Towards a unified model of Pavlovian conditioning: A solution to the reconsolidation problem

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Abstract: - Memory formation is a complex dynamic process. When memories are formed they are labile at first but gradually become progressively consolidated into stable traces via synthesis of new proteins. Recent rediscoveries show that after reactivation, a consolidated memory can become transiently sensitive to updating, enhancing or disruption and needs to undergo a process of restabilization, known as reconsolidation. The reconsolidation has challenged the classical view of how memories are consolidated over time and stored. The reconsolidation process is not fully understood, and theories about the nature and function of memory reconsolidation remain controversial. To reconcile many of the opposed views on reconsolidation, according to [43], a new theory of memory is required that would have to encompass: (i) the stabilization process after new learning, (ii) the stability of a non-reactivated memory, (iii) the lability of reactivated memory, and (iv) the boundary conditions for reconsolidation. We believe that the Theta-Regulated Attention Theory [62] and the "Neurolocator" model [25] as its implementation, can meet the above requirements. These theory and model are also able to answer the most difficult and unresolved outstanding questions of consolidation/ reconsolidation studies.

Key-Words: - consolidation, reconsolidation, novelty, hippocampus, theta-attractor

1 Introduction

When fearful memories are formed they are initially labile but gradually become consolidated into stable persistent traces via synthesis of new proteins [36]. Later, retrieval of a consolidated fear memory engages in two seemingly opposing mechanisms: reconsolidation and extinction [31], [16]. Reconsolidation occurs when these consolidated fear memories return to a transient labile state following their retrieval from which they, under certain conditions, could be updated [30], [42], enhanced [61], [29], [19], or disrupted [44], [38], [53], and then stabilized to persist in an inactive state. Consolidation and reconsolidation are similar in that they share the theta oscillatory mechanism [45] and the molecular mechanism [1], and they both require an intact hippocampus [11], [60]. However, there are some essential dissimilarities between the two processes. These dissimilarities result from different arousal levels during training [35], different brain areas and circuits involved [1], and different temporal dynamics revealed, so that the period of lability, the reconsolidation window, will only persist for some time after retrieval [14] , [15], [33], [37] , [6], but even during this period reconsolidation may prove impossible under some boundary conditions. such as concomitant extinction [16], [47], [59], [48], intensive training [63], and in some other conditions [30].

The explanation of these data is impossible in terms of traditional theories of memory such as [3], [41] and their modern extensions involving reconsolidation [4] , [66] , [64], [57], [54].

2 Problem Formulation

The main problem with reconsolidation, according to [43], is that there seem to be no the ways yet in which the consolidation theory can be changed to accommodate the main reconsolidation finding. At the same time, it was suggested [43] that the re-emergence of the reconsolidation phenomenon may provide grounds for revising the theory of memory. Such a new theory would have to address: (i) the stabilization process after new learning, (ii) the stability of a non-reactivated memory, (iii) the lability of a reactivated memory, and (iv) the boundary conditions for reconsolidation. We maintain that the Theta-Regulated Attention Theory [62] and the "Neurolocator" model [25] based thereupon, can meet the above requirements and can answer the following most difficult and still unresolved questions of the consolidation/ reconsolidation studies:

1) A major question is how memory reactivation is determined at the mechanistic level

to be updated [30] and what returns a memory to a labile state during remembering [7].

2) Another question is when memory reactivation triggers reconsolidation and when it contributes to extinction [64]. The nature of the interrelationship between extinction and reconsolidation is still an open question [13]. What is the nature of numerous negative results in relation to reconsolidation [30]? What are the true boundary conditions for reconsolidation [42]?

3) The central question is the role of the hippocampus in reconsolidation. Why does the hippocampus become critically involved again after reactivation despite its time-limited role in the dominant theory [11], [60]? What mechanism in the hippocampus is responsible for memory retrieval and hence for the requirement of protein synthesis for reconsolidation [64]?

4) Numerous studies provide strong evidence that the expression of specific proteins during an early and temporally limited phase after learning is necessary for consolidation and reconsolidation of long-term memory and for synaptic plasticity. This conclusion is still challenged by potential existence of non-specific effects, although there are a number of different approaches that all seem to converge on the concept that specific protein synthesis plays an essential role in the stabilization of both new and reactivated memories. The functional processes that this protein synthesis subserves during memory consolidation and storage still remain to be understood [2].

