Q-EEG NeuroBioFeedback: Theoretical Foundations and Anticipatory Properties

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Abstract
Mental imaging is associated with brain receptor-mediated perception of both external (sensory) and internal (interoceptive and proprioceptive) signals. The accumulation of mental images from early developmental stages provides a reference bank for further adjustment of organism functioning, i.e. homeostasy. Noxious stimuli alter the morphisms connecting neural signals and associated mental images: closed normative feedback control loops are opened into paths, continuity is broken to disease and electroencephalogram (EEG) is correlatively modified. The NeuroBioFeedback (NBF) medical technology corrects mental images through voluntary training of the brain to restore its normal activity upon anticipatory adjustment to a EEG-piloted farther corrective goal. Duration of treatments appears as a function of rehabilitation rates.
Keywords: Mental Imaging; Molecular Continuity; Neurotherapy; Quantitative EEG.

1 Introduction

NeuroBioFeedback (NBF) is a medical technology based on the voluntary regulation of brain activity, piloted by quantitative electroencephalography (Q-EEG) [27]. While NBF has wide scope and high efficiency [28], a theoretical background was so far missing to explain its mode of action [35] and the processes underlying its physiological features [1]. Former works have explored the essential properties of the mathematical spaces embedding the functioning of brain [8], and identified mental images (M.I.) with fixed points of sequences of neuronal connexions [7]. However, while mainly M.I. from perception of outside information was so far considered, the present study takes its stand on two new concepts: (i) other types of mental images arguably must exist and benefit to internal homeostatic processes and (ii) brain mental imaging has recently been proved to be an anticipatory process [9].

Conscious perception has been shown to be founded on the association of sets of Banach-type mental images (1st kind M.I.) standing for the capture of outside signals with sets of Brouwer-type mental images (2nd kind M.I.) standing for self perception [9]. Then, it could be predicted that a nonempty intersection of these two kinds of M.I. would reflect the existence of perception from inside the perceiver's body. This conjecture has recently been supported by preliminary work raising the possibility of construction of mental images from receptor-mediated control of the molecular functioning of the organism [9], [10]. Proprioceptors react to interoceptive signals emitted from both conscious internal perception, like thurst, mood, etc. and unconscious perception of the autonomous processes, like metabotropic receptor

excitation associated to blood sugar control and other molecular and physiological parameters. When signals are translated in the brain into frequency components of the cortical activity, they become able to lead to the construction of associated mental images. Importantly, this work will also emphasize the need for existence of molecular endpoints paralleling mental images, therefore justifying the phenomenon of homeostasy as a condition dictated by the regular brain-body communication.

Table 1 lists some examples of receptors and their EEG responses associated with a sequence of brain processes ranging from highest to lowest levels of conscious perception (candidating for 2nd kind mental imaging and 2nd intersecting with 1st kind).

Table 1: EEG traces of various physiological processes, from least to most conscious states of perception. ST=serotonine. Range of frequencies are as follows: Delta (0.5-4 Hz); Theta (4-8 Hz); Alpha (8-12 Hz); Beta (12-35 Hz) including SMR (13-15 Hz); Gamma (40-60 Hz).

Physiological states a	Receptors (Rc) involved	EEG frequency changes: decrease	increase	References
Comatose sedation	TBE	Alpha	Beta	[16]
metabotropic endpoints	mGlu-Rc	oscillations: Beta.	Gamma	[29]
Anesthesia	GABA-Rc	Alpha	Beta	[27]
	NMDA-Rc	Alpha. Beta		[33]
Depression (anti-)	ST uptake		Beta	[22]
	benzodiazepine	Delta. Theta Alpha	Beta	[41]
Psychotism (anti-)	chlorpromazine		Beta-1	[44]
anxiolysis. sedation		Theta	Alpha-1,2. Beta	[5]
Blood pressure	Calcium channels		Delta. Beta	[18]
Sensorial stimuli	TBE		Beta	[13]
Time perception	TBE		Beta	[42]
High vigilance. SM and cognitive integration	TBE		SMR. Beta	[24]

TBE: area to be explored.

2 The Mathematical Space of Mental Imaging

The brain system has been identified with a topological space provided with the set-distance Δ (the symmetric difference between sets), which confers the system with the property of a Δ-complete space able to convert outside signals into perception information [6]. Now, we will focus on the internal brain-body communication. The following definitions will be used as working startpoints.

2.1 Definition of the System's Components

A Functional Homeostatic unit (H) is composed of functional systems denoted as $(W)\supset\{F, M\}$ whose members (F) and (M) respectively stand for autonomous

unconscious functions and conscious functions, further connected to molecular endpoints acting as sensor targets (S). The whole of homeostatic processes is denoted by ($\mathcal{H}$) such that for any (H): (H$\in\mathcal{H}$). Flows W are flawed with instability, while S will be shown to reflect stable parts.

Brain Receptors/Transducers (R/T) receive/send signals to functions of (W) as paths represented by mappings $f_{W,S}$: $\{R,T\}\mapsto\{W,S\}$ of brain receptors and transducers to molecular flows and endpoints. Direct mappings will be written f_W: $R\mapsto W$, while reciprocal mappings $f'_W{}^{-1}$: $S\mapsto R$ connect W back to brain receptors by intermediate of molecular endpoints S. The connection of functional systems W to sensor targets is denoted by mappings g_W: $W\mapsto S$ (Fig. 1). Components {R} are connected to neuronal configurations U(R) of the fuzzy type [8] while the corresponding mental images a(UR) are fixed points. The latter are reflected to transducers T by mappings ψ: $a(UR)\mapsto T(aUR)$. For simplicity of notation, to a configuration R_k one will write the corresponding preimage (S_k) and image $a(U_k)$.

2.2 Feed Back Loops

The composition $f\circ f^{-1}$ will denote in short writing a loop actually involving the composition $\varphi_H = f\circ(g\circ f_S^{-1})$. Manifold (L) denotes the set of brain-connected functions constituting the space $\mathcal{L}=\{\cup_{i,j\in I}\{(AFi)\cup(Mj)\}, \perp_{\mathcal{L}}\}$ where the set of combination rules $\perp_{\mathcal{L}}$ contains $\{\cup(f\circ f^{-1})\}$. The set of loops corresponding to specific functions (F_λ) will be denoted by: $L_H = \{\varphi_{H\lambda}\}_\lambda$.

