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神经性疼痛对老龄大鼠学习记忆功能和海马超微结构的影响 [点此下载全文](#)

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摘要:

摘要目的:观察神经性疼痛对老龄大鼠学习记忆功能及脑脊液S100B蛋白表达、海马CA1区突触超微结构的影响。方法:18月龄雄性Wistar大鼠60只,随机分为3组:正常组(N组),假手术组(S组),坐骨神经结扎组(C组),松扎左侧坐骨神经。各组术后第1、7、14、21天再随机分为4个亚组,每组5只。测大鼠热刺激回缩潜伏期(PWTL),用Morris水迷宫测大鼠学习记忆功能,并记录基础值,麻醉后抽取脑脊液ELISA法检测S100B蛋白浓度,透射电镜观察大鼠海马CA1区GrayI型突触结构的变化。结果:①与基础值比较,C组PWTL缩短显著($P<0.01$)。②与基础值比较,C组大鼠潜伏期显著延长($P<0.01$)和穿越平台次数显著减少($P<0.01$);与N组和S组比较,差异有显著性($P<0.01$)。③与N组和S组比较,C组脑脊液S100B蛋白浓度显著升高($P<0.01$)。④透射电镜观察,C组大鼠海马突触间隙的增宽,突触囊泡减少。结论:神经性疼痛可致老龄大鼠的空间学习记忆能力下降,脑脊液S100B蛋白表达上调和突触可塑性改变可能是其机制。

关键词: [神经性疼痛](#) [老龄大鼠](#) [学习记忆](#) [S100B蛋白](#) [海马](#) [突触](#)

Effects of neuropathic pain on the abilities of learning and memory and synaptic ultrastructure of hippocampus in aged rats [Download Fulltext](#)

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Abstract:

Abstract Objective: To investigate the effects of neuropathic pain on the abilities of learning and memory, and the level of protein S100B in cerebrospinal fluid(CSF), the change of synaptic ultrastructure of CA1 area in hippocampus of aged rats. Method: Total 60 male 18-month-old Wistar rats were randomly divided into 3 groups: normal group (N), sham group (S), chronic constriction nerve injury (CCI) group(C). In CCI model sciatic nerve was ligated loosely. Each group was randomly divided into 4 subgroups at 1st, 7th, 14th, 21th d, 5 rats in each group. The paw withdrawal thermal latency(PWTL) was tested, the ability of learning and memory was assessed with Morris water maze(MWM), the concentration of protein S100B in CSF was detected by enzyme-linked immunosorbent assay(ELISA) after anesthesia, and the synaptic ultrastructure of hippocampus was observed under transmission electron microscopy. Result: ①In group C, the PWTL was shortened significantly after operation ($P<0.01$), there was no significant difference between group N and group S. ②Compared with group N and group S, in group C the escape latency was significantly longer than before($P<0.01$), and the frequency of passing through platform was less than that in group N and S($P<0.01$). ③Compared with group N and group S, the concentration of protein S100B of group C increased significantly($P<0.01$). ④The width of synaptic gap increased, and the postsynaptic densities decreased, synaptic vesicles densities decreased significantly. Conclusion: Neuropathic pain could impair the abilities of spatial learning and memory in aged rats; up-regulation of protein S100B expression in CSF and synaptic plasticity of hippocampus might be involved in the mechanism.

Keywords: [neuropathic pain](#) [aged rats](#) [learning and memory](#) [S100B protein](#) [hippocampus](#) [synapse](#)

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