

## Quantitative cytochemical method for assessing the activity of mitochondrial enzymes of peripheral blood lymphocytes in the acute period of stroke

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**Introduction:** Hypoxia is the main cause of neuron damage in stroke. Therefore, it is important to study the metabolism of mitochondria in the acute period of the disease. The study of the activity of mitochondrial enzymes will clarify the purpose of drug therapy and develop new approaches to treatment. The goal is to use a method that allows an objective assessment of mitochondrial disorders in the acute period of a stroke.

**Material and Methods:** The quantitative assessment of the cytochemical activity of mitochondria enzymes in peripheral blood lymphocytes was studied in 7 adult patients with ischemic stroke. For a quantitative cytochemical study of the activity of mitochondrial enzymes in peripheral blood lymphocytes, the method proposed by A.G.E. Pearse. Investigated the activity of succinate dehydrogenase (SDH) (complex 2 of the respiratory chain of mitochondria),  $\alpha$ -glycerophosphate dehydrogenase ( $\alpha$ -GPDH) (an indicator of fat metabolism involving carnitine), glutamate dehydrogenase (GDH) (an indicator of amino acid metabolism, LDH (a carbohydrate metabolism indicator). Blood sampling was performed in the first 24 hours from the start of the stroke. Two patients re-conducted the study after 7 days.

**Results:** All patients had an increase in the activity of SDH in the first 24 hours after the onset of a stroke.  $\alpha$ -GPDH activity was reduced in 6 of 7 patients (85.7%). GDG activity in patients was  $9.4 \pm 1.1$  g / lymphocyte (in the control group -  $8.8 \pm 0.3$ ). The average LDH activity in patients was  $14.8 \pm 1.5$  g / lymphocyte ( $15.4 \pm 0.3$  g / lymphocyte in the control group). In two patients with ischemic stroke, the activity of mitochondrial enzymes was re-determined 1 week after the first blood sampling. A week after the onset of the disease on the background of administration of the preparation of succinic acid (5% solution of mexidol 5.0), in the 1<sup>st</sup> patient with overweight, there was a decrease in the activity of SDH below the reference values, in the second patient (low power) more grown. Which indicates the need to take into account the body weight of patients in the appointment of the drug succinic acid or increase the dose of Mexidol for all patients.  $\alpha$ -GPDH activity was lower than the reference values on the first day of the disease in both patients. A week later, in the first patient, the index increased (but remained below the reference values), while in the second patient it decreased to 7.85 g / lymphocyte (in the control group - 9.0-12.0 g / lymphocyte). The level of GDG and LDH activity did not deviate from normal values in both patients.

**Conclusion:** The method used makes it possible to objectively evaluate the indicators of various types of metabolism in mitochondria and justify the appointment of energy-induced therapy, correct the dose of the drug.

### Biography

Olga Petrovna Sidorova Graduated from Russian National Research Medical University named after N.I. Pirogov in Moscow, residency and graduate school in Neurology. She defended her doctoral dissertation in the specialty of Neurology. She works as a Professor in the Department of Neurology of the Faculty of Advanced Medical Doctors of the Moscow Regional Clinical Research Institute. The chief of Neurological

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Department is Professor Sergey Kotov. Her research interests - Mitochondrial disorders in neurological diseases, hereditary neurological diseases, myasthenia gravis, photopheresis (extracorporeal photochemotherapy) in autoimmune neurological diseases and porphyria in neurology (acute, chronic and latent course). Together with Hanns Lochmuller, a new mutation has been identified in the gene responsible for one of the forms of congenital myasthenic syndrome. Studies mitochondrial disorders using A.G.E. Pearse in patients with various neurological pathologies.

## Notes: