Quasi Being of Neurons and Fuzzy Logic of Brain

TSITOLOVSKY L.
Department of Life Science
University of Bar-Ilan
Ramat-Gan 52900, Israel

SANDLER U.
Department of Mathematics
Jerusalem College of Technology
Jerusalem 91160, Israel.

Abstract: - Intentional actions may be a consequence of the endogenous compensation of a drop in excitability after damage induced by a motivational excitation. A neuron does not support factors of its inner environment within the scope of the predetermined standard at the expense of the alteration of other values. The only exception is the level of damage, which neuron tries to decrease by mean of homeostatic compensation. We consider exertion in neuronal metabolism as a driving force, which induces actions directed against actual or anticipated damage. Resulting goal-directed behavior will for a side observer look like an "aspiration to life" and is rather appropriate for description of aware decision-making and fuzzy logic.

Key-Words: - Neuron, Motivation, Damage, Compensation, Learning, Fuzzy Logic

1 Introduction

The brain is a unique object. We usually examine brain behavior like that of any other object, but the brain itself also studies the environment. When a brain makes decision, it chooses between possible alternatives. Brain evaluates magnitudes of inner and external variables and possible consequences of its own reactions to alteration of these variables. Both variables and reactions exist as continuous entities, but brain operates with them as with discrete ones. This partially concerned with the threshold for neuronal excitation and with "all-or-non" principle: neuron usually sends standard spike in its axon or does not sends anything. Past experience also stores in a memory, as a discrete events.

Conscious perception is temporally 'quantized' [7]. We can barely attend to more than one object at a time, and we can hardly perform two tasks at once [13]. Identical stimuli are sometimes fully perceived and sometimes not detected at all. When two masked targets are presented successively, identification of the first target hinders the detection of the second target [17]. If there are several rewards, animal behaves as if it had simply selected the larger of two rewards [5]. Therefore, the choice of uttermost decisions is the normal action of the brain.

The excitation producing intended actions is a dominant and inhibits any other excitations. So, micro-stimulation of two cortical areas differed substantially, for instance, by a direction selectivity produces the reaction that corresponds to the stronger responding area ("winner-take-all" mechanism). However, for smaller angular separations the results were consistent with "vector averaging" [4]. We may conclude that own initial state and environmental keys are estimated in the term of rough "fuzzy" values, i.e. by "belonging" the physical or physiological parameter to some fuzzy subset. Decision is made in the rough fuzzy terms by choosing between possible alternatives and not by combination of the alternatives as weight averaging, vector averaging, etc. Meanwhile, choice for the close alternatives may use methods of averaging, since their fuzzy values are intersected, though may have different possibility. This is a trivial case, seeing as primitive automation sorting the black and white balls makes approximate decision dealing with the grey balls. We mean fuzzy decision of brain, as an aware choice. Brain makes active, non-predetermined decision.

2 Problem Formulation

Reactions of individual neurons are also extremely non-stable. Instability decreases during narcosis and other incidents of the unconsciousness [21] and during neuron damage [24]. Absence of motivation means decrease in awareness, but while awareness does not turn into unconsciousness, instability never disappears.

At the first glance, it could be assumed that instability of the neurons' reaction has a stochastic nature. In this case instability of individual neuron activities in large neuronal networks should be averaged over the ensemble. However, in spite of availability of enormous number of brain's elements (10^{11} neurons; 10^{14} synapses and more than 10^{18} ion channels) their activity is coordinated in the aware state and is not averaged by the ensemble, since in different neurons these alterations occur coordinately [19]. Faint averaging in the brain suggests that neurons receiving the same kind of information make similar decisions. The actual choice is, evidently, simultaneously both non stable and deliberate. Instability of behavior is not ordinary inexactness. Brain tries to find new possibilities to reach the goal. Our intention is to consider an origin of aware decision and find an

adequate mathematical apparatus for theoretical description of the brain's decision-making process.