5) The issue of whether experimental amnesia, introduced to prove reconsolidation existence, is a storage or retrieval deficit has been debated for decades without resolution. Is it primarily because the applied paradigms are unable to differentiate between these two deficits [42], or is this issue irresolvable perhaps, in wich case there is no need to assume fundamental dissimilarity between the two mechanisms [4]?

6) What is it that physically persists in long-term memory? It is futile to try to understand consolidation or any possibility of reconsolidation without addressing the issue of persistence [12].

3 Problem Solution

Before answering these questions, we shall describe in brief the architecture and the key points of the "Neurolocator" model [25] and shall show how it works in the case of Pavlovian conditioning [26]. This model is designed as a distributed system of a large number of peripheral oscillators (POs) coupled with a central oscillator (CO) by two-way connections. The POs are not coupled with each other and interact via the CO. It is presumed that the septo-hippocampal system plays the role of the CO, and cortical microcolumns act as POs. Synchronization of the CO with all POs, or with some part of them, is interpreted as synchronization of oscillations between the septo-hippocampal system and some regions of the cortex. The synchronization mechanism is similar to that of a phase-locked loop (PLL)[1] system with multiple inputs. Those POs, which are phase-locked by the CO, form the 'focus of attention" governed by novelty detection. Learning and consolidation are possible due to the *Isolability Assumption,* which states that when the number of POs locked in an ensemble reaches a critical value, their *labilities* tend to be equalized, i.e., the oscillators that are gradually brought to a common rhythm in an ensemble will change their *natural frequencies* towards a common one, thus implementing isolabile coding of information.

Let us try and explain how this system of oscillators can represent, memorize, consolidate, and reconsolidate sensory information. Firstly, the neuronal representation in the system is configurational, so that its spatiotemporal pattern of synchronized POs is formed as a unitary representation of a particular stimulus, where each activated PO represents a distinct feature of a stimulus, such as a color, a form, odor, tone pitch, etc. Secondly, to fix this particular configuration, the activated POs change their natural frequency towards that of their mutual synchronization. This fixing consolidation starts during online stimulus presentation, but ends as offline improvement during rest or sleep. Thirdly, POs having different natural frequencies can be concurrently recruited into other activated configurations, thus contributing to the lability of reactivated memories and the unlimited memory capacity of the oscillatory system. The newly formed configuration is subject to the above fixing consolidation process, or "reconsolidation", which does not require complete disruption of the previous memory configuration. For comparison, a special mechanism was recently proposed which can disrupt the consolidated memory before stabilizing it through new protein synthesis [29], [30]. Although this mechanism strongly supports the original reconsolidation hypothesis [44], it is not unquestionable [52] and even contradicts to many findings (e.g. [6], [27], [48]). Our model does not require such a disrupting mechanism because, according to the "Neurolocator" model, all cortical

[1] PLL is an electronic control system that generates a signal locked to the phase of an input signal. A phase-locked loop circuit responds to both the frequency and the phase of the input signals, automatically raising or lowering the frequency of a controlled oscillator until it is matched to the input signal in both frequency and phase. The architecture with many inputs is presented by Lindsey [32, Fig. 3.37], but the general theory leans more to a single-input case.

memories are largely found in a "disrupted" state of permanent unbinding until they are reactivated and synchronized at the theta frequency of their initial learning. Memory is reactivated through oscillatory binding of spatially distributed memory features, and then it is consolidated or reconsolidated depending on the novelty and arousal level (and, hence, on the optimal theta-frequencies) during training without the need for complete erasing of the old memories. Similarly, it can be shown that our model is able to explain qualitatively all the data as described in the Introduction. However, in order to be able to answer the above mentioned unresolved questions we need to describe some mathematical properties of the "Neurolocator" model.

