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Inhibitive effects of electroacupuncture on pain response and c-fos expression in spinal cord induced by substance P in rats

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Abstract Objective: To observe the effects of electroacupuncture at the "Jiaji" point on pain response and c-fos expression in spinal cord induced by intrathecal injection of substance P (SP, 10μg) in rats. Methods: Immunohistochemistry and measurement of pain were used. Results: The intrathecal injection of SP produced pain response and evoked c-fos expression in spinal cord. 1h after the injection of SP, the FLI neurons were localized in laminae I, II, V and VI of both sides of the spinal cord. Electroacupuncture at the "Jiaji" point could inhibit pain response and c-fos expression, which could be partially reversed by naloxone. Conclusion: Electroacupuncture might exert its analgesic effects through stimulating the internal opiate peptide to inhibit SP.

Key Words substance P; electroacupuncture; c-fos; spinal cord; rat

大鼠生后脑发育和缺血再灌流过程中编程性细胞死亡及其可能机理的研究

A study of programmed cell death and its possible mechanism during postnatal brain development and ischemia-reperfusion in rats

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编程性细胞死亡(Programmed cell death, PCD)是脑发育及损伤中神经细胞死亡的重要形式。已知的 PCD调节基因中, bcl-2家族及白介素-1β转化酶(Interleukin-1β converting enzyme, ICE)基因在 PCD的调节中起重要作用。体外研究提示, bcl-2和 bcl-xl基因表达可阻止神经细胞 PCD,而 Bax及 ICE则促进 PCD。这些基因在脑发育及缺血再灌流中的表达及与 PCD的关系报道资料较少。本实验应用 DNA片段原位末端标记(TUNEL)、核酸原位杂交(ISH)、免疫组化(ICC)、流式细胞仪(FCM)以及电镜(EM)等方法,研究大鼠生后脑发育及缺血再灌流过程中 PCD的发生、以及 bcl-2 bcl-xL Bax和 ICE基因的表达,旨在了解生后脑发育变化以及在调节 PCD中的规律; bcl-2 bcl-xL Bax和 ICE基因表达变化以及在调节 PCD发生中的作用。主要结果如下:① 应用 TUNEL FCM电镜,结合图像分析对大鼠生后脑发育过程中 PCD的观察发现: 在 TUNEL染色上 PCD细胞表现为染色质凝聚成块、移向核边缘、有时形成环半月状,偶尔可见见凋亡小体等。TUNEL法与电镜结合显示,只有 TUNEL深染细胞核有凋亡细胞特征,而核淡染细胞不具有凋亡细胞表现: 本实验首次将 TUNEL与 FCM结合研究神经细胞 PCD发生规律; P1-P7d PCD细胞呈上升趋势, P7d达到高峰,之后 PCD细胞减少, P14d后急剧下降, P28 d以后 PCD细胞基本消失。大脑皮层、海马、皮层下白质和基底节区均存在 PCD细胞,提示生后脑内 PCD发生在四周之内, PCD是生后脑发育中神经细胞死亡的主要方式。② 应用核酸原位杂交和免疫组化等技术,对生后脑发育中 ICE bcl-2 bcl-xL和 Bax基因表达进行观察。结果显示, ICE在生后一周内表达较强, P7-P28 d呈减弱趋势; Bax蛋白在生后一周上升, P7 d后表达减弱, P14 d后降至较低水平, ICE和 Bax具促进 PCD发生作用。bcl-2在胚胎期有明显表达而生后未见明

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Effects of benazepril on congestive heart failure and its neuro-humoral mechanism

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Abstract Objective: To investigate the effects of benazepril (BNZ) on congestive heart failure (CHF) and its neuro-humoral mechanism. Methods: Anti-CHF trratment (digoxin+ diuretic) was given to 32 cases of CHF (NYHA class: Ⅱ, n = 19; Ⅲ, n = 10; Ⅳ, n = 3), but the β-adrenergic blooking agents and calcium antagonist were withdrawn. Benazepril (BNZ) 10mg/d for 20~ 30 d were added. Plasma renin activity (PRA), the coutents of angiotensinⅡ (ATⅡ), ventricular functions (SV, CO, EF, and PEP/LVET) were determined. Results: In all patients, the symptom of CHF was considerably alleviated and exercise time significantly increased (P < 0.01). Consequently, the left ventricular function was markedly improved. The mean blood pressure in patients of the hypertensive heart disease group(n = 11) were decreased from 22.7/13.8 kPa to 18.8/11.2 kPa. After the treatment with BNZ, PRA was significantly increased (P < 0.05) and the contents of ATⅡ, NE, E, ET and ANP significantly decreased (P < 0.01). Conclusion: BNZ definitely exerts therapeutic effects on CHF through inhibiting the activation of sympathetic system, renin-angiotensin system and endothelial system.

Key Words benazepril; ACE inhibitor; heart failure; renin-angiotensin; endothelin; atrio-natriuretic peptide

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显表达及变化; Bcl-xL在 P1-P14d表达上升,在成年一直维持较高表达。生后 ICE和 bcl-2家族基因表达变化与 PCD发生密切相关,提示这些基因参与调节生后 PCD的发生。本实验进一步证明了 PCD的发生是多种基因表达的综合结果。③对脑缺血再灌流过程中细胞 PCD研究发现,轻度脑缺血(10 min)和重度缺血(30 min)再灌流中均可见 PCD细胞, PCD细胞出现高峰时间在 48~ 72 h,重度缺血 PCD细胞较轻度缺血多;轻度缺血时坏死细胞很少,仍可见到明显 PCD细胞;大脑皮层、海马 CA1缺血敏感区 PCD细胞相对较多。提示脑缺血能诱导神经细胞 PCD,加重缺血程度可能促进 PCD发生,细胞 PCD可能为轻度缺血和迟发性神经细胞损害的主要形式。给予大量牛磺酸可降低细胞 PCD发生,反映牛磺酸对脑缺血的保护作用可能部分由于降低细胞 PCD的发生。④脑缺血再灌流中 bcl-2基因未见明显表达变化, bcl-xL表达下降, ICE和 Bax表达增加,这些变化参与细胞 PCD的调节。在分布上, Bcl-xL和 Bax表达在缺血损伤的敏感区如大脑皮层及海马 CA1区变化更为明显。

关键词　脑;发育;脑缺血; bcl-2; ICE;牛磺酸
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