3 Problem Solution

Numerous literature's sources and original experiments show that capability of perceiving could take place already on a neuron level. So, if we want to develop an adequate phenomenological theory of neuron information processing, we are, in a certain sense, compelled to search an apparatus, which could operate with "perceptions" as with mathematical objects. Another reason to develop a theory of neuron's behavior, which is based on notation of perception, is the nature of the experimental data for the real neurons. In fact, only a few parameters of a system are observable and controllable, while a number of the other ones are "hidden" or remain out of control. For a complex phenomenon it leads to considerable variability in the results from trial to trial and makes it difficult to estimate accuracy of the obtained values. For such a phenomenon fair description of a system behavior is based on our "percept" of observed tendencies rather than on precise numerical values of the experimental data.

Mathematical basis for "computing of perceptions" was proposed by L.Zadeh almost half century ago and one was named as "fuzzy logic". In the following decades fuzzy logic has been intensively developed and applied to numerous applications in several thousand articles and books. It has been shown that fuzzy logic is amazingly effective in processing of information with high level of non-stochastic uncertainty.

A real neuron is a complex dynamic system, so for description of its behavior we need an extension of fuzzy logic on evolutionary processes: "evolution of perceptions". Such a theory called "fuzzy dynamics" has been developed and studied during the last decade [8,15,16]. Fuzzy dynamics was successfully used in the situation, where both the system states and the dynamics laws have considerable uncertainty, which is typical for description of the systems on a "perceptive level". In this approach notations like: "neuron activity", "neuron damage", "level of compensation" and "expectation of punishment" are considered as phenomenological variables, which describe our "perception" of a neuron state and the theory directly operates with these variables without additional arbitrary assumptions and hardly verified hypotheses.. Of course, such variables are very qualitative and have considerable level of uncertainty, but they fair reflect our real knowledge about the considered system. In [8,15,16] one has been shown how fuzzy Hamiltonian and fuzzy Liouville-inclusion for the neuron system can be obtained.

Does, however, the brain itself use fuzzy logic for description of an environment and for decision-making? Aware decision-making is a central point of goal-directed (motivational) behavior. Thus, analysis the nature of motivation is necessary for comprehension the emergence of fuzzy logic in the brain.

3.1 Motivations injure neurons, while reward protects them

Motivation is usually understood as an organism's subjective attitude to its current physiological state, which somehow modulates generation of actions until the organism attains an optimal state. Motivation is satisfied by reward or by avoiding punishment. Motivation is undoubtedly associated with the need for stability of the internal milieu or homeostasis [3]. Homeostasis, by mean of restorative, compensational mechanisms, maintains stability of physiological systems and holds the parameters of an organism's internal milieu (or correspondence between these parameters) within limits that allow for survival. The compensation plays a role in the endogenous protection against metabolic disruption, like a reward that exerts an exogenous protection. If the adaptive response cannot be generated internally by means of compensational homeostatic processes, it must be achieved via interaction with the environment. In this case, an adaptive behavior can assist to avoid the death.

An organism is built of unreliable elements and self-regulation is directed to protecting them from damage. A motivation arises as the result of a shift of inner constants from their optimal values and is related to transient injury or to the threat of injury. Neuronal damage is a fee paid for excitation. During homeostatic disturbance, in specific neurons degrade morphology, ion metabolism, electrical activity, and other factors that prevent normal functioning and may eventually lead to cell death. Treatments that induce damage intensify motivations, and treatments that protect neurons usually satisfy them. In contrast, treatments that usually increase motivation induce instability of neuronal reactions and damage, while that which decrease motivation protect neurons [19].

At the same time as motivation and punishment are connected with excitation and neuronal injury, the reward inhibits and protects neurons. The substances that satisfy motivation as the result of reward, such as opioids, cocaine, caffeine, alcohol and cholecystokinin protect neurons from damage. However, because of the development of compensatory processes, chronic or high doses of cocaine, alcohol, opiates, etc. increase excitability and exacerbate damage [19]. Although the protective role of reward means that neuronal damage during motivation is transient, neurons are sometimes irreversibly injured, such as during drug-dependence, self-stimulation, stress and redundant mating.

3.2 Nature of homeostatic compensation

The goal of brain is survival of the organism and the "goal" for neuron is its own survival. Just as motivation and the life of an organism depend upon homeostasis, so does neuronal behavior and life depend upon neuronal homeostasis. Motivational behavior is present at the neuronal level and, furthermore, the homeostasis of a neuron is a unit of motivational behavior [19,20]. An organism by means of homeostatic compensation supports values of inner characteristics that do not damage its neurons.

