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The effect of oxygen therapy on brain damage and cerebral pO(2) in transient focal cerebral ischemia in the rat.

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Source

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Abstract

We examined the effect of hyperbaric oxygen (HBO) and normobaric oxygen (NBO) on neurologic damage and brain oxygenation before and after focal cerebral ischemia in rats. A middle cerebral artery occlusion (MCAO)/reperfusion rat model was used. The rats were sacrificed 22 h after reperfusion, and the infarct volume was evaluated. In study A, HBO (2.0 ATA), NBO (100% oxygen) and normobaric air (NBA) were each administered for 60 min in five different rat groups. The sizes of the infarcts after HBO and NBO applied during ischemia were 8.8 +/- 2.8% and 22.8 +/- 3.7% respectively of the ipsilateral non-occluded hemisphere. The infarct size after HBO applied during ischemia was statistically smaller than for NBO and NBA exposure ($p < 0.01$). In study B, cerebral pO(2) was measured before and after MCAO and HBO exposure (2.0 ATA for 60 min) in six rats using electron paramagnetic resonance (EPR) oximetry. The pO(2) in the ischemic hemisphere fell markedly following ischemia, while the pO(2) in the contralateral hemisphere remained within the normal range. Measurements of the pO(2) performed minutes after HBO exposure did not show an increase in the ischemic or normal hemispheres. The mean relative infarct size was consistent with the changes observed in study A. These data confirm the neuroprotective effects of HBO in cerebral ischemia and indicate that in vivo EPR oximetry can be an effective method to monitor the cerebral oxygenation after oxygen therapy for ischemic stroke. The ability to measure the pO(2) in several sites provides important information that should help to optimize the design of hyperoxic therapies for stroke.

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