

Hyperbaric oxygen treatment attenuated the decrease in regional glucose metabolism of rats subjected to focal cerebral ischemia: A high resolution positron emission tomography study .

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## Abstract

Cerebral hypoxia may be the main component of cell damage caused by ischemia. Previous studies demonstrated a neuroprotective effect of early hyperbaric oxygen (HBO) treatment in various animal models of focal cerebral ischemia. Neuropathologic study showed that exposure of HBO may prevent cell death in ischemic cortex. In the present study, we aimed to assess cellular function of ischemic rat brain after HBO treatment by means of a high-resolution positron emission tomography scanner (microPET) used specifically for small animal imaging. The male Sprague–Dawley rats were subjected to permanent middle cerebral artery occlusion (MCAO), with the regional cerebral blood flow monitored *in vivo* by laser Doppler flowmetry. One hour after ischemia, HBO therapy (3 atm absolute, 1 h) was initiated. Local cerebral glucose utilization in the ischemic area was measured before, 1 h and 3 h after ischemia, with 2-[<sup>18</sup>F]-fluoro-2-deoxy-d-glucose (FDG) as a tracer. Neurological deficits and infarct volumes were assessed at 24 h after ischemia. Our study showed that early HBO therapy significantly reduced infarct volume of brain 24 h after ischemia. Moreover, glucose utilization in the ischemic area underwent a severe decrease during 1–3 h after MCAO, while the early HBO treatment significantly attenuated the decrease in cerebral metabolic rate of glucose in the ischemic core of the cortex compared with controls. We report for the first time the application of microPET to quantify the rates of glucose metabolism in the ischemic core of rats exposed to HBO. Our results suggest that the early exposure of HBO can partially reverse the downward trend for glucose utilization in the ischemic core, which might contribute to the reported beneficial effects of early HBO therapy on permanent cerebral ischemia.

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