Cognitive Impairment in Oncology Practice in the Context of Entropy Neuron-glial Network of the Brain

Rosman SV*1 and Filonov SM2

1Physician of functional diagnostics of SBIH, Regional psychoneurological clinic, Russia
2Head of the branch № 7, expert oncologist FSI, “Main Bureau of medical-social examination across the Tver region” of the Ministry of labour and social development

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*Corresponding author: Rosman SV, Physician of functional diagnostics of SBIH, Regional psychoneurological clinic, Tver, Russian Federation, Russia, Tel: +7-903-800-11-05; Email: seros2005@mail.ru

Abstract

The article presents new approaches in the diagnosis of cognitive impairment, particularly in Oncology, on the basis of a new research method, the variance of the amplitude-frequency characteristics of the alpha rhythm, allowing not only to diagnose these disorders at early stages, but also new insights on the pathogenesis of cancer.

Keywords: Cognitive disorders; Oncology; Dispersion of alpha-rhythm; Diagnosis of mental illness

Introduction

One of the objectives of the series of articles devoted to the new method of diagnostics of mental illness, the variance of the amplitude-frequency of diseases the alpha rhythm of the EEG (DAFCAR), is a presentation of the problems of psychopathology in society in a somewhat unusual perspective psycho-organic problems arising in the course of a violation of reception afferent information, its encoding and generate the efferent response in the brain. The coverage of these issues allows us to understand that many problems are not only mental, but also somatic diseases are interrelated and DAFCAR is an effective method for the diagnosis of the disorders underlying them [1].

Is no exception and is a real issue that encompasses two seemingly little linked process - cognitive disorders and oncological problems. Naturally, both of these processes will be considered from the standpoint of violations of the process control from the Central nervous system. The problem of cognitive impairment is far beyond the interests of only psychiatrists. Taking into account the prevalence of this disease (about 1% of the population in all countries of the world) could count on a greater interest of physicians to this disease [2]. However, oddly enough, generally prevails a relatively conciliatory attitude to this problem, although each publication has consistently indicated its social importance. This occurs because so far not found a sufficiently effective and universal method of diagnosis of these conditions.

Methods of assessing the nature of cognitive symptoms are one of the fastest growing forms of psychopathological, pathopsychological and instrumental diagnosis of. Not lost its relevance screening procedure, such as the scale MMSE (Mini Mental State Examination); Clinical Dementia Rating Scale,
test clock drawing in Alzheimer’s disease, GPCOG (General Practitioner Assessment of Cognition), Frontal Assessment Battery, FAB; The Montreal scale assessment of cognitive impairment; test "5 Swords"; California Verbal Learning test, CVLT; Rey Auditory Verbal Learning Test; Behavioural Assessment of the Dysexecutive Syndrome - BADS; Trail Making Test-B; Stroop Color-Word Interference Test; Wisconsin Card Sorting Test - WCST; Digit Symbol Test; Corsi block tapping test; Controlled Oral Word Association Test; the Boston Diagnostic Aphasia Examination and finally, IQ-tests WAIS and Eysenck [1-8].

All this impressive set of methods are not able to solve the main task - to reveal for the disease and identify ways of prevention and treatment - they only state the fact of the presence of cognitive impairment. Cognitive impairment is mental disorders, which include various diseases in which reduced cognitive functionality. Cognitive functions are called the most complex functions of the brain, by means of which the subjective perception of the world in order to adequately interact with it. Very conditionally it is possible to divide cognitive impairment in congenital mental retardation (F7x according to ICD-10) and acquired dementia (mostly F00-003 ICD-10). The initial stages of dementia are mild cognitive disorder (F06.7x according to ICD-10). Violation of cognition entails a lot of social problems, which in General can be described as the difficulty an adequate response of the patient to the complex changes of the environment.

Oncology - field of medicine, quite far removed from psychiatry, at least, so it seems. Actually it is based on an incredibly complicated disorder of internal organs and tissues from the Central nervous system. It is impossible not to take into account the fact that detection of cancer is traumatic for the patient strongest factor, often all of his future behavior and even voluntary exit from life. Not coincidentally, therefore, the study of the mental state of cancer patients has been given is significant, though still insufficient, attention [3].