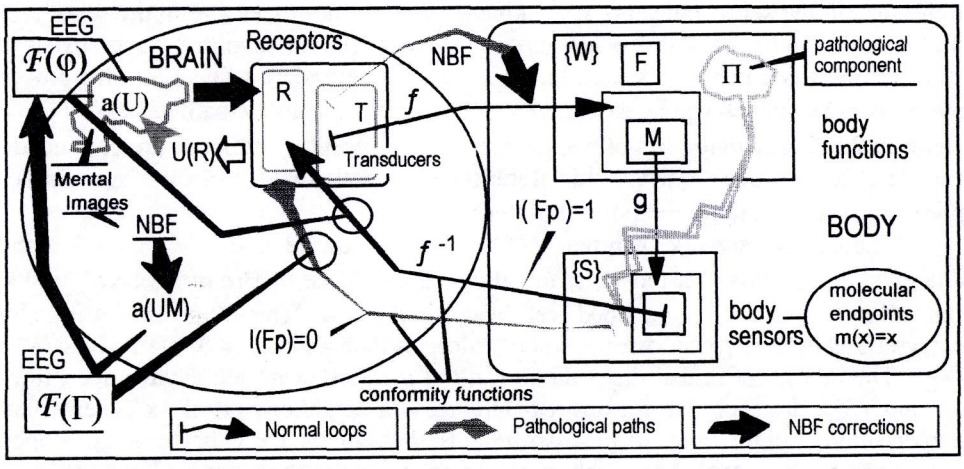

Figure 1: The sites of NeuroBioFeedback control. The indicatrix I(Fp) of the physiological functioning is valued in {0,1} and the associated conformity distribution function P(Fp) is valued in]0,1[. The mental image a(UM) is formed through Neuro-BioFeedback (NBF) training for a normative c(U) target. Electroencephalographic (EEG) signatures are denoted as functions $\mathcal{F}(\Gamma, \varphi)$ with (φ) the correct loop and (Γ) the path resulting from a pathologically opened loop.

Figure 1 schematically depicts the outlines of the system in its normal and pathological configurations (see Section 3).

Remarks 2.2. (i). All classes of biochemical effectors are represented in {T,W,S,R}. Regarding functional components {F,M}, any imbalance in one class will be reflected in some other class and finally be recorded by some sensor.

(ii). loop components will have to be assigned specific properties when continuity conditions are specified. This point will be examined below.

2.3 Continuity of Metabolic Functions

Conditions for existence of Brouwer's fixed points are continuity of functions and topological closure of relevant spaces. Their fulfilment have already been demonstrated in the space of brain functions, essentially as concerning mappings of discrete spaces into topological spaces [6]. Here, metabolic pathways remain to be examined.

Theorem 2.3.1. The space of metabolic functions is topologically continued.

Proof. Let $x \in W$ and globally call m the transformation and translocation functions mediated by enzymes, transporters and translocation facilitator systems. Then, for any x_i as a substrate a function m_i will give the product $m_i(x_i) = x_{i+1}$.

The chain of reactions continues with:

$$m_{i+1}(x_{i+1}) = x_{i+2}\ , \text{etc.}, \ldots, m_{i+k}(x_{i+k}) = x_{i+k+1} \qquad (1)$$

For any x_a a distance $d(x, x_a)$ can be defined as a number of intermediary steps (a measure in the set of integers), or as kinetic parameters (a measure in the set of real numbers). In both cases given any distance e between metabolites, there exists a distance h between precursors such that $d(x, x_a) \leq h \Rightarrow d(m(x), m(x_a) \leq e$. This proves continuity at x_a , which can be extended over and across metabolic pathways.

Theorem 2.3.2. The mappings of body functions into receptor endpoints are continued.

Proof. Let $x_j \in W$: there exists some iteration of g such that: $g^{n+k}(x_j) = x_k$, further appearing as an accumulation point. In effect:

There exists some k such that $g^{n+k+1}(x_j) \approx g^{n+k}(x_j)$, i.e. $g(x_k) = x_k$. Call S(x) the adherence of x_k : this is a closed space allowing existence of Brouwer's fixed points. This allows space S to embed reference endpoints. This state is essentially characterized by a narrower distribution of values of the variables in S relatively to W.

The density of probability functions dP of x and S(x) around the average values <x> and <Sx> are such that for any x belonging to a neighborhood of <x> (i.e. within an open interval in the range of the distribution), there exists a S(x) belonging to a open interval about <Sx>, that is a neighborhood of <Sx>. Hence the reciprocal mapping of a neighborhood of g(x) is a neighborhood of x, which demonstrates continuity.

Theorem 2.3.3. Mappings of metabolic endpoints to brain receptors are continued.

A receptor R is activated by a subset S of the set of values of the effector's concentrations: f^{-1}: $S \mapsto R$. This case therefore reduces to that of theorem 2.3.2.

Thus, all needed continuity conditions are fulfilled.

3 Main Results

3.1 Mental Imagery and Control Loops

Lemma 3.1.1. The function of proprioception owns a norm and a value.

A function F_p of class F owns a norm $\|F_p\| = I_{Fp}$ as an indicatrix function $I_{Fp} \in \{0, 1\}$ depending on whether the system does not work, or works correctly. $\|F_p\|$ and $\|F^{-1}{}_p\|$ are not necessarily identical. A particular value of F_π is given by $|F_\pi| = I_{Fp} \circ P(F_\pi)$ where $P(F_\pi) \in]0, 1[$ is called a 'conformity function' corresponding to intermediate stages. This is supported by the Urysohn's theorem: in effect, let $|F|$ value a continuous function of class F_p (showed above to be continued) of brain's delta-metric space E into [0,1] and let the respective spaces of correct and altered physiological functions be denoted by (C) and (A). Then, $|F(x)|=0$ for any $x \in A$ and $|F(x)|=1$ for any $x \in C$, so that $|F(x)| \in]0, 1[$ in all intermediate cases (Fig. 1). Thus finally $|F_\pi| \in [0, 1]$.

Remarks 3.1. (i) This provides the corresponding space with fuzzy properties, and justifies the legitimity of the strictly probabilistic approach of enzyme activities [32].

(ii) The structure of the union $\{\cup |W_\omega|\}$ is similar to that of a momentum.

Lemmas 3.1.2. Molecular regulation is based on functionals of feedback loops.

(i) There exists neuronal chaining configurations U having neither preimage nor image of members of {L}. (ii) A configuration X of a state of (W) can lead to a set of neuronal connections U(x) for some $x \in X$ further converging to a fixed point a(Ux) [6]. Let a(UB) be an abstract M.I.: then Λ: {U(X),Y} $\mapsto$ {a(UX), a(Y), a(UB)} denotes a functional of loops whose origin can be surjective, injective or bijective. This opens semiological perspectives in connecting physiological states to brain control possibilities, and determines properties of physiological/molecular endpoints, as well as their relation with treatment duration versus efficacy (see 3.5.2).

