

BRAIN TOLERANCE AND PRECONDITIONING

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In this review article the authors describe a phenomenon of “brain tolerance” which represents transient resistance of brain tissue to a lethal insult established by preconditioning with a mild insult of short duration. Tolerance evoked by brief ischemia resembles transient ischemic attack(s) (TIA) often preceding full-blown ischemic stroke in a clinical setting. A series of recent studies have described another relevant phenomenon termed “chemical preconditioning”. Several substances interfering with cellular energy metabolism applied in subtoxic doses may provide protection against lethal insults of a different type. For example, 3-nitropropionic acid (3-NP), antibiotics erythromycin and kanamycin, acetylsalicylic acid, and 2-deoxyglucose have been shown to evoke tolerance. Recently, we have reported that NMDA receptor antagonists and 2-deoxyglucose used at relatively low doses were potent agents to potentiate the protective anticonvulsant effect induced by transient brain mild ischemia. Further studies are expected to prove similar action of these drugs in other experimental models. Based on the accumulated experimental and clinical data the brain tolerance subsequently reinforced by pharmacological intervention might become a successful prophylactic strategy against serious brain insults in patients.

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