All of these seemingly diverse medical problems have one common theme - cognitive impairment when they are fertile ground for the development of addictive disorders. Alcoholism and drug addiction, compulsive gambling is becoming a frequent companion of such patients. Psycho organic syndrome, which develops thus, brings enormous suffering to the sick, ending by deviant behavior; crime and suicides.

Not by chance, therefore, that the search for true causes changes in the brain, causing cognitive psychosomatic disorders, is the most urgent health problem. As in previous articles in this series, we will examine these disorders from the standpoint of entropy NGNB [4-7]. The purpose of the study: detection of changes in NGNB characteristic of cognitive impairment in General, and diseases of an oncological profile.

Materials and methods

A comparative study of patients with cognitive impairment with the presence of cancer pathology and without it with this purpose were examined with standard techniques of EEG and HVT according to the scheme described in previous publications of series of articles [5]. As a study of the contingent studied EEG following patients (Table 1). Patients with mild cognitive disorders acquired this disease due to various reasons - the main role was played by vascular, toxic and traumatic factors. Cancer was not associated with brain tumors, which in classification is in the category of F06.73. 55% of these patients had the indication of the presence of oncological diseases of the gastrointestinal tract, 29% respiratory, 14% of liver cancer, 2% of tumors of different localization; cognitive impairment they were in the category of F06.77-79 for ICD-10.

| Diagnosis | women | Age | men | Age | Общий итог |
|-----------|-------|-----|-----|-----|------------|
| MCD (in connection with other, non-oncological diseases) | 26 | 30.7±1.1 | 44 | 23±2.2 | 70 |
| MCD ONCO (associated with cancer - F06.77-F06.79) | 15 | 38.5±2.1 | 32 | 41.9±3.6 | 47 |
| MMR (F70.x) | 30 | 31.3±2.2 | 47 | 23.6±1.7 | 77 |
| Control (absence of mental illness) | 14 | 34.5±1.5 | 60 | 21.5±0.9 | 74 |
| Общий итог | 71 | 123 | | | 194 |

EEG data were processed according to the method DAFCAR [5].
Results

Comparative dispersion diagrams presented in Figures 1-3. Data for the comparative analysis of indexes of DAFCAR in different studied groups are presented in tables 2-3. The results of the hyperventilation a sample in the studied category of patients is presented in Figure 4.

Figure 1: Dispersion chart for mild mental retardation (F70) top - left hemisphere, bottom – right here and below, the horizontal axis is the frequency of the alpha rhythm in Hz, vertical axis - EEG: even - right hemisphere; odd - left hemisphere.

Figure 2: Dispersion chart for MCD (F06.7).

Figure 3: Dispersion chart for MCD ONCO (F06.77-79).

Figure 4: The distribution of the results of GW according to the type of reaction between different categories of patients with cognitive disorders.

Discussion of the Results

The main differences between cognitive impairment from the norm on the cartogram are slowing of the alpha rhythm, which is manifested by a decrease in its modal values. This fact in psychiatry and especially in the examination is given undeservedly little attention. The value of the fact of communication frequency of the alpha rhythm and cognitive brain activity was observed in the 50-ies of the XX century N. Wiener. The essence of this phenomenon is the need for sweep time of afferent information for which it is sampled with the frequency of the alpha rhythm of 10 Hz. Result and significance of this process is clearly demonstrated by the following example (Figure 5).

The more the refresh rate, the more information we can get from the image. Some of the details at low frequency we can't tell the difference. The same is happening in the brain the higher the frequency of the alpha rhythm, the result of the cognitive abilities of the brain, and vice versa. In ontogenesis of the brain established fact is the increase in the frequency of the alpha rhythm in the process of maturation of approximately 1 Hz/year, in puberty is the process stabilized gradually, reaching up to 10-11 Hz, and then, individually, each has been steadily decreasing. But especially the obvious importance of the frequency of the
alpha rhythm for assessing the functional abilities of the brain becomes in the study of psychopathology.