3.2 Theorem of the Homeostasy

Theorem 3.2. The involvement of second kind mental images associated with molecular endpoints is necessary to the homeostatic control of the organism.

The proof involves three steps.

(i) **Lemma 3.2.1**. Strict homeostasy is described by bijective constraints on $\perp_\Lambda$ such that one mental image $a(U_k)$ is mapped into one and only one molecular endpoint configuration S_k.

Proof. the result comes from *demonstratio ad absurdum* . Suppose there exists $S'_k \neq S_k$ mapped from $a(U_k)$: this would mean that there exists a Brouwer-type fixed point accounting for molecular/physiological configurations differing from the normal homeostatic levels. Then, two possibilities would occur: (i) loops $\varphi(a(U),S,R)$ could be recursively enlarged, and no stable configuration of the body's properties would be reached. (ii) Receptors could no longer be able to capture information from S and loops would be opened into paths, so that no fixed points would be reached (see 3.2.2).

Such cases correspond to various forms of pathological states.

Corollary 3.2.1. The union of neuronal configurations provides a functional cognitive historic of the organism likely involved in the homeostatic reference bank described in section 3.3.

Corollary 3.2.2. The functional $\mathcal{H}$ is not independent from the abstract mental imagery, since injectivity of Λ stands on the existence of mental images a(UB) that would be independent of {(F, SF), (M, SM)}.

(ii) **Lemma 3.2.2** (of loop opening). Let Λ such that $\perp_\Lambda$ is injective. Then, there exists $T'_k \neq T_k$ resulting in W'_k mapped into $S'_k \neq S_k$ or S'_k mapped into $R'_k \neq R_k$: this denotes a break of homeostasy and the subject becomes a patient. Loops are opened in $\Gamma'_k = f_k \circ (\varnothing \circ f^{-1}_k)$ or $\Gamma''_k = f_k \circ (g_k \circ \varnothing)$ i.e. reduced to a path, either discontinued or continued. Hence the space of loops $\Gamma_{(W,S,R)}$ is open: this precludes the existence of Brouwer-type fixed points and thus prevents S from embedding reference endpoints.

However, there still exists a normative loop (φ_{Hk}) such that: $\varphi_{Hk} = f_k \circ g_k \circ f_k^{-1}$. Note that such loops are non commutative and would form a nonabelian group.

(iii) **Lemma 3.2.3.** NBF induces both quantitative and qualitative specificities.

The mental image a(Uk) is the origin of φ_{Hk} and also of Γ'_k and Γ''_k . Thus the metabolic turnover of the system (organism) will involve a sequence $\{\varphi_{Hk}\}$ of loops such that $\{\varphi_{Hk}\} \supset \{a'(Uk), \Gamma'_k, \Gamma''_k\}$. This sequence converges to some a'(Uk) such that:

(a) either $a'(Uk) \subseteq a(Uk)$: thus homeostasy is restored.

(b) or $a'(Uk) \supset a(Uk)$: Then set-distance $\Delta(a'(Uk), a(Uk))$ is a measure of the severity of the disease. This situation will need medical intervention.

NBF foundations can be now identified as providing the diseased organism with an anticipatory mental image $c(Uk) \subseteq a(Uk)$ by acting on the EEG trace of a(Uk) instead of trying to chemically influence space {W, S, R}.

Corollary 3.2.3. The visualization of a healthy state is a type-M particular case of a construction of mental images $a(UB_M)$ able to restore a strict homeostasy.

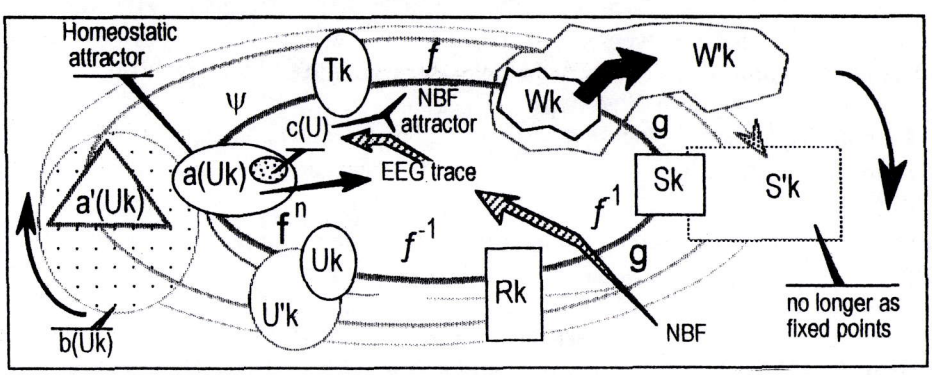

Figure 2: Representation of the homeostatic feedback loops. When the natural homeostatic attractor a(Uk) is failing, NBF supplies an alternative attractor c(U) produced from training the brain to restore its activity back to the normative EEG trace of a(Uk).

However, when specific receptors must be involved, just visualization not involving these receptors may be inoperative, while the correction of EEG frequencies necessarily implies a corrected receptor activity. This completes the proof.

3.3 Constitution of a Homeostatic Reference Bank

Proposition 3.3. Let $\{Bi\}_{(i=0\to n)}$ be a sequence of successive phases of embryonic development. Each phase includes $L(Bi) = F(Bi) \cup M(Bi)$, the part of W which is connected to brain. Let then $\{Aj\}_{(j=0\to m)}$ denote successive phases of the formation of the brain itself. Then the following immediately infers:
(i) There exists a surjective mapping f^{-1}: $\{Bi\} \mapsto \{Aj\}$ and (ii) The ordered sequence $\{\mathcal{B}_\delta\}_{(\delta=\beta\to n)}$ of phases which are images mapped by f converges to $(\mathcal{B}_n)$ such that $(\mathcal{B}_{n+1})$is homeomorphic with $(\mathcal{B}_n)$. Thus the main result: denote by $\{\mathcal{A}_\delta\}_\delta$ the domain of f such that: $\{\mathcal{A}_\delta\}_\delta \mapsto \{\mathcal{B}_\delta\}$ for any δ. Since this relation is bijective, sequence $\{\mathcal{A}_\delta\}_{(\delta=\beta\to n)}$ converges to $(\mathcal{A}_n)$ which is such that $(\mathcal{A}_{n+1})=(\mathcal{A}_n)$.
Remark 3.3. Mappings $f_{|A} : (\mathcal{A}_n) \mapsto (\mathcal{B}_n)$ are a restriction of f.

