



2.3 3组在不同条件下的错误率及干扰量的比较 对3组分别在3种条件下的错误率进行组间独立样本 Kruskal-Wallis 检验,结

果 AD组 > MCI组 > 正常对照组 ( $P < 0.05$ )。干扰量 AD组明显高于 MCI和正常组 ( $P < 0.05$ ), MCI组与正常组无显著性差异(表3)。

表3 3组在不同条件下的错误率及干扰量

组别	错误率(%)			干扰量
	冲突	一致	中性	
AD组	15.00 ± 8.60 <sup>1)2)</sup>	7.50 ± 8.26 <sup>1)2)</sup>	7.50 ± 4.75 <sup>1)2)</sup>	7.50 ± 4.75 <sup>1)2)</sup>
MCI组	5.00 ± 4.50 <sup>2)</sup>	5.00 ± 3.95 <sup>2)</sup>	5.00 ± 4.40 <sup>2)</sup>	0.00 ± 1.34
正常组	5.00 ± 2.63	0.00 ± 1.62	1.25 ± 2.43	2.50 ± 3.31

1)与 MCI组相比较,  $P < 0.05$  2)与正常组比较,  $P < 0.05$

### 3 讨论

本研究表明冲突情况下轻度 AD组较其他两组干扰效应更明显,犯错误更多,提示轻度 AD患者对干扰的抑制减弱,已出现选择注意功能异常。MCI组和正常对照组间干扰量无明显差别,但 MCI组总错误率和各条件下的错误率已明显高于正常对照组,这可能提示 MCI患者选择注意并非完全正常,已出现注意转换上的困难。

国外研究提示 AD患者早期即出现注意障碍,认为注意是记忆以后 AD患者第二个受损的功能,早于语言和视空间<sup>[2-3]</sup>,而且多数研究认为选择注意是最早受累的注意亚型,极早期的 AD患者即出现选择注意障碍。本研究得到相似的结果,而且发现 MCI患者可能已经出现选择注意障碍。

### 参 考 文 献

- Petersen RC, Smith GE, Waring SC, et al. Mild cognitive impairment: Clinical characterization and outcome. *Lancet Neurol*, 2000, 5(2):202.
- Grady CL, Haxby JV, Horwitz B, et al. Longitudinal study of the early neuropsychological and cerebral metabolic changes in dementia of the Alzheimer type. *Journal of Clinical and Experimental Neuropsychology*, 1988, 10(5):576.
- Richard J, Peter W, John R. The nature and staging of attention dysfunction in early (minimal and mild) Alzheimer's disease: relationship to episodic and semantic memory impairment. *J Neuropsychologia*, 2000, 38(3):252.
- Arnold HM, Burk JA, Hodgson EM, et al. Differential cortical acetylcholine release in rats performing a sustained attention task versus behavioral control tasks that do not explicitly tax attention. *J Neuroscience*, 2002, 114(2):451.
- Gill TM, Sarter M, Givens B. Sustained visual attention performance-associated prefrontal neuronal activity: evidence for cholinergic modulation. *J Neurosci*, 2000, 20(12):4745.
- Wojcik J, Wessely M, Pagan AE. Deficits in selective and divided attention associated with Cholinergic Basal Forebrain immunotoxic lesion produced by 192-Saporin: motoric/sensory deficit associated with Purkinje cell immunotoxic lesion produced by OX7-Saporin. *J Neurobiology of Learning and Memory*, 1999, 71(3):325.

tion associated with Cholinergic Basal Forebrain immunotoxic lesion produced by 192-Saporin: motoric/sensory deficit associated with Purkinje cell immunotoxic lesion produced by OX7-Saporin. *J Neurobiology of Learning and Memory*, 1999, 71(3):325.