In Figure 1 presents the dispersion chart with mild mental retardation (F70). A characteristic feature of it is the increase in the representation of the non-REM part of the alpha rhythm; DAFCAR indicators point to a slowing of the alpha rhythm to 9.5 Hz, the appearance of difference of modal values in the frontal and occipital leads up to 0.5 Hz, the increase in the variance of the distribution of CDα1 from the normal compared with the personality disorder that is manifested by decrease of indexes of dispersion (Tables 2 & 3). Thus, we are witnessing a process of progressive disorganization NGNB compared to the norm and a personality disorder with a slowing of the alpha rhythm and appearance of the phenomenon of functional hypofrontality more significant increase DAFCAR compared to other parts of the brain. This phenomenon is clinically and theoretically many times described, there is evidence of his possible diagnosis with P, but in practice, in addition to the registration of the polymorphic slow wave oscillations in the frontal lobes, this phenomenon is verified and visualized was not.

Table 2: The summary table of parameters DAFCAR with MCD in comparison with the norm and MMR, the left hemisphere.

| Indexes         | MMR | MCD | MCD ONCO | Control | MMR | MCD | MCD ONCO | Control |
|-----------------|-----|-----|----------|---------|-----|-----|----------|---------|
| Alpha-1/Alpha   | 0.322 | 0.292 | 0.344 | 0.121 | 38 | 50 | 40 | 40 |
| Alpha-2/Alpha   | 0.277-0.367 | 0.233-0.352 | 0.268-0.421 | 0.093-0.149 |
| Alpha-3/Alpha   | 0.523 | 0.564 | 0.512 | 0.813 | 29 | 33 | 27 | 7 |
| CDα1            | 0.467-0.578 | 0.49-0.639 | 0.436-0.588 | 0.779-0.846 |
| CDα2            | 0.18 | 0.173 | 0.17 | 0.096 | 68 | 55 | 86 | 64 |
| IIDA            | 0.185-0.226 | 0.135-0.212 | 0.089-0.251 | 0.06-0.131 |
| ADA             | 0.523 | 0.564 | 0.512 | 0.813 | 29 | 33 | 27 | 7 |
| O Mo f          | 0.467-0.578 | 0.49-0.639 | 0.436-0.588 | 0.779-0.846 |
| F Mo f          | 1.51-4.042 | 2.761-5.455 | 1.504-5.454 | 5.558-7.789 |
| O Mo f - F Mo f | 1.51-4.042 | 2.761-5.455 | 1.504-5.454 | 5.558-7.789 |
| IIH             | 1.51-4.042 | 2.761-5.455 | 1.504-5.454 | 5.558-7.789 |
| AH              | 0.185-0.226 | 0.135-0.212 | 0.089-0.251 | 0.06-0.131 |
| Age             | 0.185-0.226 | 0.135-0.212 | 0.089-0.251 | 0.06-0.131 |
### Table 1: Neurological Markers in Men

