Results

Rotarod: A Student t-test revealed a significant difference between groups on D100 (F(1,18) = 4.16, p = 0.05). Vertical pole test: While the mice were placed head upward on a vertical wooden pole, the latency to fall was measured. For this test, mice were placed head downward on a vertical wooden pole. Both the time to fall (s) and the time to reach the fluid (s) were recorded. Over three trials on D 0 and D 21, mice were injected with d-biotinylated IgG (Chemicon (100 µg/mouse)). Twenty minutes later, they were placed in a box 50 cm in the air. 3 min rotation behavior were analyzed with a videotaping software (Neuroscan). Serial Order Learning. After acquisition of the operant sequence, food deprivation was reduced (12 sessions of 60 min). Each session was repeated twice, and the latency to complete the sequence was measured. No significant difference was found between the two groups. At each trial, mice were injected with d-biotinylated IgG (Chemicon (100 µg/mouse)). Other sequences were not reinforced. As in inter-trial intervals separated two trials, lesions obtained were mostly be delineated or after an hour, because file test. Antibody (anti-NeuN, Chemicon) was used to detect NeuN (n = 5 mice per group). The number of neurons was counted in the insular cortex, the striatum, and the cortex. The difference between the two groups was distinguished according to the latency between two groups. The difference between the two groups was distinguished according to the latency between two groups. One of the most practical advantages of the SOL model is its sensitivity to changes in the action outcome contingency in a mouse model of mild to moderate ischemia. Cogn Behav Neurosci. 2010; 30(4): 379–387.

Discussion

Our results pointed to a deficit regarding procedural learning after a 30 min MCAO in mice, as well as an impaired motor coordination and an amphetamine-induced rotation bias. Moreover, we used accelerated rote learning, vertical pole test and amphetamine-induced rotation test to further assess the consequence of MCAO on sensory and motor functions.

Methods

Session 0 (D0): 21 days old male C57/Bl6J mice underwent 30 min transient middle cerebral artery occlusion. This model was performed using a modified model from the original MCAO method. Briefly, the mice were anesthetized by pentobarbital (50 mg/kg, i.p.) and placed in a stereotaxic frame (David Kopf Instruments). The left common carotid artery and the internal carotid artery were exposed by a midline incision and a median approach. The external carotid artery was ligated. A 0.5 mm steel filament was inserted into the left common carotid artery, and then the middle cerebral artery was occluded for 30 min. Following occlusion, the steel filament was removed, and 100 µl of saline was injected into the right internal carotid artery. The mice were allowed to recover for 2 h after surgery. On D 0 and D 21, mice were injected with d-biotinylated IgG (Chemicon (100 µg/mouse)). Twenty minutes later, they were placed in a box 50 cm in the air. 3 min rotation behavior were analyzed with a videotaping software (Neuroscan). Serial Order Learning. After acquisition of the operant sequence, food deprivation was reduced (12 sessions of 60 min). Each session was repeated twice, and the latency to complete the sequence was measured. No significant difference was found between the two groups. At each trial, mice were injected with d-biotinylated IgG (Chemicon (100 µg/mouse)). Other sequences were not reinforced. As in inter-trial intervals separated two trials, lesions obtained were mostly be delineated or after an hour, because file test. Antibody (anti-NeuN, Chemicon) was used to detect NeuN (n = 5 mice per group). The number of neurons was counted in the insular cortex, the striatum, and the cortex. The difference between the two groups was distinguished according to the latency between two groups. One of the most practical advantages of the SOL model is its sensitivity to changes in the action outcome contingency in a mouse model of mild to moderate ischemia. Cogn Behav Neurosci. 2010; 30(4): 379–387.