Homeostasis is a key idea in biology. Nevertheless, it is unclear, how are vital constants regulated. It was supposed that cells have special proteins, sensors, each one responsible for regulation of the given mother-value. Such particular homeostatic regulation of specific characteristic may support basic cell functions, which does not connect with awareness. Neurons use activity sensors to regulate the number and kind of ion channels and receptors in their membrane to maintain reliable performance. For example, intracellular Ca^{2+} sensors detect change in Ca^{2+} level and trigger changes in the number or distribution of calcium channels, intracellular iron concentration affects the rate of synthesis of iron regulatory proteins and the measure of energy-reach molecule concentrations in hypothalamic neurons during feeding behavior triggers the appropriate appetitive responses as similar to other hypothalamic homeostatic centers for temperature, oxygen consumption and osmolarity [12].

Even so, the separate regulation of individual characteristics does not explain a general control of cell activity. Homeostasis somehow regulates a certain general quality of neural cell. Similar firing properties can be produced by widely different combinations of ion currents and the effect of varying the number of channels of one current can differ considerably depending on the numbers of each of the other kinds of channels in the neuron. It was supposed that the final level of activity of a neuron, or excitability, is tightly controlled, rather than the number of each kind of ion channel individually [12].

There are a few general attributes that characterize state of a neuron, as a whole: membrane potential, input resistance, excitability, energetic resource, inner pH, metabolism intensity and level of damage. There are also some less specific attributes that affect the general state of a cell, such as levels of second messengers, chemosensitivity, intracellular contents of calcium ions $[Ca^{2+}]_i$, correspondence between $[Na^+]_i$ and $[K^+]_i$, etc. Majority of these characteristics are attributing of any cell, even plant, whereas excitability and high level of cell metabolism are typical just for neural cells. The brain, which mass maintains less than 2% of body, is responsible for 20% of body oxygen consumption. The basic diversity of chemical reactions in body also belongs to brain. Excitability is intimately connected with cell damage, while metabolism level is coupled with damage compensation. An animal can support any factor at the value that differs on standard, usual value by means of the alteration of other values, but never to treat a threat to life as a reward [6]. Such important factor, as neuron entirety, level of damage, or, in the first approximation, excitability, may serve as the mother-value of all values.

Such as we have described various trails affecting excitability, there are multiple pathways leading to neuronal death and numerous mechanisms protecting a cell from death and not one of them is obligatory. A neuron may be injured if the homeostatic regulation of any ion current participating in neuronal function is disturbed, but each of the parameters may decline from the homeostatic equilibrium without symptoms of damage due to compensatory changes of other parameters. For example, neuronal damage can proceed without acidosis, in the absence of hypoxic depolarization, without increase in free radical levels, may be independent of elevation of $[Ca^{2+}]_i$ and excitotoxicity [19]. Hence, damage is not strictly determined by a membrane potential, $[Ca^{2+}]_i$, free radicals, pH, etc. Neuron must somehow sense levels of damage-protection itself. Some more, a neuron needs in a criterion for choice of the preferable action.

3.3 Common currency for feeling: being between displeasure and pleasure.

Although a neuron needs to support many important factors, it can detect only one object at a time, has only one output, can make only one decision and only one integral factor has to determine neuron's fate, such as excitability, which is intimately connected with the cellular damage-protection. A neuron regulates its major demands, the avoidance of injury and the aspiration to life, as its shift towards death or towards life. We suggest that excitation and inhibition are perceived by a neuron as negative and positive states. It would be plausible to consider cellular excitation and damage as a primary punishment of a cell, whereas inhibition of neuronal activities and protection from damage is apparently a positive signal. Treatments that protect neurons usually inhibit their activity and exert psychotropic actions related to sensory pleasure states and relief. For instance, opiates and other rewarding substances have anxiolitic and antiepileptic effects [2], whereas their antagonists are convulsants and evoke withdrawal. Investigation of the postmortem brains of suicide victims and those suffering from a mood disorder allows an evaluation of physical signs of negative emotional states. The brains of suicide victims reveal neuronal atrophy, a disturbance of intracellular biochemical machinery and a pronounced increase in

excitatory neurotransmitters, while inhibitory system is repressed [11]. Though the brain responds differentially to positive and negative rewards, but the same neurons may be responsible for pleasure and suffering, dependently on their shift to or out of damage. Two basic emotions – pleasure and sadness - resulted in similar areas of activation [10].

