Brain Disorders/Neurological

Hyperbaric oxygenation mitigates focal cerebral injury and reduces striatal dopamine release in a rat model of transient middle cerebral artery occlusion.


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The usefulness of the administration of hyperbaric oxygen (HBO) in the treatment of acute focal cerebral ischemia remains debatable. A significant association exists between focal cerebral injury and an excessive release of extracellular dopamine (DA). In vivo microdialysis was used in the present study to examine the effect of HBO on DA release in the striatum during ischemia and reperfusion in rats. The histological changes occurring were also evaluated. Focal cerebral ischemia was induced by occlusion of the middle cerebral artery (MCA) using a surgically placed intraluminal filament. Control rats (n=8) were subjected to 1 h of ischemia, whilst the study rats (n=8) were in addition treated with HBO (2.8 atmospheres of absolute pressure 100% O(2)) during ischemia. Both groups were returned to breathing room air at normal pressure during reperfusion. Microdialysis samples were continuously collected at 15 min intervals at 2 microl.min(-1). The [mean (SE)] increase in release of striatal DA attained significance after 30 min of occlusion of MCA [170 (24)%], and continued to increase [268 (26)% at 45 min] reaching a peak level at 60 min [672 (59)%] before returning to the baseline level during the late reperfusion phase. There was no significant change in the level of DA in HBO treated rats during the period of ischemia.

A significant reduction in edema and neuronal shrinkage were observed by histological examination in HBO treated rats when compared to the control rats. The results showed that HBO, when administered during ischemia, offered significant neuroprotection in our experimental model of transient focal cerebral ischemia in the rat. The mechanism seems to imply, at least in part, a reduced level of DA.

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