These results lead up to the following:
Theorem 3.3 (of homeostatic development). The homeostasy of the organism is founded on the set of mental images mapped from the initial members of the ordered set of phases of the biological evolution of the organism from the achievement of a correct embryonic development. Hence, all functional subsystems in the developping organism are driven to fixed points which are the corresponding brain mental images imprinted as references for further control over the body at adult stages

Clinical cases supporting these statements will be presented in section Discussion.

3.4 The NBF Practice

Proposition 3.4. The space of NBF is a functional constituted with a set of elements of rehabilitation of a function F_p such that ideally:

$$NBF(F_p) = \{a\,(F_p) : I_{a(Fp)} = 1\} \text{ where } I_{a(Fp)} = \|a(F_p)\| \qquad (2)$$

Remark 3.4. The limSup of the image of the set $\cup(L)$ of functions F and M (connected to brain control) is $\cup a(L)$ such that: $\cap\,(I_{\cup a(L)}) = 1$. This defines a new indicatrix function from the former ones.
Corollary 3.4.1. Since the limSup of NBF action is identical to the optimum state of the functioning of the organism, there exists no risk of adverse excess effect. In contrast, sustained NBF training could lead to maximization of brain/body capabilities: this proposition will be supported below in section 3.5.
Corollary 3.4.2. The number of sessions required for a given rehabilitation rate R%(p) on a patient p is an information about its former state. A basal number of sessions SNo can be required to drive the patient on the way to the formation of the first mental images. Globally, the rehabilitation rate will be related below to the number SN# of treatment sessions.

3.5 NBF and the Brain Responses

Lemma 3.5.1. A pathological state results from the opening of feedback control loops.

Let m(x) a metabolic function with physiological endpoints in an individual compound or family of compounds x. Then, the loop $\varphi_{(mx)}$ is bijectively mapped into a frequency spectrum $\mathcal{F}(\varphi_{(mx)})$ measurable in the cortex as the trace of M.I. a(Umx).

A pathological state opens the loop into a path $\Gamma_{(mx)}$, which therefore cannot be homeomorphic with $\varphi_{(mx)}$. Thus $\mathcal{F}(\Gamma_{(mx)})$ cannot be homeomorphic with $\mathcal{F}(\varphi_{(mx)})$.

The spectrum of frequencies is altered into a form $\mathcal{F}(\Gamma_{(mx)}) = \mathcal{F}(\Pi \perp \varphi_{(mx)})$ where Π denotes a pathological causality component (see Figure 1).

Through NBF training there occurs a creation of mental images a(U$\underline{mx}$) such that: the 'curative functional' $(\Pi \perp \varphi_{mx}) \circ (a(U\underline{mx}))$ is homeomorphic with φ_{mx} which implies that in its developed form $(\Pi \circ a(U\underline{mx})) \perp (\varphi_{mx} \circ a(U\underline{mx}))$ it is either homeomorphic with or equal to φ_{mx} reflecting m(x). In the latter case, NBF implies a resulting $\Pi = \emptyset$ while in the former this implies that constraints exist on the laws ($\perp$ and $\circ$) resulting in a cancellation, at least apparently, of the pathological component Π (Fig.3).

Corollaries 3.5. The above results imply a clause of specificity of the anticipatory mental image and a clause of specificity of the pathology. If these constraints are not fulfilled, then the anticipatory mental image acting for the rehabilitation of the patient may be partly active on another target or partly incomplete. Thus, at least the number of sessions SN# required for healing will be increased. This introduces the next proposition.

Proposition 3.5.2. The duration of treatments is a function of at least two variables: the targeted rehabilitation rate and the status of patients' initial symptom loads.

Proof. There exists as many feedback loops as there are different endpoints of the (S) type associated with clinical symptoms and their severity. Hence, an equation of SN# as a function of the number of initial symptoms (Sy) may be tentatively proposed as follows. Let b_i denote a function of cleaning of the corresponding symptoms (for instance, one can have $b_i = (1 - Sr_i / Sy_i)$), a_i the eigenweight of signs Sy with regard to the global disease η a coefficient of shape, and Sr the final symptom load. Then:

$$SN\# \propto \sum_{i=1 \to N} \{b_i \perp [a_i (Sy_i)^{\eta i}]\} \cup f(SN_o) \qquad (3)$$

The rehabilitation rate R% is a function of b_i, the above relation is bounded by R% = 100%. $f(SN_o)$ is a basal number of sessions possibly needed to start the first corrective loops. This will be detectable by clinical correlation studies.

Two major possibilities follow the NBF action: (i) either the curative functional maps a attractorwise-produced $\underline{m(x)}$ to an injective reciprocal image of the normative m(x) or (ii.) $\underline{m(x)}$ is injective image of m(x). In the first case the image a(U$\underline{m}$) is necessarily mapped into a molecular effect situated within the range of homeostatic needs: then, SN# may be reduced to its lower boundary. In the second case, there exists a nonempty distance $\Delta(\underline{m(x)}, m(x))$ in $\underline{m(x)}$ and SN# may be proportionaly

increased since treatments will have to be continued until this distance moves closer to emptiness.

Taken together, these results predict the occurence of specific functions mapping the rehabilitation rates and the duration of NBF treatments.

Let NBFx denote in a simplified form a treatment targeted to function x and let Hx be the normative homeostatic level of x (as reflected in S(x)-type endpoints). The results will be amenable to three configurations of $\{\underline{m(x)}, m(x)\}$ (Figure 3) in which $\underline{m(x)}$ is topologically closed [6], [7] while m(x) is open (e.g. like a bell-shaped distribution of cellular components is open on the interval of the variable values).