Mathematical analysis of this model, supported by numerical simulations, was carried out in [24] for stochastic neural networks and [34] for the case of deterministic neural networks with time-delayed connections of POs with the CO. Some results of this analysis will be used below in a discussion concerning the existence of global and partial synchronization, and the multiplicity of frequencies of stable synchronization. Specifically that every stable synchronization frequency has its *basin of attraction* in the space of initial condition, and that all attractors undergo *bifurcation* with a change of the system parameters. As a result, the system has a multitude of various coexisting stable oscillatory and point attractors that represent cortical memories. Strictly speaking, the stable attractors should be replaced by the *metastable states* of stochastic neural networks (see [24], [25]). However, the language of point attractors [5] , [34], [57] is preferable for this discussion since it is actually the same as in the connectionist modeling and will help to explain reconsolidation results to those unfamiliar with oscillatory models of memory and attention. We shall return to the metastable state phenomenon at the end of this paper where the main points of discussion on reconsolidation will be summarized.

When CS is paired with US, their corresponding oscillatory cortical representations are bound together in a mutual synchronization if they are inside of the *locking range* of the CO (maximal mutual detuning between CO and POs when synchronization is still possible, see [32]). The binding can become stronger, even without additional learning, through repeated synchronization by means of the CO (in sleep or rest), provided the natural frequencies of POs can be changed towards a common frequency of synchronization as suggested by the *Isolability Assumption*. This process of binding through synchronization with gradual change of natural frequencies of oscillators has been proposed [25, p. 155] as a model explanation of the consolidation effect. The rate of such consolidation is directly related to the pretraining frequency interval (the magnitude of differences between the initial natural frequency of POs and the frequency representation of CS) and to the strength of response to the CS. The POs that were tuned closer to CS and were more responsive to the CS completed their tuning changes within one hour whereas the POs that were tuned to more distant frequencies and were less responsive to the CS took about three days to complete their tuning shifts. This modeled process is similar to the consolidation of conditioning described in [65, p.281] for primary auditory cortical neurons, but it can be generalized to any modality by using a cortical microcolomn oscillator instead of a single neuron as a functional unit. From this perspective, an oscillatory mechanism for reconsolidation is similar to that of consolidation and extinction in accord with the data [45] and [46], but differs from them in the input novelty and the value of synchronization frequency resulting in different oscillatory attractors. For better understanding, consider the interrelations between the three components of conditioning, i.e. consolidation, reconsolidation and extinction memories, in terms of their attractors.

On the one hand, consolidation and reconsolidation of memories are known to be initially very similar because they have the same context and common CS-US association, but later updating with new information introduces some point of difference [30]. Reconsolidation will follow if we change perceptibly the basin of the original attractor by changing either its depth (memory strength), or width (locking range of synchronization), or the location of the minimum (novelty). Such modification is consistent with the finding that consolidation and reconsolidation correspond to activation of different brain areas, with some degree of overlapping [1] because they correspond to POs with different natural frequencies and locations. It is obvious that this modification and the ensuing reconsolidation can have the same mechanism of theta synchronization as that of consolidation (cf. [45]); and that reconsolidation does not require erasing of old memory traces, as suggested by the findings of [60]. At the same time, such modification and hence reconsolidation would be difficult to impose if old memories are very strong. In such a case, the stationary CO frequency is difficult to change, since in the above model it is roughly equal to the weighed sum of the POs' natural frequencies of old and new memories. Therefore, a strong old memory is a temporary obstacle to reconsolidation until the fading away of this old memory along with input novelty moves the CO frequency towards a new theta frequency that is a new attractor. This corresponds to the recent data showing that strong associative fear memories form a transient

boundary for reconsolidation and that such a boundary requires an intact hippocampus [63].

On the other hand, reconsolidation and extinction processes are known to be initially very similar because both are initiated by applying the same CS without a US, but they can diverge later by switching the memory process towards reconsolidation or extinction depending on the strength of extinction learning (e.g. [59]). In the last case, the basin of the extinction theta-attractor gets separated from that of reconsolidation, which imposes another boundary condition for reconsolidation as a result of extinction learning, leading to the desynchronization of CS and US representation. However, this boundary is transient because the ever-changing arousal (e.g. due to new contexts) can alter the theta frequency during extinction memory retrieval causing resumption of reconsolidation, which, in turn, renews or spontaneously recovers a conditional reflex (cf. [10]).

