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电针对局灶性脑梗死大鼠Nogo-A及其受体NgR和运动诱发电位的影响

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Title: Effect of electroacupuncture on motor evoked potential and expression of Nogo-A and its receptor NgR in rats with focal cerebral ischemia

作者: 伍芳; 龚标; 李学智; 黄思琴; 王力; 方毅; 李凤; 吕凯
重庆医科大学中医药学院

Author(s): Wu Fang; Gong Biao; Li Xuezhi; Huang Siqin; Wang Li; Fang Yi; Li Feng; Lu Kai
College of Traditional Chinese Medicine, Chongqing Medical University, Chongqing, 401331, China

关键词: 电针; 脑梗死; 运动诱发电位; Nogo-A; NgR

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摘要: 目的 观察电针对局灶性脑梗死大鼠运动诱发电位和梗死周围组织神经抑制因子Nogo-A及其受体NgR等的影响,探讨电针治疗脑梗死的机制。 方法 将36只成年SD大鼠,分为正常对照组、模型组和电针组(每组12只)。电针组和模型组按照改进后的Longa的方法制作大鼠中动脉闭塞模型。正常组和模型组不做任何治疗,电针组在造模成功后第1天进行电针治疗。3组均在造模后1 d和14 d进行运动诱发电位(MEP)检测,14 d时进行HE染色和Nissl染色观察大鼠脑组织病理变化,免疫组化染色和Western blot法检测Nogo-A和NgR在脑组织内的表达。 结果 14 d时,电针组MEP N1波的潜伏期(15.38±1.58) ms和N2波的潜伏期(33.60±3.58) ms较模型组N1波的潜伏期(21.28±4.00) ms和N2波的潜伏期(41.78±3.07) ms明显好转($P<0.01$); HE染色结果显示,电针组脑组织较模型组梗死区域的神经细胞病变程度明显改善; Nissl染色结果显示,电针组的Nissl小体数目较模型组增多,肿胀的神经细胞内可见Nissl小体分布;免疫组化检测结果显示,电针组的Nogo-A和NgR的表达较模型组明显减少($P<0.05$, $P<0.01$); Western blot检测结果显示,电针组Nogo-A蛋白(22.45±0.95)%和NgR蛋白(26.76±1.14)%较模型组[Nogo-A蛋白(43.75±6.21)%,NgR蛋白(54.50±5.00)%]明显减少($P<0.01$)。 结论 电针能明显改善

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善急性脑梗死大鼠的神经传导通路,改善梗死区组织病理表现,减少Nogo-A和NgR在脑组织内的表达,促进神经功能缺失症状的恢复。

Abstract: **Objective** To observe the effect of electroacupuncture (EA) on motor evoked potential (MEP) and expression of Nogo-A and Nogo receptor (NgR) in brain tissues of rats with focal cerebral ischemia, and to investigate the mechanism of EA in treatment of focal cerebral ischemia. **Methods** Thirty-six adult SD rats were divided into a normal group, a model group and an EA group. Rat models of middle cerebral artery occlusion were successfully established by improved Longa procedures in the model group and EA group. The rats of the normal group and model group were not given any treatment, while those of the EA group were given EA treatment on the 1st day after operation for 14 d. The latency of MEP was examined on the 1st day and 14th day after operation. HE staining and Nissl staining were used to observe the pathological changes of brain tissues, and immunohistochemistry and Western blotting were used to detect the expression of Nogo-A and NgR on the 14th day after operation. **Results** After 14 d, the latency of MEP N1 waves $[(15.38 \pm 1.58)\text{ms}]$ and N2 waves $[(33.60 \pm 3.58)\text{ms}]$ in the EA group was significantly shorter than that [N1 waves $(21.28 \pm 4.0)\text{ms}$ and N2 waves $(41.78 \pm 3.07)\text{ms}]$ in the model group ($P < 0.01$). There was significant difference in brain pathology between the model group and EA group. Immunohistochemical staining and Western blotting results indicated that the expression levels of Nogo-A and NgR in the EA group were significantly lower than those in the model group ($P < 0.05$, $P < 0.01$). **Conclusion** EA is an effective method to improve the neural pathways and cerebral histopathological status, decrease the expression of Nogo-A and NgR, and promote the recovery from neurological deficits after focal cerebral ischemia.

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