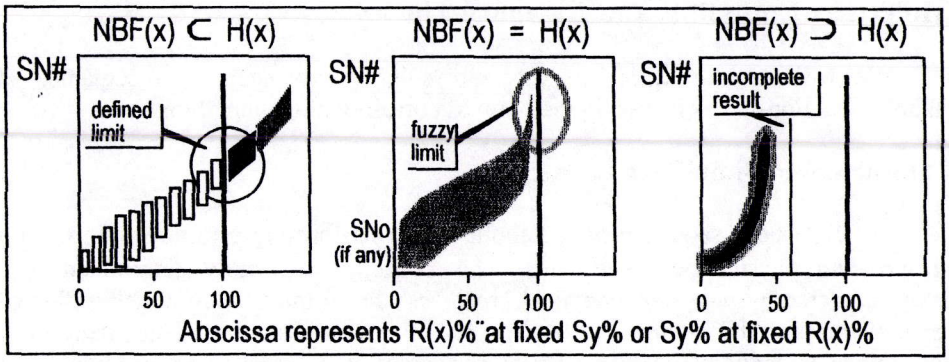

Figure 3: Predicted relations between the duration of NBF treatments (here measured by the number SN# of treatment sessions) and either the rate of rehabilitation (R(x)%) of patient's impaired functions at fixed initial symptom load (Sy) or (Sy) at fixed (R(x)%. H(x) denotes the normative homeostatic control over x and NBFx is the simplified notation for NBF action upon x.

NBFx $\subset$ Hx $\Rightarrow$ $\underline{m(x)} \subset m(x)$: then $\Delta(\underline{m(x)}, m(x)) \in m(x)$ and the duration of NBF treatments up to $I_x \approx 1$ is minimalized. This implies that there exists a nonempty neighborhood $\mathcal{V}(mx)$ which is included within the range of NBF control, which means that ultimately, other molecular/physiological functions related to x and function F(x) are stimulated.

NBFx = Hx $\Rightarrow$ $\underline{m(x)} = m(x)$: then $\Delta(\underline{m(x)}, m(x)) = \emptyset$ and $I_x = 1$. Here, the duration of treatment will be simply bounded by the initial symptom load and the restoration rate. Full restoration may be fuzzily attained due to W and U features.

NBFx $\supset$ Hx $\Rightarrow$ $\underline{m(x)} \supset m(x)$: then $\Delta(\underline{m(x)}, m(x)) \in \underline{m(x)}$. This corresponds with the indicatrix $I_x < 1$ since there exists a part of $\underline{m(x)}$ (and therefore of the related $S(\underline{x})$) which escapes the boundaries of homeostatic requirements. In this case, it is likely that only an incomplete restoration is achievable, within a finite duration of treatment.

Remarks 3.5. The restoration from pathological states and the recordable correction of brain frequencies are not necessarily timely correlated. In contrast, there may occur shifts in their respective occurrence. In effect, a correct EEG pattern can be recorded if and only if the totality of molecular and mental endpoints send correct signals to the totality of brain receptors.

This means that a stable restoration of normative EEG spectra should follow, rather than precede, the restoration of metabolic functions. On the other hand, the NBF treatment is targeted to induce short-term corrections of frequencies: the latter in turn influence metabolic endpoints in a desired way, while such frequency changes may not last until the altered functions are stably restored. Therefore, restoration of EEG patterns may provisionaly precede or parallell, while it should more durably follow, the restoration of physiological functions.

All these points are currently under verification by clinical studies.

4 Molecular Endpoints and Category Theory

We will now examine the way molecular events such as a sequence of metabolic reactions are reflected in the brain as corresponding mental images.

4.1 Morphisms in Brain-Body Connection

Let $\{F_i\}_i$ be a sequence of metabolic reactions forming a turnover flow, with corresponding physiological endpoints. This sequence is fuzzily determined (e.g. weakly regulated by purely biochemical processes), but it must be ultimately reflected in mental images a(U), which are fixed points, i.e. stable structures. Thus, there must exist molecular endpoints standing for preimages of a(U). Most likely candidates for this function are provided by structures abovely called S. Then, there must exist a sequence $\{S_k\}_k$ indexed on a subsequence of $\{F_i\}_i$ such that the image of $\{F_i\}$ by f^{-1} is $\{R_h\}_h$ ($h<k$). Consider two states, e.g. $\{S_1, S_2\} = g(\{F_{x,i}\}, \{F_{y,i}\})$ in turn mapped into $f^{-1}(S_1, S_2) = \{R_1, R_2\}$. There exists specific relations in the body, $(R_1 \rho R_2)$ in brain and $(a(U)_1 \alpha a(U)_2)$ at mental image level. From the above results, one must necessarily have the correspondences: f^{-1}: $\{(S_1 \sigma S_2) \in S\} \mapsto \{f^{-1}(S_1) \rho f^{-1}(S_2) \in R\}$. Since neuronal connections (U) have fuzzy-like behavior until their sequence reach their fixed points [5], there must be a mapping h: $(R_1 \rho R_2)$ into $h(R_1) \upsilon h(R_2)$ in U, so that $h=f^n$ for some n. The loop is completed with $(a(U)_1 \mu a(U)_2)$ being (likely bijectively) mapped into $(\psi(aU_1) \tau (\psi(aU_2))$ as subsets in T. These operations are (homo)morphisms ($\mathcal{M}$or), and in particular cases where they are bijective, they constitute isomorphisms (iso$\mathcal{M}$or), in turn becoming homeomorphisms within topological structures.

4.2 Metabolic Endpoints: Fixed Points in Molecular States

Equivalence classes are constituted for each functional system of the organism by K:{X, m, J}, in which: X={x1, x2, ...} are successive substrates and products; m denotes metabolic transformations or translocations; J denotes the mapping of some components X of set W to a stable state. Since a level S must be further mapped into one and only one mental image a(U), the isomorphic mapping of a fixed point must be a fixed point. Therefore, there exists an isomorphism such that a component $x \in F_i$ or M_i of W comes to a state where there exists some q: $g(m^{q+1}(x)) = g(m^q(x))$.

The associations of such morphisms with the mapped objects represented by the set {T, W, S, R, U, a(U)} constitute as many categories.

Remark 4.2.1. This further supports the existence of a bijective mapping of transduction T into S. Since for some subset (X) of W there exists g(X)= S(X) a subset of (X), mapping g is contractive: thus it owns a Banach-type fixed point s(z)=z for some $z \subset S(X)$. Hence, for a(U) as the preimage, then T as intermediate, compositions gf and $gf\psi$ behave as retractions on T and a(U) respectively.

Remark 4.2.2. Consider g: $W \mapsto S$. For $x \in X$ in W, one can discern g'(x) such that $g'^{n}(x) \neq x$ whatever x, and $g_{|S}(x)$ such that for some n: $g^{n}_{|S}=x$. The latter stands for the actual function called (g) above, and it represents a restriction of g.

Remark 4.2.3. Unconscious endpoints correspond to "pertinent components" of conscious perception and their M.I. transduction realizes the coded anticipation already suspected in neural network research [19].

These properties appear as a funding characteristics of the homeostatic control.