Awareness is intimately connected with availability of excitation, metabolic disturbance and transient damage in the brain. Substances which disturb awareness, barbiturates, benzodiazepines, general anaesthetics lead to hyperpolarization, potentiation of inhibitory signals and decrease in impact of excitatory aminoacids [9,22]. Volatile anesthetics reduce neuronal injury caused by overexcitation [23]. Blockage of awareness may be determined as a oppression in neuron's ability to perceive damage. Mortality is not an annoying inevitability. A capability to feel pleasure rests on the fact of mortality and awareness consists of being between life and death and, in the narrow sense, between rest and depression.

3.4 A physiological description of voluntary actions.

Alterations in neuronal responses during learning does not follow to a well known neuronal property. Neuronal responses decrease during habituation in the trials when the membrane potential decreases. Correspondingly, after the acquisition of classical and instrumental conditioning, conditioned response augmentation coincides with membrane potential increases. Hence, inhibition can inconsistently activate neurons. Depolarization of the membrane potential did not correspond to an augmentation in the value of the neuronal responses to conditioned and unconditioned stimuli. Therefore, depolarization is not the immediate cause of the spike generation while hyperpolarization is not the immediate cause of the spike failure. There is constant tonic inhibitory control of the excitatory spike responses of pyramidal neurons to conditioned stimuli in conscious animals. So, the logical constraint between changes in the membrane potential and the output reaction is broken [18,19].

Both damage and motivational excitation evoke depolarization, the growth of excitability in neurons and distort membrane properties. In dying neurons, a phase of high excitability is inevitably transformed to an unexcitable state. An injurious agent usually evokes short-term depolarization and excitation, which, after a few minutes, is replaced by a slow compensatory hyperpolarization. This is followed by a persistent depolarization until the membrane potential reaches zero and excitability decreases. In this case, hyperpolarization protects neurons and paradoxically recovers excitability [20].

It was established around century ago that after injury to the neural system a paradoxical stage ensues [21]. When responses are reduced, they become larger as the irritant decreases. This phenomenon is agreed with the development of compensational processes in neurons, which paradoxically recover excitability through development of inhibition.

We, therefore, suggest that a homeostatic plasticity controls behavior plasticity and that processes of compensation participate in the organization of behavior. Generation of instrumental reactions may be a consequence of the recovery of neuronal excitability after excitotoxic damage induced by a motivational excitation. Absence of a steady cause-and-effect connection between an input signal and an output reaction favors the emergence of such reactions rather than their being elicited.

The disturbance of an equilibrium in the damage-protection mechanism leads to a changing of the compensation mechanism into delayed damage, or to delayed protection. Overprotection leads to a compensatory increase in excitability and the state of the cell, hereafter, shifts toward damage. Therefore, a neuron tends to vary its behavior. We may suppose that voluntary actions arise as the result of a counteraction of compensational mechanisms with excitotoxic damage of specific neurons, which thus anticipate compensation evoked by reward. Such actions are targeted to compensate metabolic alterations and are thus goal-directed. Taking into consideration that emergence of the action is accompanied by a rise in instability in the corresponding neurons, this action may be both intended and non-predetermined. This means that essence of motivation cannot be reduced to modulation of a readiness to the action, so that activation of this action became facilitated. A motivation probably activates metabolic recourses promoting action emergence.

3.5 Compensational processes during homeostasis participate in neuronal plasticity

Compensational processes, directed against both damage and overprotection, can modulate neuronal excitability and may participate in the plasticity of motivational behavior. As a result of experience, homeostasis acquires the capability to counteract the reason of the damage instead of damage itself. Capability to compensate damage becomes anticipatory after experience. Preconditioning with brief, non-lethal injury leads to compensation and stimulates neurons to become more resistant to a subsequent damage. An acute action of a factor usually has opposite results to its chronic action [1,17]. A classic explanation for addiction is that use of a drug causes compensatory physiological adaptations. Acute administration of opiates lowers neuronal excitation and reduces cell

damage, whereas cellular homeostasis compensates for this overprotection, aggravates neuronal damage and compels one to take new doses of opiates [19].