| Marker          | Value 1 | Value 2 | Value 3 | Value 4 | Value 5 | Value 6 | Value 7 | Value 8 | Value 9 |
|-----------------|---------|---------|---------|---------|---------|---------|---------|---------|---------|
| Alpha-1/Alpha   | 0.216   | 0.236   | 0.335   | 0.129   | 57      | 43      | 50      | 35      |
| Alpha-2/Alpha   | 0.625   | 0.626   | 0.549   | 0.773   | 28      | 26      | 37      | 18      |
| Alpha-3/Alpha   | 0.196   | 0.169   | 0.139   | 0.134   | 87      | 83      | 59      | 128     |
| CDa1            | 0.195   | 0.186   | 0.185   | 0.284   | 42      | 43      | 56      | 14      |
| CDa2            | 0.583   | 0.551   | 0.986   | 0.745   | 29      | 29      | 264     | 9       |
| IIIDA           | 3.496   | 3.562   | 3.665   | 6.895   | 104     | 97      | 119     | 30      |
| ADA             | 1.866   | 1.858   | 1.798   | 2.657   | 42      | 44      | 53      | 12      |
| O Mof           | 9.57    | 9.61    | 9.06    | 10.25   | 5       | 2       | 9       | 6       |
| F Mof           | 9.8     | 9.3     | 8.98    | 10.26   | 12      | 15      | 14      | 5       |
| CDa1F           | 0.147   | 0.163   | 0.142   | 0.23    | 45      | 46      | 54      | 23      |
| CDa2F           | 0.454   | 0.447   | 0.42    | 0.623   | 32      | 40      | 36      | 16      |
| O Mof - F Mof   | 0.02    | 0.045   | 0.05    | 0.04    | 129     | 114     | 96      | 228     |
| IIH             | 2.672   | 3.622   | 1.873   | 6.534   | 110     | 105     | 161     | 36      |
| AH              | 1.542   | 1.715   | 1.105   | 2.542   | 52      | 57      | 60      | 18      |
| Age             | 23.617  | 23.045  | 41.906  | 21.508  | 27      | 33      | 24      | 27      |
|                 | 21.7-25.5 | 20.7-25.4 | 38.3-45.5 | 20.0-23.0 |
### Table 3: The summary table of parameters DAFCAR with MCD in comparison with the norm and MMR, the right hemisphere.

| Indexes DAFCAR | Mean | CV% |
|---------------|------|-----|
|               | MMR  | MCD | MCD ONCO | Control | MMR  | MCD | MCD ONCO |
| Alpha-1/Alpha | 0.3  | 0.271 | 0.351 | 0.113 | 45   | 58  | 45    | 55  |
|               | 0.249-0.351 | 0.207-0.334 | 0.263-0.44 | 0.077-0.149 |
| Alpha-2/Alpha | 0.548 | 0.593 | 0.513 | 0.822 | 31   | 33  | 31    | 9   |
|               | 0.484-0.611 | 0.515-0.671 | 0.426-0.601 | 0.778-0.866 |
| Alpha-3/Alpha | 0.152 | 0.136 | 0.135 | 0.065 | 75   | 70  | 98    | 58  |
|               | 0.11-0.195 | 0.098-0.174 | 0.062-0.209 | 0.043-0.087 |
| CDα1          | 0.17  | 0.19  | 0.146 | 0.275 | 56   | 45  | 35    | 14  |
|               | 0.134-0.205 | 0.156-0.225 | 0.117-0.174 | 0.253-0.296 |
| CDα2          | 0.455 | 0.535 | 0.466 | 0.776 | 41   | 36  | 35    | 8   |
|               | 0.386-0.524 | 0.457-0.613 | 0.376-0.556 | 0.742-0.811 |
| IIξA          | 3.252 | 4.032 | 1.976 | 6.031 | 109  | 83  | 112   | 26  |
|               | 1.929-4.575 | 2.686-5.379 | 0.748-3.203 | 5.126-6.935 |
