Acute hypoxia or ischemia ranks among the most significant etiological factors in neurology leading to sudden brain dysfunction. In the early phase, it is characterized by membrane depolarization, resulting in excessive ion and water shifts. Cellular edema leads to a reduction in the volume of the extracellular space (ECS), and thus to an increase in the concentration of substances within it, including toxic metabolites. The duration of the pathological stimulus is the determining factor for the development of irreversible cellular damage in the form of apoptosis or necrosis.

Changes in diffusion parameters caused by hypoxic stimuli have so far been studied experimentally only in a model of global anoxia induced by cardiac arrest. In this model, dramatic changes in the size and tortuosity of the ECS of the cerebral cortex were observed within minutes after the cessation of cerebral perfusion.

We therefore decided to study changes in the diffusion parameters of the cortical ECS in models where the duration of the pathological stimulus was time-limited—specifically, models of transient hypoxia and transient hypoxia/ischemia. The obtained data were correlated with changes in extracellular concentrations of lactate, glucose, and glutamate.

During transient hypoxia/ischemia, we observed a gradual development of changes in ECS diffusion parameters throughout the duration of the stimulus. The final values were comparable to those found in terminal anoxia. In contrast, during transient hypoxia alone, the changes in ECS size and tortuosity were significantly smaller and were recorded only at the end of the hypoxic stimulus.

Changes in lactate and glucose concentrations were similar in both models; however, in the case of glutamate, the extracellular concentration at the end of hypoxia/ischemia was twice as high as at the end of hypoxia alone.