

论文

气导听觉剥夺大鼠螺旋神经节NSE表达及凋亡

谢宁<sup>1</sup>, 朱晓全<sup>1</sup>, 王玉平<sup>2</sup>, 王海波<sup>2</sup>, 夏阳<sup>1</sup>

1. 空军航空医学研究所附属医院, 北京100089;
2. 空军后勤部卫生部

摘要:

目的 观察发育期气导听觉剥夺大鼠螺旋神经节神经元特异性烯醇化酶(NSE)表达及细胞凋亡变化。方法 60只新生SD大鼠随机分为听觉剥夺组和对照组,每组30只,听觉剥夺组于出生后早期行外耳道隔离,饲养于密闭隔音室,对照组正常食水、声音环境饲养;42 d后进行听觉脑干诱发电位(ABR)检测,耳蜗切片进行螺旋神经节NSE染色神经元计数及细胞染色观察。结果 听觉剥夺组大鼠ABR反应阈值[(38.18±5.54)dB SPL]较对照组[(26.67±3.89)dB SPL]增高( $P<0.01$ );NSE免疫组化染色神经元着色浅淡,神经纤维着色基本正常;听觉剥夺组大鼠神经元计数[(7.883±0.987)个/视野]较对照组[(10.643±1.104)个/视野]减少( $P<0.05$ );听觉剥夺组大鼠螺旋神经节可见散在凋亡细胞。结论 发育期气导听觉剥夺可造成大鼠螺旋神经节 I 型NSE表达下降,神经元数量减少,出现细胞凋亡。

关键词: 听觉剥夺 螺旋神经节 神经元特异性烯醇化酶(NSE) 细胞凋亡

Neuron-specific enolase expression and cell apoptosis in spiral ganglion in rats with air conduction hearing deprivation

XIE Ning, ZHU Xiao-quan, WANG Yu-ping, et al

Affiliated Hospital of Aeromedicine Institute, Air Force of People's Liberation Army, Beijing 100089, China

Abstract:

Objective To observe spiral ganglion neuron-specific enolase (NSE) expression and cell apoptosis in spiral ganglion in rats with air conduction hearing deprivation. Methods Totally 60 newborn Sprague-Dawley rats were randomly divided into auditory deprivation group ( $n=30$ ) and the control group ( $n=30$ ). The rats in auditory deprivation group were administered external auditory canal isolation after birth and reared in confined soundproof room. The rats in control group were reared with normal drinking water in sound environment. Auditory brainstem evoked potentials (ABR) was measured. Cochlea tissue slice and spiral ganglion staining were conducted for neuron number count and observations of NSE expression and cell structure change. Results The ABR reactor threshold (38.18 ? 5.54 dB SPL) of auditory deprivation rats was significantly higher compared with that of the control group (26.67 ? 3.89 dB SPL) ( $P<0.01$ ). The staining of NSE immunohistochemical neurons was pale. The neuron count (7.88 ? 0.99 per field of view) was significantly reduced in auditory deprivation rats ( $P<0.05$ ). Scattered apoptotic cells were observed in spiral ganglion of auditory deprivation rats. Conclusion Air conduction hearing deprivation can decrease type I NSE expression and the number of neurons, and induce cell apoptosis in spiral ganglion of rats during development period.

Keywords: auditory deprivation spiral ganglion neuron-specific enolase apoptosis

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作者简介:

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