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# Early Effects of Hypoxia on Brain Cell Function

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This article reviews the changes in neuronal function produced by oxygen lack, especially as observed in hippocampal slices in vitro. An early cessation of electrical activity (”firing”), caused by a K+ conductance-mediated neuronal hyperpolarization and disappearance of excitatory synaptic potentials (EPSPs), can be seen as a protective mechanism that prevents the cellular damage resulting from severe mismatch between energy needs and supplies. These changes are triggered by such hypoxia-induced signals as a rise in cytoplasmic free calcium, fall in adenosine triphosphate (ATP), and extracellular accumulation of adenosine (produced by ATP breakdown). Upon reoxygenation, the suppression of neuronal/synaptic activity is quite reversible, as long as hypoxic nerve cells have an adequate supply of glucose. But if sufficient ATP cannot be obtained by anerobic glycolysis to maintain essential Na-K pump activity and protein synthesis, long-term cell function and survival are compromised. Thus, when both oxygen and glucose are deficient, as in strokes, the cellular protective mechanisms cannot prevent the lethal effects of excessive Ca2+ influx.

Key words: adenosine; ATP; brain; calcium; cell death; electrophysiology; hypoglycemia; hypoxia; nerve cells; neurons