Thus, extinction and a strong old memory are both *transient* boundary conditions for reconsolidation. In fact, reconsolidation itself is known to underlie spontaneous recovery after fear extinction [58], [51], [17]. Therefore, disruption of reconsolidation can be an explanation of the two recently reported cases of persistent attenuation of fear memory in rats and humans [38], [53], or may be account for by the updating with non-fearful information as proposed by the authors of those reports.

This qualitative analysis of reconsolidation boundaries can be clarified by using the theta-attentional mechanisms of the "Neurolocator" model. Specifically, the process of consolidation through synchronization of many POs by the CO is accompanied by attention with different degrees of stability, depending on the task complexity and the arousal level. In particular, if consolidation takes place after relatively simple learning, such as pairing of CS with US, then in accord with the key properties of the "Neurolocator" model, the stability of attention against external interference will be an inverse U-shaped curve of arousal (reminding of the Yerkes-Dodgson law) with a small width and a comparatively high optimal level of arousal. But if consolidation is performed after an act of more complex learning, such as extinction or retrieval in a new environment, the stability of attention and memory trace would be represented by a lower and wider inverted U-shaped curve, with an optimal arousal at a relatively low level[2], as shown in Figure 1. Since theta frequency in the "Neurolocator" model

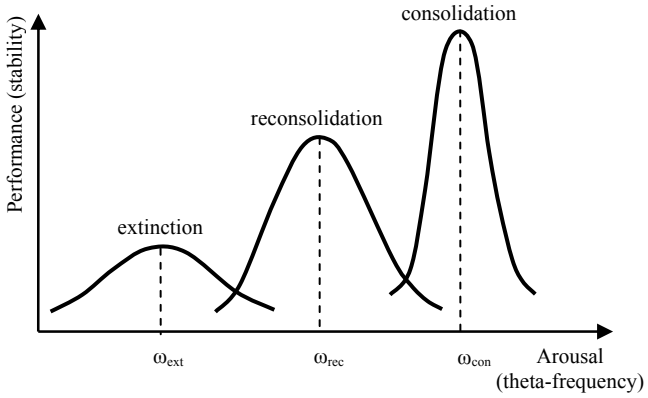

Fig.1. Schematic illustration of the Yerkes-Dodgson law as applied to fear conditioning. Performance of any task appears as an inverted U-shaped function of arousal or unspecific nerve excitation. Moreover, the range over which the performance improves with increasing arousal varies with the task complexity, so that the optimal level of arousal which corresponds to the best performance is relatively lower for complex tasks. Note that reconsolidation and extinction are complex tasks as compared with consolidation due to novelty of CS-noUS association and possible change of extinction context.

is proportional to arousal, each curve in Figure 1 can be taken to show the stability of different theta attractors corresponding to consolidation, reconsolidation, and extinction, respectively. Hence, reconsolidation as prolonged consolidation is impossible if the optimal theta after new learning (say, ω_{ext}) differs from that of consolidated memory (ω_{con}) by a value exceeding the PLL locking range of synchronization. On the other hand, reconsolidation occurs if novel information at the time of reactivation shifts the theta frequency to a lower value (ω_{rec}), as compared to that of consolidation (ω_{con}) by a value less than the PLL locking range, with the hippocampus playing the role of a frequency / phase comparator[3]. The fact of theta frequency decrease in response to the novelty, with the hippocampus acting as a comparator, has been recently discovered by Jeewajee et al [22].

[2] For detailed explanation of Yerkes-Dodgson law in terms of the "Neurolocator" model, see [23, p.337]. For numerical example of attention stability and its inverted U-shaped function of arousal, see [24, Fig.9.3] .

[3] Formally this can be expressed by the inequality $(\omega_{con} - \omega_{rec}) < AK$, where A is a magnitude of theta oscillations in the reconsolidation state and K is PLL loop gain (see [32]). The boundary condition for reconsolidation can be obtained by reversing this inequality. The boundary condition which takes into account the hippocampus acting as a comparator can be obtained by reversing similar inequality derived in [25, ineq. (3)].

So, our model can describe all boundary conditions found in fear conditioning as well as explain why they are so diverse and cannot be described by a single factor or parameter. It is important to note that a strong consolidated memory and extinction should be considered as a two-sided boundary, with one side acting as an "entrance", the other as an "exit" boundary for reconsolidation, and with their co-dependence on each other. This fact can be interpreted as competition or trace dominance in the experiments in which consolidation blockers erase the most active trace [16].

