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论著

异丙酚对缺血再灌注损伤大鼠海马氨基酸递质水平变化及神经元凋亡的影响

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摘要 目的： 观察异丙酚对脑缺血再灌注损伤大鼠海马组织氨基酸递质水平变化及神经元凋亡的影响。方法：雄性Wistar大鼠60只，随机等分为假手术（A）组、缺血再灌注对照（B）组和异丙酚处理（C）组，后者按异丙酚用量又分为50 mg/kg（C_1）、100 mg/kg（C_2）和150 mg/kg（C_3）3个亚组。全脑缺血10 min再灌注60 min时，各组断头处死大鼠6只，检测海马组织中氨基酸水平的变化；于再灌注72 h时，断头处死余下大鼠，用流式细胞仪检测海马组织神经细胞凋亡指数（AI），光镜检测海马CA1区神经元凋亡的密度。结果：与假手术组比较，各组海马组织谷氨酸（Glu）、天冬氨酸（Asp）含量有不同程度的增高，γ-氨基丁酸（GABA）、甘氨酸（Gly）的含量有不同程度的降低(P<0.05或P<0.01)。异丙酚组海马组织Glu、Asp含量低于对照组，GABA、Gly的含量高于对照组（P<0.05或P<0.01）。再灌注72 h时，各组海马组织神经细胞AI有不同程度的增高，海马CA1区神经元凋亡密度明显增加。异丙酚组海马组织神经细胞凋亡数量明显减少。结论：异丙酚对脑缺血再灌注损伤的拮抗作用可能与其抑制兴奋性氨基酸的释放、降低抑制性氨基酸的耗竭、继而减少神经元的凋亡、提高神经元的存活数目有关。

关键词 二异丙酚； 脑缺血； 再灌注损伤； 氨基酸类； 细胞凋亡； 神经元

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Effects of propofol on levels of amino acid and neuronal apoptosis of hippocampus after global ischemia-reperfusion in rats

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Abstract

<FONT face=Verdana>AIM:To investigate the effects of propofol on levels of amino acid and neuronal apoptosis of hippocampus after global ischemia-reperfusion in rats.
METHODS:60 male Wistar rats were randomly assigned to five groups (twelve animals each). After global cerebral ischemia for 10 min then reperfusion for 60 min and 72 h,the animals were decapitated and the brains were removed respectively. HPLC was adopted to measure the contents of amino acids in hippocampus. The density of apoptosis neurons in the hippocampal CA1 subfield was evaluate with light microscope. Flow cytometry technique was applied to detect the neuronal apoptosis index in the hippocampus.
RESULTS:The contents of Glu and Asp increased markedly and the levels of GABA and Gly decreased obviously in hippocampus after ischemia-reperfusion. The levels of Glu and Asp were lower in propofol group than those in control group (P<0.05 or P<0.01),and the contents of GABA and Gly were higher in propofol group than those in control group (P<0.05 or P<0.01). Apoptosis index and density of apoptosis neurons in the hippocampus were higher in control group than those in propofol group.
CONCLUSION:Propofol inhibits neuronal apoptosis of hippocampus after global ischemia-reperfusion,and suppresses the excessive release of excitory amino acids and the exhaustion of inhibitory amino acids in

hippocampus after ischemia-reperfusion. Its mechanism may be related with decreasing the neuronal apoptosis.</FONT>

Key words Propofol Brain ischemia Reperfusion injury Amino acids Apoptosis Neurons

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