4.3 Self/Nonself Recognition and Immunity

Self recognition concerns information (W) coming from inside-parts of an organism denoted as (A). Then, homomorphisms will be represented by endomorphisms (endo$\mathcal{M}$or) and isomorphisms by automorphisms (auto$\mathcal{M}$or), that is for instance:

(i) (endo$\mathcal{M}$or): $T \mapsto W$, $W \mapsto S$ and $R \mapsto U$

(ii) (auto$\mathcal{M}$or): $a(U) \mapsto T$, $S \mapsto R$ and $R \mapsto a(U)$

all constituting morphism classes on (A): $\mathcal{M}(A)$.

Remark 4.3. Let W° denote a nutriment coming from a outer source: then, W° is of foreign origin, but it is normally incorporated and transformed in some members of W. This is typical of homomorphism further composed with isomorphism cases.

Figure 5 illustrates these various patterns. Self-nonself recognition is a general system, now identified also in invertebrates [43].

Nonself recognition involves (W*) coming from a foreign organism (C): e.g. either a parasite or predator, or a foreign or xenobiotic molecule or set of molecules like food and nutrients . Then:

(i) $W^* \mapsto S'$ are (homo$\mathcal{M}$or) while $S' \mapsto U$ remain (endo$\mathcal{M}$or)

(ii) $S' \mapsto R'$ (auto$\mathcal{M}$or): and $R' \mapsto a(U')$ shifts to a (endo$\mathcal{M}$or)

which implies that $\mathcal{M}(C) \neq \mathcal{M}(A)$. Thus the following:

Theorem of the immunity. A correct immune function implies a discrimination of categories involving morphisms of objects from inside and outside the organism.

Remark 4.3.1. Non-nutriment food components, which have been called "a-nutrient" [40], behave like xenobiotics and should be eliminated.

Remark 4.3 2. It happens that parasites mimic some molecules of their host, so that the equivalence class K of the host will also include members from the parasite. Therefore, one will have: $\mathcal{M}(A)$ K $\mathcal{M}(C)$, which denotes "category confusion" and break in immune defense or immunosuppression. The same situation occurs if some cause (e.g. a xenobiotic intoxication) alters the processes which differentiate W* from W.

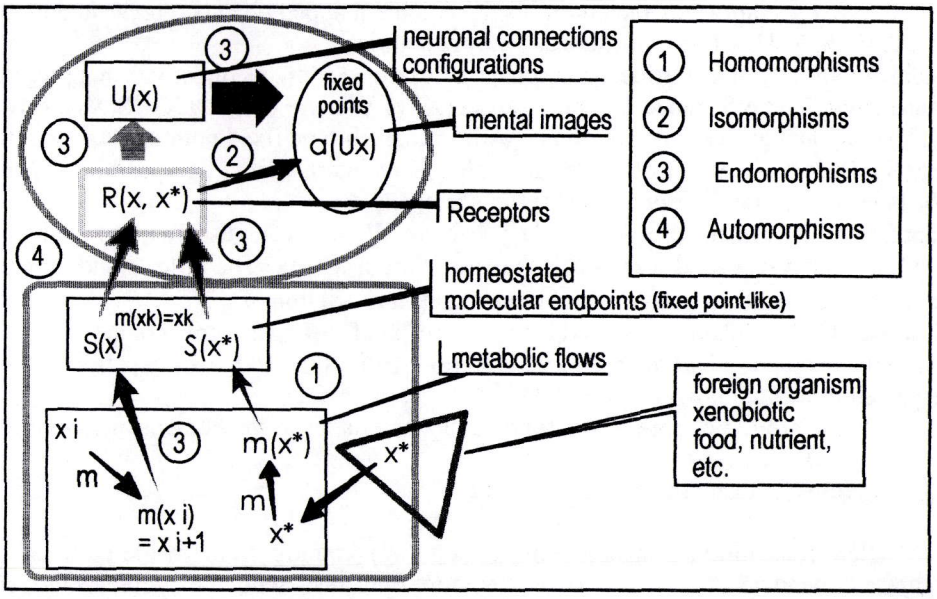

Figure 5: Schematic representation of the various morphisms holding on the self and nonself interactions in a canonical living organism.

5 Discussion and Conclusions

5.1 Recapitulative Overview

So far, it was assumed that a "working theory of what in fact is reflected by the EEG and how neurotherapy works" have not yet been settled on [35]. However, neurophysiological bases have been clearly stated for the treatment of attention deficit/hyperactivity disorders (ADD/ADHD) [34] and physiological phenomena that underlie neurobiofeedback treatment of ADHD have been thoroughly explored by Abarbanel (1995) [1]. The present work sketches the main parts of a founding theory based of a sequence of formerly established formalisms: perception function infers from existence of a physical world and mental images are described as fixed points in sequences of neuronal configurations originating from bursts of perceptive sensorial information [6]. Then, the space of mental images exhibits anticipatory properties, along with some fractal features. Anticipatory phenomena are described by incursive and hyperincursive relations in which the state of a system at time or step t: X(t) is dependent on former states and on X(t+k), $k \geq 1$ with several possible endpoints in hyperincursivity [15]. Here, an image is created in the brain cortex through confrontation of the responses recorded as fast Fourier transformed (FFT) frequencies with the goals assigned by the physicians as progressive thresholds rewarding spontaneous changes to more correct values. These changes in the activity of the cortex

anticipate on a sound health state to which the organism progressively fits the patient's physiology. Thus, the state at time t actually reflects a situation resulting from a former pathological event, and leads to a farther healthy state. Since various endpoints are possible at each time, the system fulfills all of the characteristics of a hyperincursive process and was called 'Neuro-BioFeedFarther' [9]. Reciprocally, the homeostatic reference bank should contribute to construct the self-referential system characteristic of life, and provide the stable part of what [38] emphasized as the "singular, unique history" of individuals.

The theory also explains why a small number of surviving neurones allow recovery of lost functions [26], [37]: if some thousands of neurones are involved in a sequence of connections [14], of which each include one to ten thousands synaptic connections, then about 10^5 neurones are required at each iteration of the sequence leading to the fixed point. Since the duration of the sequence must not last for more than microseconds, and that ligand-receptor activity at neuronal level may fall into the picosecond range, about 10^8 neurons may be required. Since a normal brain contains about 200 to 300 billion neurones [36], approximately a thousandth of the brain capacity is required for the creation and processing of a mental image, while neurones released during the process are available for the next mental image.

