

Anoxic depolarization is a progressive and uncontrollable [depolarization](#) of [neurons](#) during [stroke](#) or [brain ischemia](#) in which there is an inadequate supply of blood to the brain.

Anoxic depolarization is induced by the loss of neuronal selective membrane permeability and the ion gradients across the membrane that are needed to support neuronal activity. Normally, the Na⁺/K⁺-ATPase pump maintains the transmembrane gradients of K⁺ and Na⁺ ions, but with [anoxia](#), the supply of energy to drive this pump is lost.

The hallmarks of anoxic depolarization are increased concentrations of extracellular [K⁺](#) ions, intracellular Na⁺ and Ca²⁺ ions, and extracellular glutamate and aspartate. Glutamate and aspartate are normally present as the brain's primary excitatory neurotransmitters, but high concentrations activate a number of downstream apoptotic and necrotic pathways. This results in neuronal dysfunction and death.[3]

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Last update: **2024/06/07 02:50**

