

Introduction

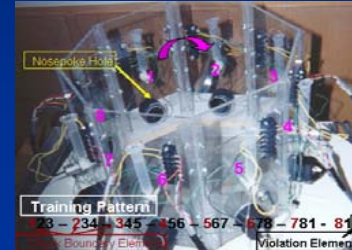
In prior research medial caudate putamen lesions caused small but distinct deficits in rat serial pattern learning. These deficits were different than those previously observed for the drugs MK-801 and atropine, hippocampal lesions, or medial frontal lesions. In the present study 12 rats with medial caudate putamen (MCPu) lesions and 12 controls learned to nose poke receptacles for water reward in a sequential pattern. MCPu lesions caused small learning deficits for all pattern elements. However, of the 12 experimental rats, two received lesions of distinctive proportions. Rat 13, after histological examination was found to have an accurate MCPu lesion while examination of rat 30 revealed a very large lesion extending beyond MCPu to encompass the majority of the caudate putamen structure. Interestingly, Rat 13 with the smaller lesion showed profound learning deficits on the Violation and Chunk Boundary elements while Rat 30 with the more extensive CPU lesion learned the violation at least as well as controls and the chunk boundary element with little difference than controls.

Methods

24 naïve male Long Evans rats at least 90 days old, underwent surgery. 12 received bilateral Medial Caudate Putamen (MCPu) lesions. The excitotoxic lesions were produced with quinolinic acid injections to the MCPu. 12 control rats received the same surgery with no lesioning procedure.

In a Plexiglas octagonal chamber equipped with a nose poke receptacle on each wall, rats learned to produce a highly-structured serial pattern of responses for water reinforcement: 123-234-345-456-567-678-781-818

Integers indicate the clockwise position of correct responses on successive Trials. Trials were separated by 1 s except where dashes indicate 3-s phrasing cues. The final element, 8, was the violation Element.



Discussion

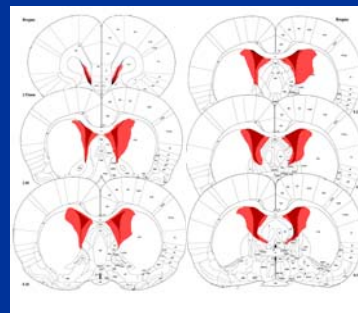
Present theory attributes cognitive Motor and visual function to the Head, tail and body of the CPU respectively. MCPu lesioned rat 13 demonstrated profound learning deficits while lesioning nearly the entire CPU of rat 30 resulted in less severe deficits. Possible explanations for this dissociation could be the differing roles of different parts of the CPU or disruption of inhibitory processes and disinhibition.

Conclusion

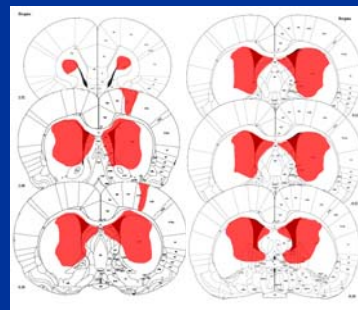
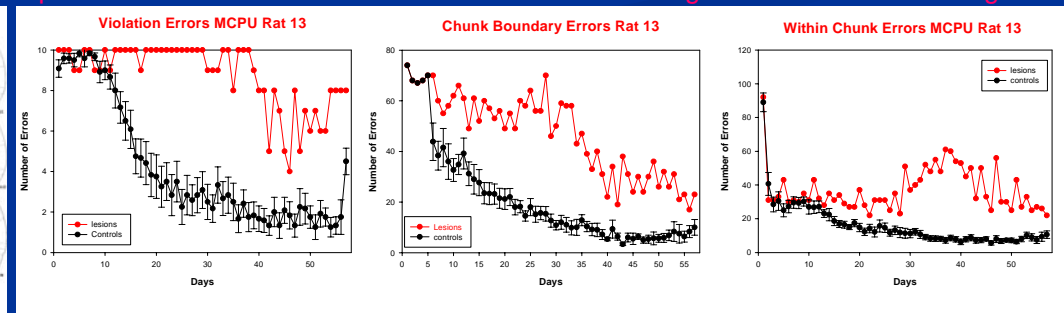
In serial pattern learning, MCPu lesions produce more profound learning deficits than combined medial and lateral lesions

Results

Rat 13 with the smaller MCPu Lesion had profound learning deficits for the violation and chunk boundary elements in Comparison to Rat 30 with medial and lateral CPU lesioning and less severe learning deficits



Rat 13 Histology



Rat 30 Histology