Now we can answer the questions listed at the beginning of this paper.

1) Memory reactivation upon retrieval in "Neurolocator" is actually restoration, or rather reconstruction of the isolabile configuration of the POs that existed at the last activation or acquisition stage but were soon brought into an inactive state of decayed oscillations. Being in that inactive state, memory traces are stabilized against disturbance, interference, superimposition of new information traces, damage by electric shock, etc. Memory reactivation is a system state of partial synchronization in which originally inactive decayed oscillations of POs are revived to be active again by simultaneous action of input stimulation, reset of septal theta activity, and elevated arousal from reticular formation in accordance with data [62]. To update memory by new information, the system should be in the same context (and hence near the same theta frequency) as during the initial learning. In the absence of novelty, the system reverts to its original inactive state in anticipation of a new stimulus without changing any parameter [62] that is without reconsolidation [18]. That explains the data of [8] which show that a pure unemotional contextual memory, unlike contextual fear memories, does not appear to undergo reconsolidation. In short, memory reactivation in our model starts with CS stimulation which causes nonspecific activation of reticular formation, resets the theta rhythm in septum and neocortex and thereby activates the septo-hippocampo-cortical system to detect the input novelty and update the old memory in response to a novel input or to revert the system to its initial inactive state if the input of familiar.

2) In general, not all new information will cause transition from consolidation to reconsolidation: information should cause relatively small changes in the theta frequency which will modify the same consolidation attractor. On the contrary, an entirely new attractor is formed by drastic changes while new learning is initiated by extinction. Hence, reactivation triggers reconsolidation or contributes to extinction, depending on the level of input novelty (such as partial familiarity, unexpectedness, context change, duration of extinction training, etc.). Indeed, when reactivation occurs in the same context (and, hence, approximately at the same theta frequency), it will usually be followed by reconsolidation, but when it takes place in a new context, it leads to an entirely new representation [39]. The true boundary conditions in the "Neurolocator" model, which separate the basins of two neighboring attractors, can not be expressed by a single factor or a fixed value of a single model parameter, but depend simultaneously on memory strength, age, arousal, learning intensity, etc. In short, all these system parameters determine the system theta frequency and the corresponding basin of memory attractors. The true boundary condition for reconsolidation can only be described in terms of the basin attractor parameters induced by the PLL system.

3) Traditional consolidation theories, such as the Standard Consolidation Theory [3] or the Multiple Traces Theory [41] cannot account for the reconsolidation phenomenon – the former, due to its incorrect supposition that after consolidation the hippocampus is no longer needed to retrieve memory [40], and the latter, due to its incorrect account of reactivations as multiple copies of memory stored in the hippocampus [11]. The main function of the hippocampus in reconsolidation is the same as in the Theta-Regulated Attention Theory [62], that is, it acts as a comparator of new and consolidated information processed for novelty detection and adjusts the theta frequency to an optimal value required for new learning and retrieval. The hippocampus alone is not responsible for novelty detection, retrieval, and requirement of protein synthesis – it is done by a global system.

4) The controversy concerning the specific proteins is resolved by the new mechanism of learning in the "Neurolocator" model based on the *Isolability Assumption* that calls for changes of POs' natural frequencies through nonspecific protein synthesis or some other molecular mechanism of lability changing. Specific protein synthesis is not required for such a general function, which is suggested by the finding that memory can be changed by different nonspecific molecular mechanisms even in its inactive state. Indeed, it is reported that post-translational modification of an existing protein may be sufficient to represent memory [50], [20], [49], and also that persistent memory depends on an ongoing activity of protein kinase long after memory consolidation is over [56], [55]. Careful reviews of this subject made in [21] and [20] stress the nonspecificity of protein synthesis inhibitors and show that the debate over the precise role of protein synthesis in memory consolidation has come to a stalemate. We propose a simple solution to this problem through nonspecific isolability learning which allows storage of specific memories due to

modality-specific POs and a new type of cortical plasticity analogous to that of auditory receptive fields.