Homeostatic plasticity is observed at the cellular level. Shifts in ionic homeostasis can concomitantly change the informational capability of the neuron and increase a neuron's vulnerability to excitotoxicity. The mechanism of compensation may be developed on the basis of the K^+, Na^+-pump, alteration of inhibitory processes, calcium-activated K^+ channels, which provide a connection between cellular metabolism and membrane potential or be based on the homeostatic balance between producing free radicals and free radical scavengers. Activation of these mechanisms inhibits neuronal spiking and develops the cellular protection. Regulation of ion homeostasis itself has been shown to undergo plastic changes such as habituation, sensitization and conditioning [19]. Hence, homeostasis may be directed toward anticipatory compensation of the factors that lead to a disturbance of the homeostasis.

4 Conclusion

A brain does not support factors of its inner environment within the scope of predetermined standard, at the expense of the alteration of other values. The exception is the level of damage, which neuron aspires to decrease and that may serve as the mother-value of all values. Yet, what physical sense has "aspiration to decrease the damage"? What is the agency that compel animal to generate action in other to avoid danger? We have demonstrated that intentional actions may be a consequence of the homeostatic compensation of suppression after damage induced by a motivational excitation. Existence of a neuron between the death and the life results in by turns excess the motive to generate or fail to generate spikes. Properties of homeostatic compensation make it tempting to consider it as a driving force, which induces actions directed against actual or anticipated damage.

The fuzzy dynamics approach leads to a good compatibility with experimental observations, allows understanding some basic features of the neuron being (Fig.1) and properly match animal behavior. In particular, it predicts strong sudden, non-stochastic alterations in the neuron's activity. Such alterations are typical for a real neuron's behavior and it has an effect on macro-behavior of an animal. It is well known that even when an animal is familiar with a good solution to a given problem, it tries from time-to-time to find a new solution and if the new solution is a worse then the animal returns to its previous behavior. Such exploratory activity is very beneficial, since it enables the animal to effectively optimize its behavior in continuously changing environmental conditions.

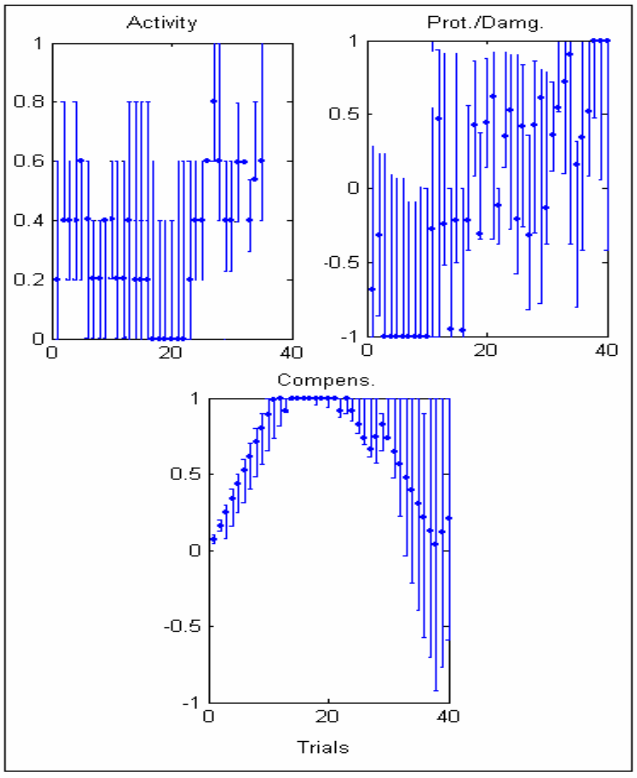

Fig.1 Fuzzy dynamics prediction of a single neuron behavior under instrumental reflex elaboration.

It should be emphasized; that in fuzzy dynamics approach such behavior is neither the consequence of random inner influences of the neural system nor only the result of the sudden changes in environment, but rather a fundamental feature of neurons.

Exertion in neuronal metabolism, leading to goal-directed behavior, will for a side observer look like an "aspiration to life" and is rather appropriate for description of aware decision-making and fuzzy logic.

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