5.2 Clinical Probation

One important problem is that a sustained state of altered functions may turn the internal sensors into an adjusted state and change the mental images for a new set with altered goals. This has long been documented by the case of brain receptors of HPA activation, and the observed progression of changes in receptor density and properties leading from depressive states to suicide [4]. Further evidence is provided by several works showing that perinatal exposure to chemicals active on estrogen receptors results in alteration of receptor functions: the latter have been correctly interpreted by authors as a "false imprinting", and found to later alter responses of the progeny to natural steroid ligands [30]. This situations fits very well with the case of the formation of mental images constructed from an alteration of molecular endpoints (standing for the false imprinting), so that further brain to receptor interactions would be falsified. Among possible mechanisms, wrong signals (W*) could be translated into wrong receptor endpoints (R*) resulting in desynchronization at cortical level. Then, loops would be opened through chaotic-like behavior [28]. Conversely, an enriched bank of mental images should provide an advantage, which has just been documented with dopamine receptor involvement [31]. Since mental imaging is based on various kinds of sensory perceptions, alterations in sensory development and functioning should interact with physiological functions: this has also been recently documented for neuro-immuno-teratogenicity of some drugs: in this case, an acceleration of the sensorial development was observed [3], which suggests a compensatory process raised to increase the input of reference mental images. Similarly, the construction of fetal mental imaging should predictably be altered by exposure to neurotoxicants, and in effect, severe irreversible damage can be observed in offspring of mothers exposed during pregnancy and lactation [2]. False neurotransmitter binding (e.g. various

substances exhibiting GABA-like structures) may contribute to the neural manifestations of hepatic encephalopathy (HE) [20]. The alteration of mental imaging states is assessed by a correlation of the severity of HE with increases of Beta and Gamma EEG frequencies as subjected to interaction with external sensory stimulation. It is noteworthy that on one hand, decrease in Gamma frequencies has been interpreted as a depress of global activity of the brain with impairment of coordinated information processing over the neocortex [20] and Gamma oscillations have been reported to reflect feedback interactions between groups of neurons [17]. On the other hand, previous research reported a relation between abnormal ligand binding to GABA-type receptors and acute liver failure [39].

Such situations are thus likely to impair liver detoxification capabilities and further to increase the susceptibility of patients to toxicants. These findings emphasize the existence of a complex system of mental imaging of both sensorial and molecular origins interacting with the expression of organs diseases. The mechanisms of emotion as localized in amygdala and other related brain areas was recently reviewed by Calder et al. (2001) [12]. This work suggests self-operated rehabilitation working on a NBF-like way, and supports the importance of the bank of mental images in setting up the capability of recognition of emotion types on the face expression of others. Moreover, the concept of non-conscious processing is addressed in a way that matches with the theoretical prediction of our second kind and type II mental images.

From a clinical viewpoint, we currently are statistically assessing the relationships predicted by the theory and practically reflecting the link of the required number of treatment sessions with the former state of the patients and the rate of achievable rehabilitation. A full set of clinical results actually demonstrates the existence of highly significant correlations [9], and the problem is now focused on the identification of the algebraic nature of the observed functions.

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References

[1] Abarbanel, A., 1995. Gates, states, rhythm, and resonances: the scientific basis of neurobiofeedback training. Journal of Neurotherapy, 1(2), 15-38.

[2] Andersen, H., Nielsen, J., Grandjean, P., 2000. Toxicological evidence of developmental neurotoxicity of environmental chemicals. Toxicology, 144(1-3), 121-127.

[3] Benesova, O., Tejkalova, H., Kristofikova, Z., Panajotova, V., Husek, P., 1999. Neuro-immuno-teratogenicity of drugs used in neonatal pharmacotherapy in relation to the ontogenic stage at the time of their administration. Gen. Physiol. Biophys., 18, 21-27.

[4] Biegon, A., 2000. Regionally selective increases in β-adrenergic receptor density in the brain of suicide victims. Psychopharmacology, 100, 165-167.
[5] Bogdanov, N.N., Yakupova, L.P., Gorbachevskaia, N., Kojushko, L.Ph., Pankratova, Y.A., 1995. Pharmacodynamics of EEG after single-dose administration of seduxen in healthy volunteers. Phys. Chem. Biol. Med. 2(2), 97-102.
[6] Bounias, M., 2000. A theorem proving the irreversibility of the biological time, based on fixed points in the brain as a compact delta-complete topological space. In: Computing Anticipatory Systems: CASYS'99 - Third International Conference, ed. D.M. Dubois. American Institute of Physics, CP517, 233-243.
[7] Bounias, M., Bonaly, A., 1997. The topology of perceptive functions as a corollary of the theorem of existence in closed spaces. BioSystems, 42, 191-205.
[8] Bounias, M., Bonaly, A., 2001. A Formal Link of Anticipatory Mental Imaging with Fractal Features of Biological Time. In: Computing Anticipatory Systems: CASYS 2000 - Fourth International Conference, ed. D.M. Dubois. American Institute of Physics, CP573, 422-436.
[9] Bounias, M., Laibow, R.E., Stubblebine, A., Bonaly, A., 2002. EEG-NeuroBioFeedback treatment of patients with brain injury. Part 4. Treatment duration as a function of both the initial load of symptoms and the rate of rehabilitation. Journal of Neurotherapy, 6(1), 23-38.
[10] Bounias, M., Stubblebine, A., Laibow, R., 2001b. Anticipatory mental imaging and "NeuroBioFeedFarther" in Neurotoxicology. 19th Annu. Symp. American Environmental Health Foundation, Dallas, 7-11 June 2001, vol. 2, 17-19.
[11] Bounias, M., Laibow, R.E., Stubblebine, A., 2001c. NeuroBioFeedback, Mathematics and Neurotoxicology. 19th Annu. Symp. American Environmental Health Foundation, Dallas, 7-11 June 2001, vol. 4, 14-17.
[12] Caldern A.J., Lawrence, A.D., Young, A.W., 2001. Neurophysiology of fear and loathing. Nature Reviews Neuroscience, 2(5), 352-363.
[13] Chapman, C.A., Xu, Y., Haykin, S., Racine, R.J., 1998. Beta-frequency (15-35 Hz) electroencephalogram activities elicited by toluene and electrical stimulation in the behaving rat. Oxford), 86(4), 1307-1319.
[14] Cox, C., Denk, W., Tank, D., Svoboda, K., 2000. Action potentials reliably invade axonal arbors of rat neocortical neurons. P.N.A.S., 97(17), 9724-9728.
[15] Dubois, D., Resconi, G., 1993. Introduction to hyperincursion with applications to computer science, quantum mechanics and fractal processes. CCAI, 10(1-2), 109-148.
[16] Feshchenko, V.A., Veselis, R.A., Reinsel, R.A., 1997. Comparison of the EEG effects of midazolam, thiopental, and propofol: the role of underlying oscillatory systems. Neuropsychobiology, 35(4), 211-220.
[17] Freeman, W.J., 1995. Societies of brains. Lawrence Erlbaum Ass., Hillsdale, USA.
[18] Fulga, I.G., Stroescu, V., 1997. Effect of nifedipine on electrical activity of the brain in rata. Rom. J. Physiol., 34(1-4), 115-125.
[19] Hérault, J., Jutten, C., 1994. La mémoire des réseaux neuromimétiques. La Recherche, 25, 824-830.
[20] Kim, C., Choi, W., Park, J., Lee, H., Ha, J., Lee, M., 1996. Electro-encephalogram power spectra in thioacetamide-induced hepatic encephalopathy. Korean J. Pharmacol. 32(3), 293-300.