5) The question as to whether post-reactivation amnesia is due to an impaired storage or impaired retrieval cannot be resolved by this model in view of circular causality in PLL. On the one hand, impaired storage results in a low amplitude of the POs' theta oscillations which cause low retrieval arousal as compared to arousal at acquisition and, hence, lead to a retrieval deficit. On the other hand, impaired retrieval results in low arousal; the low arousal leads to low amplitude of the POs' theta oscillations that cannot be differentiated from impaired storage or consolidation. In other words, low arousal as a cause of the retrieval deficit is also a cause of storage deficit, and vice-versa. Thus, our model can reconcile both views of memory processing, and there is no need to assume their fundamental dissimilarity.

6) The question of memory persistence has been thoroughly discussed in our previous work [25] and here we are giving its brief outline. According to the proposal made in [12], the persistence problem involves two questions. First, why does the engram endure sometimes for a lifetime in spite of the short-lived individual components of the cellular material in which the trace is registered? Second, how do memory traces persist over periods in which they are not expressed? Memories, according to the proposal [12], are encoded not by hippocampal LTP, as most theories suppose, but by spatiotemporal patterns of neural population activity. These memories may still involve synaptic plasticity, but their synapses are not necessarily the original ones, and may degrade, so that the memories do not remain intact, but are reconstructed after staying in the inactive state of physical metastability. Long-term traces persist in such a dormant inactive representational state until the reactivation in retrieval occurs. Concerning neural realization of this theoretical proposition, the "Neurolocator" model can be a valid option. Indeed, here memories are encoded by spatiotemporal configurations of globally synchronized cortical microcolumns oscillators which the natural frequency of which could be modified and preserved according to the *Isolability Assumption* by the synaptic protein synthesis, or some other molecular mechanism to fix a new natural frequency. By means of PLL action the "Neurolocator" is capable of generating or reconstructing various learned patterns of cortical activity, with theta rhythm being virtually a binding code for cortical feature oscillators. As for the stability in an inactive state, it is resolved here by automatic lowering of the parameter of arousal during rest or sleep, which leads to transition of the whole system or some part of it into a state of desynchronization and inactivity, resulting in the local effect of *persistent spots*[4]. By returning to normal arousal, given the appropriate cues, the system will automatically resume its global active state with a specific cortical configuration defined by the system's dynamics and specific cues. In short, the persistence problem is solved by two effects: (a) a local collective effect of spot persistence, a kind of long-lived physical metastable state [24]; and (b) a global system effect of partial synchronization in PLL which allows post-retrieval reconstructing of the various spatiotemporal configurations of cortical oscillators similar to the ones that existed during the acquisition, without the need of storing the original memory configuration. Thus, the "Neurolocator" provides a radically new solution to the issue of trace persistence in the memory system composed of nonpersistent elements.

The solution to the reconsolidation problem offered by the proposed model leads to several testable predictions:

Reconsolidation is a global system phenomenon (not local, say, amygdala-based); it should be an effect similar to physical metastability, but showing a frequency-specific plasticity and novelty detection.

- Synaptic modification, together with specific protein synthesis, plays a subsidiary, rather than a fundamental role in consolidation and reconsolidation. Theta rhythm is an organizing factor not only at the behavioral and neurobiological levels, but also at the molecular level of new protein synthesis.
- Long-term memories are normally ineradicable, but their behavioral and emotional impacts may be persistently attenuated by their intensive retrieval with properly timed extinctions, in some way resembling confessions with deep

[4] This effect can be described as follows. A two-dimensional computer network of 30x30 integrate-and-fire neurons with fixed nearest neighbor synaptic connections of identical strength starts with a spot of units of zero background taken as an initial state, and evolves as follows. The net activity dwindles to the DOWN state if the synaptic strength is small, so that the configuration consisting mainly of zeros establishes. On the contrary, if the synaptic strength is high enough, the spot spreads out, thus forming the UP state. A network with critical parameter values, however, is capable of remaining for a long time in a state close to the initial one (Kryukov et al, 1990; Kryukov, 2008). Lately, new simulations of the metastable states have been carried out with different initial configurations confirming the existence of the critical regime, UP and DOWN states, and anomalous fluctuations in-between (Borisyuk and Cooke, 2007).