| ADA           | 1.608 | 1.946 | 1.451 | 2.558 | 58   | 41  | 48    | 11  |
|               | 1.262-1.954 | 1.624-2.267 | 1.062-1.839 | 2.393-2.722 |
| O Mo f        | 9.17  | 9.17  | 8.97  | 10.16 | 6    | 8   | 7     | 5   |
|               | 8.98-9.36 | 8.88-9.46 | 8.63-9.31 | 9.89-10.44 |
| F Mo f        | 9.33  | 9.37  | 8.93  | 10.09 | 15   | 13  | 14    | 5   |
|               | 8.8-9.87 | 8.88-9.85 | 8.22-9.65 | 9.79-10.39 |
| O Mo f - F Mo f | 0.46 | 0.44 | 0.8 | 0.07 | 98   | 100 | 83    | 254 |
|               | 0.29-0.63 | 0.26-0.62 | 0.43-1.17 | -0.03-0.18 |
| CDα1F         | 0.137 | 0.131 | 0.133 | 0.208 | 51   | 49  | 41    | 24  |
|               | 0.111-0.163 | 0.105-0.157 | 0.103-0.164 | 0.178-0.237 |
| CDα2F         | 0.396 | 0.392 | 0.392 | 0.61  | 31   | 38  | 34    | 18  |
|               | 0.35-0.442 | 0.332-0.451 | 0.317-0.466 | 0.546-0.675 |
| IIH           | 2.425 | 2.069 | 1.358 | 4.993 | 154  | 157 | 133   | 43  |
|               | 1.027-3.823 | 0.754-3.383 | 0.357-2.359 | 3.758-6.229 |
| AH            | 1.424 | 1.35  | 1.043 | 2.281 | 63   | 62  | 43    | 20  |
|               | 1.089-1.76 | 1.014-1.685 | 0.794-1.292 | 2.016-2.547 |
| Age           | 31.3  | 30.7  | 31.5  | 34.9  | 33   | 36  | 42    | 35  |
|               | 27.4-35.2 | 26.2-35.1 | 24.2-38.9 | 27.8-41.9 |
Analysis of variance of the alpha rhythm allows such diagnosis to be performed, and methods available, inexpensive, non-invasive and easily implementable. On all subsequent maps (Figures 2 & 3) planned the same trend - total slowing of the alpha rhythm, which is a cardinal symptom for all cognitive disorders. However, there are differences that relate to the ONCO group. On average in this group, compared to all the others, is more pronounced the difference between the frequency of the alpha rhythm in the frontal and occipital leads (more than 0.75 Hz) - the phenomenon of functional hypofrontality. The reasons for this may be several to talk about them only hypothetically. However, given the fact that this phenomenon has similarities with the changes DAFCAR with paranoid schizophrenia, it can be assumed that these changes are associated with a fast-acting psychotraumatic factor of the paranoid focus of the frontal lobes of the brain.
Psychologically this is evident in the increased concern for their health, fixing on their feelings about cancer. The danger of such condition is the high level of suicide in such patients; therefore, timely diagnosis of such conditions can contribute to the preservation of life in these patients. The analysis of the statistics shows that all cognitive disorders have significant differences from the normal parameters DAFCAR. Mild mental retardation and its characteristics are similar to mild cognitive disorders, which is not surprising. In principle, they are based on the same mechanism, show-famous for slowing of the alpha rhythm, only with mental retardation; this mechanism is valid since childhood.

