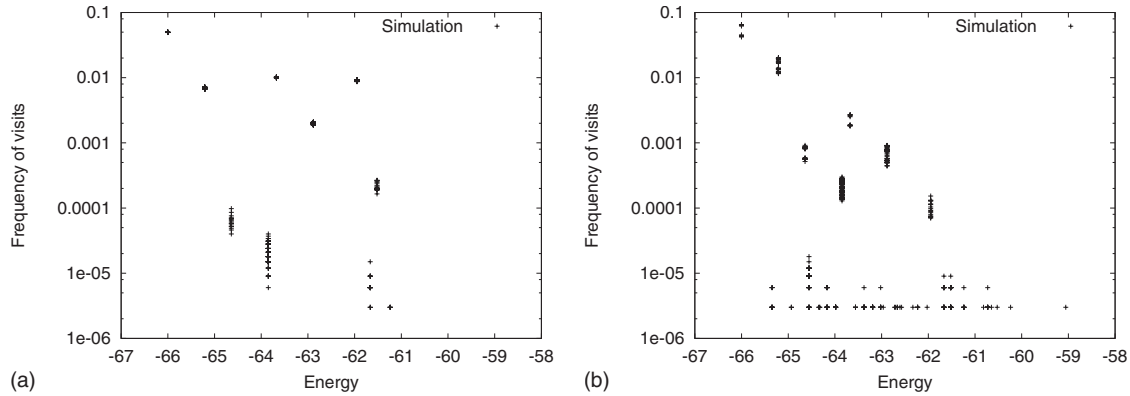

FIG. 7. (a) Visiting frequency to stored patterns by the BM for $T=0.2$. (b) Similar to (a) for GSA at $q_A=1.3$ and the same temperature.

associated with memory functioning in the brain and psychic apparatus functioning. In particular we would like to interpret more deeply the relation of model parameters to the effects of neurosubstances.

We would also like to connect the present model with experiment. A possibility could be through comparison of fNMR brain images associated with neurotic processes, before and after psychoanalytical working-through. These images could reveal brain areas activated by stimuli that generate neurotic acts before working-through. One could then test if, after a period of working-through that decreases symptoms, brain areas associated with symbolic processing were increasingly activated, for the same stimuli that once generated neurotic acts. In order to assess whether the changes observed should be attributed to changes from unconscious to conscious memories, similar measurements should be performed with neutral (non-neurotic) concepts. Also images of neurotics that engage in working-through should be compared to those that do not. We believe that the schematic model described in this work may prove to be a useful tool to help understand such experiments.

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