


incomplete global ischemia, short communication. Brain Research, 894:145-149


Cohadon F. (1995). The Concept of secondary Damage in Brain Trauma in Ischemia in Head Injury, Smith TCG ed. 10th European


Desagher S, Jean-Claude M. (2000). Mitochondria as the central control point of apoptosis, trend in cell biology,10

is Responsible for Mitochondrial Cytochrome c Release during Apoptosis. The Journal of Cell Biology, 144(5):891-901


and Human Services, Food and Drug Administration. Available at: http://www.fda.gov/cder/guidance/index.htm


reduces vasospasm after aneurysmal subarachnoid hemorrhage: results of a pilot randomized clinical trial. Stroke, 36:2024-2026


Menon DK. (2003). Procrustes, the traumatic penumbra, and perfusion pressure targets in closed head injury. Anesthesiology 98(4):805-7


Prins J. (1998). Tumor Necrosis Factor Induced Autofagi and Mitochondrial Morphological Abnormalities are Mediated by TNFR-1 and/or TNFR-II and don’t invariably Lead to Cell death. Biochem Soc. Trans, 26:S314


Schinder AF, Olson EC, Spitzer NC, Montal M. (1996). Mitochondrial dysfunction is a primary event in glutamate neurotoxicity. J Neurosci, 16(19):6125-6133


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Cerebral Cortex Following Traumatic Brain Injury in Rats. Journal of Neurotrauma, 22(22):658-68