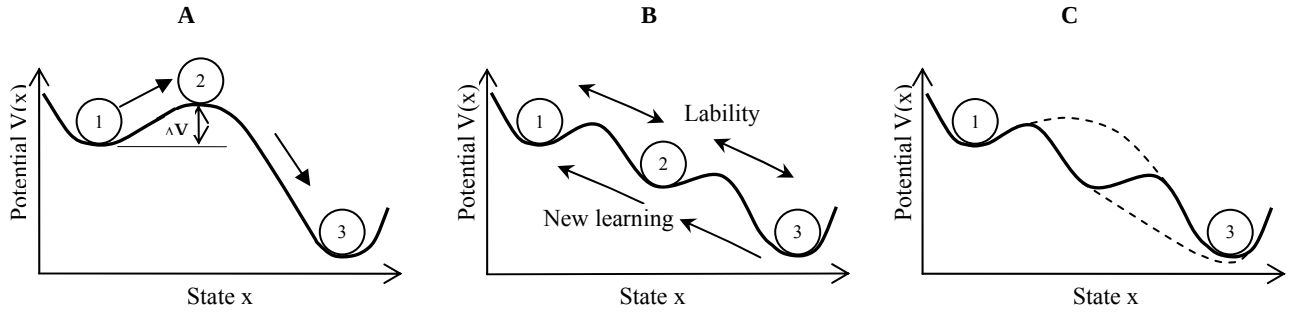

Fig.2. **A.** Metastability of neural nets in a weakly stable state (1), an unstable state (2) and a comparatively stable state (3) is defined by a state function V(x), called dynamic potential, which usually has one local and one global minimum (Kryukov et al, 1990). **B**. Special treatment of the system can result in two local minima of potential V(x), corresponding to the extinction state (1) and the reactivated state (2). **C**. There exist two ways of transforming the two-humped potential into a one-humped potential, which correspond to the boundary conditions eliminating the reactivated state (2).

repentance, described in one of Dostoevsky's best-known novels "Crime and Punishment".

4 Conclusion

Let us summarize the main points in the discussion of reconsolidation in terms of the metastaible states.[5] Although the existence of metastable states in neural networks has been proved independently from the existence of reconsolidation, the latter as reformulated by Nader et al (2005) may be treated as a problem of temporal stability of the metastable states of reactivated and non-reactivated memories. Figure 2 is a mechanistic picture illustrating the nature of such stability separately for extinction (1), reactivated (2) and consolidated (3) states:

(i) Stabilization of a memory process after new learning is achieved by recruiting new oscillators into the learned configuration, pulling their natural frequencies towards that of common ensemble rhythm, and fixing new natural frequencies via synthesis of new proteins.

(ii) The stability of a non-reactivated LTM memory is a result of complete desynchronization of the corresponding POs, thus putting the system into the state (3), with the global potential at its minimum. This comparatively stable state (3) can be transformed into a metastable state (1) or can become unstable (2) through reactivation and new learning, as shown in Figure 2A.

(iii) The lability of reactivated memory in state (2) is the capability of the system to change the frequency of its synchronization by recruiting new POs to represent the modified memories. This is capability acquired by metastable state (2) to move down or up of the potential function profile as shown in Figure 2B.

(iv) The boundary conditions for reconsolidation are defined by a special system treatment which transforms a two-humped potential into a one-humped potential, leading to disappearance of metastable state (2), as shown in Figure 2C.

Thus, an oscillatory approach central to this model allows meeting all the requirements of a new memory theory, as was proposed by Nader et al (2005) [43].

Acknowledgment

I would like to thank Hegumen Innokentiy and Olga Jedyaeva for their technical assistance, Vladimir Arkhipov for careful reading and comments, John Reynolds, Ludmila Melnikova, and Irina Reynolds for editorial assistance.

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[5] A system is in a metastable state when it is in equilibrium but may to fall into lower-energy states with only slight interaction. It is analogous to being at the bottom of a small valley when there is a deeper valley close by — a local stability of a system at a local (rather than global) minimum of a potential. For discussion of metastability in neural networks, its connection with physical phase transitions, its application to various brain structures and to the "Neurolocator" model see [24].

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