[21] Kinoshita, T., Saito, M., Isotani, T., Okajima, Y., Yagyu, T., Nobuhara, K., Saito, N., Ohashi, T., So, K., Kovalev, Chapman, C., Xu, Y., Haykins, S., Racine, R.J. , 1998. Beta-frequency (15-35 Hz) electroencephalogram activities elicited by toluene and electrical stimulation in the behaving rat. Neuroscience, Oxford, 86(4), 1307-1319.
[22] Kinoshita, T., Saito, M., Isotani, T., Okajima, Y., Yagyu, T., Nobuhara, K., Saito, N., Ohashi, T., So, K., Kovalev, G., Vorob'ev, V., Akhmetova, E., Shibaev, N., 2000. Phase relationships between glutamate-dependent EEG effects in α and β frequency ranges during acute and chronic piracetam administration in rats. Eksp. Klin. Farrmakol., 63(1), 3-6.
[23] Kuginuki, T., 1997. Quantitative pharmaco-EEG study of sertraline hydrochloride in healthy volunteers. Shinkei Seishin Yakuri, 19(6), 449-450.
[24] Laibow, R.E., 1997. Significance of SMR Beta frequencies in human cognition and vigilance. Alexandria Inst. Med. Report, 3 pp.
[25] Laibow, R.E., 1999. Medical applications of NeuroBioFeedback. In: Introduction to quantitative EEG and Neurofeedback. Evans and Abarbanel eds., Academic Press, New York, 83-102.
[26] Laibow, R.E., Bounias, M., Stubblebine, A.N., Sandground, H., 1996. Rehabilitation of brain injured adults and adolescents through neural therapy (voluntary regulation of EEG activity).In: Effective strategies for Assessment and Intervention, Proc. 20th Annu. Postgraduate Course on Rehabilitation of the Brain Injured Adult and Child, Office of Continuing Medical Education, Virginia Commonwealth University, Medical College, Williamsburg, June 6-9, 153-155.
[27] Lancel, M., Faulhaber, J., Schiffelholz, T., Mathias, S., Deisz, R.A., 1997. Muscimol and midazolam do not potentiate each other's effects on sleep EEG in rats. J. Neurophysiol., 77(3) 1624-1629.
[28] Long, W., Zhang, C-F., Zhao, SL., Shi, RH., 2000. On the synchronization of EEG spindle waves. Commun. Theor. Phys., 33, 665-672.
[29] Martin, S.J., 2001. Activation of metabotropic glutamate receptors induces gamma frequency oscillations in the rat dentate gyrus. Neuropharmacology, 40(4), 634-637.
[30] Metcalfe, T., Metcalfe, C., Kiparissis, Y., Niimi, A., Foran, C., Benson, W., 2000. Gonadal development and endocrine responses in japanese medaka (*Oryzias latipes*) exposed to o,p'-DDT in water or through maternal transfer. Environ. Toxicol. Chem., 19(7), 1893-1900.
[31] Neddens, J., Brandenbury, K., Teuchert-Noodt, G., Dawirs, R., 2001. Differential environment alters ontogeny of dopamine innervation of the orbital prefrontal cortex in gerbils. J. Neurosci. Res., 63(2), 209-213.
[32] Ninio, J., 1987. Alternative to the steady-state method: derivation of reaction rates from first-passage times and pathway probabilities. Proc. Natl. Acad. Sci., 84, 663-667.
[33] Obrenovitch, T., Hardy, A., Zilkha, E., 1997. Effects of L-701,324, a high affinity antagonist at the N-methyl-D-aspartate (NMDA) receptor glycine site, on the rat electroencephalogram. Naunyn-Schmiedeberg's Arch. Pharmacol., 355(6), 779-786.
[34] Ramos, F., 1998. Frequency band interaction in ADD/ADHD neurotherapy. Journal of Neurotherapy, 6, 26-41.
[35] Ramos, F., 1999. Personal correspondence with the author.
[36] Restak, R.M., 1994. Receptors. Bantam Books, New York, Toronto , 228 pp.

[37] Sabel, B., 1997. Unrecognized potential of surviving neurons: within)-systems plasticity, recovery of function, and the hypothesis of minimal residual structure. The Neuroscientist, 3(6), 366-370.
[38] Schwarz, E., 2001. Will computers ever think? On the difference of nature between machines and living organisms. International Journal of Computing Anticipatory Systems, Vol. 8, 3-17.
[39] Schafer et al., 1983. Sensitivity to ethanol-induced ataxia in HOT and COLD selected lines of mice. J. Lab. Clin. Med., 102, 870-880.
[40] Srivastava, A.N., Sharma, I.R., 1987. Toxicology of processed and packaged food. Def. Sci. J., 37, 161-172.
[41] Tan, X., Uchida, S., Matsuura, M., Nishihara, K., Iguchi, Y., Kojima, T. , 1998. Benzodiazepine effects on human sleep EEG spectra: a comparison of triazolam and flunitrazepam. Life Sci. 63(8), 675-684.
[42] Wacker, M., 1996. Alpha brainwave training and perception of time passing: preliminary findings. Biofeedback and Self-Regulation, 21(4), 303-309.
[43] Wago, H., 1994. A prototype of self-non self recognition in invrtebrates. Med. Philos., 13(4), 257-263.
[44] Yun, J., Lee, M., 1999. Electroencephalographic effects of chlorpromazine in rats. Korean J. Physiol. Pharmacol., 3(3), 245-250.