Significant differences in the parameters DAFCAR observed in the group of CANCER. As was clearly evident on the maps, the main difference of this group is the significant increase in slow wave representation of the alpha rhythm in the frontal divisions. The main symptom of this is the increase in the difference between the frontal and occipital departments to 0.75 Hz or more. However, there are statistically significant differences in other indexes DAFCAR (Table 4) - ADA, IHH, AH. This indicates the presence of severe functional hypofrontality in the ONCO group.

Table 4: Comparative indicators of differences in the average values of certain parameters of the MCD compared with MMR and normal by Student’s test (t-value).

| Mean          | MMR  | MCD ONCO | t-value | df  | p     |
|---------------|------|----------|---------|-----|-------|
| Alpha-1/ Alpha| 0.251| 0.343    | -4.873  | 246 | 0.000 |
| Alpha-2/ Alpha| 0.596| 0.533    | 2.643   | 246 | 0.009 |
| AH            | 1.494| 1.101    | 3.903   | 246 | 0.000 |
| O Mo f        | 9.422| 9.074    | 4.427   | 246 | 0.000 |
| F Mo f        | 9.529| 9.019    | 3.109   | 246 | 0.002 |
| O Mo f - F Mo f| 0.393| 0.739    | -5.136  | 246 | 0.000 |
| MCD           |      | MCD ONCO |         |     |       |
| Alpha-1/ Alpha| 0.248| 0.343    | -5.084  | 232 | 0.000 |
| Alpha-2/ Alpha| 0.618| 0.533    | 3.53    | 232 | 0.001 |
| CDα1          | 0.197| 0.173    | 2.062   | 232 | 0.040 |
| ADA           | 1.969| 1.724    | 2.2     | 232 | 0.029 |
| IHH           | 2.882| 1.802    | 2.465   | 232 | 0.014 |
| AH            | 1.535| 1.101    | 4.016   | 232 | 0.000 |
| O Mo f        | 9.338| 9.074    | 3.15    | 232 | 0.002 |
| O Mo f - F Mo f| 0.402| 0.739    | -4.884  | 232 | 0.000 |
| Control       |      | MCD ONCO |         |     |       |
| Alpha-1/ Alpha| 0.12 | 0.343    | -15.961 | 241 | 0.000 |
| Alpha-2/ Alpha| 0.789| 0.533    | 12.54   | 241 | 0.000 |
| CDα1          | 0.208| 0.173    | 13.265  | 241 | 0.000 |
| IIDA          | 6.89 | 3.321    | 9.473   | 241 | 0.000 |
| ADA           | 2.667| 1.724    | 12.116  | 241 | 0.000 |
| CDα1 F        | 0.226| 0.14     | 10.863  | 241 | 0.000 |
| CDα2 F        | 0.622| 0.433    | 9.55    | 241 | 0.000 |
| IHH           | 6.143| 1.802    | 13.141  | 241 | 0.000 |
| AH            | 2.476| 1.101    | 19.907  | 241 | 0.000 |
| O Mo f        | 10.23| 9.074    | 14.08   | 241 | 0.000 |
| F Mo f        | 10.228| 9.019    | 10.585  | 241 | 0.000 |
| O Mo f - F Mo f| 0.049| 0.739    | -13.258 | 241 | 0.000 |
| MMR           | VCD  |          |         |     |       |
| ADA           | 1.760| 1.969    | -2.143  | 292 | 0.033 |

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Data HVT is also very interesting (Figure 4) obviously, it is pathologically changed NGNB functional, so a significant proportion of pathologists-clinical forms of reaction - endogenous, exogenous and mixed. With mental retardation, the greater the proportion of endogenous response may be due to certain innate properties NGNB, violating the childhood of the cytoarchitectonics of the brain. In light cognitive disorders of the higher proportion of exogenous reactions - the effect of exogenous factors - vascular, toxic, traumatic, psychogenic.

The significance of all these facts is very large. First, the method DAFCAR gives the researcher information about the presence or absence of the patient for objective signs of dementia and allows you to track these changes in the dynamics. Secondly, all these changes are objective, i.e. independent of the will of the patient and the researcher, it is impossible to falsify, and their results are documented. This allows you to survey people on the presence of cognitive impairment, irrespective of their national, cultural, linguistic and other differences, what is the main drawback of all the many psychological tests given in the beginning of the article.

Very significant is the fact that early detection of cognitive disorders, contributes to the prevention of alcohol, drug and gambling addiction, because timely medical assistance to such patients prevents the formation of these harmful habits. Very interesting observation in the group of ONCO not only because we showed signs of disorganization NGNB-related mental trauma as a result of the experiences of the patient’s condition and fears for his life. And is there feedback, and certain defects in the administrative structures contributes to the development of malignant tumors? This question remains open, however, in the practice of social rehabilitation of the cancer centers need to give serious attention to the full EEG examination of cancer patients.

### Conclusion

a. DAFCAR methodology allows to identify cognitive dysfunction and objective to provide verified evidence of their degree

b. Using techniques DAFCAR it is possible to identify cognitive disorders, regardless of the level of national, cultural, linguistic and other differences among patients, which distinguishes it from time-consuming psychological tests.

c. Cancer patients have a fairly pronounced cognitive impairment paranoid orientation, which allows us to recommend a more thorough EEG examination of their social rehabilitation cancer centers.

d. The method DAFCAR can provide valuable objective evidence for the development of psi-chocolate cake direction in medicine, in particular to clarify the role of the Central nervous system in the pathogenesis and mechanism of cancer.

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