Investigation into the features of the brain damage by the liquidators of the Chernobyl accident has become an urgent issue of today due to a number of circumstances. According to the classical concept dominating radiobiology until recently, the brain being composed of highly differentiated nerve cells, presents a radioresistant structure responsive to radiation injury induced by high and very high radiation doses (10000 rem and higher) only. It has been universally recognized that there exist the subthreshold radiation doses harmless to human organism while higher doses produce dose-dependent biological effects. Proceeding from this theoretical assumption, the condition of the nervous system exposed to radiation has been studied quite rarely while dealing with effects of small-dose radiation on the brain was considered to be the most irrational.

The results of clinical examinations given to the Chernobyl accident recovery workers at Kiev Institute of Neurosurgery, Academy of Medical Sciences of Ukraine, show that even the so-called "small-dose" radiation, when consumed continuously, produces neurological signs of the brain damage whose development was noted to be divided in certain phases:

1. Initial response phase which should be regarded as an acute radiation encephalopathy (headaches, asthenia, nausea, vomiting, vertigo, insomnia).

2. Temporary compensation or pseudorecovery phase which occurs after people have been removed from radioactive area.

3. Decompensation phase characterized by reverting to previous complaints and disorders or worsening thereof, with addition of newly acquired symptoms, this giving the clinical picture of progressive encephalopathy which impacts all the three brain levels: the cortex, subcortex and stem. The hypothalamic and stemic disorders always prevail, however (Fig. 1).

![Diagram showing damage incidence to various brain structures in the liquidators 8-9 years after the Chernobyl accident, %](image-url)

**Fig. 1.** Damage incidence to various brain structures in the liquidators 8-9 years after the Chernobyl accident, %
Pathogenetically, this organic brain damage is a polyfactor one. Therefore, maintaining its etiological unity, it ceases to be just a radiation disease but transforms into a multi-cause postradiation encephalopathy.

Retrospective analysis of case histories showed that 89% of patients had the first phase of the brain damage. The rest 11% have assessed their condition during the first days under radiation as normal. It should be taken into account, however, that 6-9 years have passed since then, and the patients could easily forget whether they had any disturbances. This assumption may be justified by the fact that in a considerable part of patients (57%) the signs of the 1-st phase correspond to those of a mild radiation injury. Moreover, it should be noted that, firstly, all the patients were radiation-injured and, secondly, all of them demonstrated differently manifested hemogramic shifts even feeling relatively well.

Regarding the 2-nd phase, i.e. pseudorecovery and temporary compensation, this definition should be also considered as just a conventional one. According to our data, 9% of patients believed that this phase had not been recognized in them and reported no improvement in their health after the 1-st acute phase. This percentage was probably higher in fact but self-assessment is usually much influenced by personality characteristics and the extent to which one is concerned about his health condition. Nonetheless, there is no doubt that after leaving radiation zone, some improvement in health, psychological relaxation in particular, took place. For all that, unfortunately, it was just a temporary improvement and pseudorecovery. In some way or other, the 3-rd phase of the disease, i.e. progressive decompensation, occurred later on which both patients (subjectively) and doctors (objectively) have discovered in absolutely all the liquidators. Progressive nature of radiation encephalopathy is seen from our clinical records: the much time passes the fewer patients with mild symptoms are admitted to our Institute and the greater becomes the number of those diagnosed as having severe variance of the disease, i.e. the 3-rd phase of postradiation encephalopathy as illustrated in Fig.2.

![Fig.2. Incidence of differently severe postradiation encephalopathy in the liquidators as revealed 5-6 and 8-9 years after the Chernobyl accident.](image-url)
Comparing the patient population of 1991-92 with that admitted to the Institute 3-4 years later (1994-95) without any special preselection, it appears that the percentage of moderate cases remained practically stable whereas severe cases (3-rd phase) increased 3 times with simultaneous equal decrease in mild cases (1-st phase).

The 3-rd phase of the disease is characterized mainly by subcortical-stemic disturbances which are manifested in hypothalamic dysfunction (dyencephalic, hypertensive and sympathoadrenal crises, obesity, impotency, etc.) and vestibular ataxia. All the patients reveal the syndrome of dysadaptation to bodily and mental exercises as well as a drastic decline of recent memory. As the disease progresses in the 3-rd phase, it becomes possible to distinguish the 2-nd and the 3-rd degrees of severity of postradiation encephalopathy induced by the growing endogenic intoxication and hemodyscirculation.

The cause of the disease progression is that, on the one hand, the patients develop alterations in every functional system (vascular, immune, endocrinal, gastro-intestinal, etc.) and heavy metabolic disorders involving the entire biochemism and oxidative processes, thereby creating unfavourable general somatic background. On the other hand, the brain damage at the level of hypothalamic, i.e. higher vegetative regulators, promotes the development of dysadaptation syndrome. All the above factors diminish the resources of sanogenetic recovery processes and result in patient's rapid decompensation and disability.

Evidently, disability in liquidators occurs during the 3-rd phase of postradiation encephalopathy due to both somatic and neurological diseases. However, national-scale statistical data evidence that disability among the liquidators due to the brain damage increases from year to year becoming predominant. Psychoneurological deficiency in these patients is explained on the one hand by their still aggravating social dysadaptation both in

**Fig. 3** Correlation between the severity of postradiation encephalopathy and the expression of endogenic intoxication
private life and business mainly because of catastrophic memory decline. On the other hand, this results from such defects as paresis and paralysis following hemodiscirculation. The latter as seen in Fig. 4., was found in severe forms in about 15% of patients.

Of significance in the etiology of disability is that 5 - 10% of patients reveal epileptic syndrome (Fig. 5).

Thus our investigations evidence that:

1. In contrast to the postulate on the CNS radioresistance, we have found the CNS to be radiosensitive. Therefore the patients exposed to the so-called "small-dose" radiation develop progressive organic brain damage.

2. The most severely damaged are hypothalamic and stemic structures, this fact being confirmed by vegeto-vascular disorders, endocrinal shifts, vestibular ataxia and memory loss.
Parallel with clinical / neurological findings concerning the regularities in the development and course of postradiation encephalopathy, we have discovered in the liquidators multiple systemic disorders.

Preliminary correlative analysis of different pathogenic factors involved in the disease shows that these factors do not progress unidirectionally. There are probably some specific disagreement and dissociation between them.

In particular, we have found that concurrently with progressive neurological deficiency whose rate indicates the severity of postradiation encephalopathy, the serum levels of autoantibodies to the brain neuroproteins tend to rise progressively, though not so rapidly as neurological deficiency does.

This fact acquires principal significance since it proves once again the presence of organic brain damage in our patients. It is also important that parallel with the progression of the two above said characteristics, pathologic alterations in biochemistry grow up correlative, though with some fluctuations. Undoubtedly, these alterations being involved in pathological process, become one of its major parts.

In view of the above, an extremely interesting and extraordinary seems to be our finding that the levels of autoantibodies to glial cells and MBP elevate only up to the 2-nd phase of encephalopathy inclusive, and then fall down apparently. This phenomenon may be indicative of either the exhaustion of a given immunologic component or, conversely, the occurrence of adaptation.

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**Clinical characteristics**

- Level of autoantibodies to the brain proteins NSE
- Level of autoantibodies to the brain proteins S - 100, MBP
- Level of biochemical disorders (characteristics of endogenic intoxication of the acid - base balance)

Fig.6. Levels of clinical, neuroimmunological and metabolic characteristics in patients with differently severe postradiation encephalopathy.