SPECIALIA

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Uptake of a Metabolically Inert Amino Acid by Brain Tissue During High Pressure Oxygen Exposure

Large intraperitoneal doses of γ-aminobutyric acid (GABA) protect animals against high pressure oxygen (HPO)-induced convulsions[1], apparently by elevation of brain GABA levels. Although normally impermeable to GABA[2,3], the blood-brain barrier may be affected by HPO to facilitate GABA transfer into the brain[4]. Attempts to demonstrate such an HPO-accelerated neuronal accumulation of GABA have been unsuccessful[5,6], possibly due to metabolism of accumulating GABA by brain GABA-transaminase.

Use of an inert amino acid, such as α-amino-isobutyric acid (AIB)[7–9], to assess blood-brain barrier function under HPO would circumvent the problem of intraneuronal metabolism of the administered amino acid. The effect of HPO on the uptake of AIB into the brain from the blood is reported in this study.

Methods. Male Wistar rats, weighing 180–190 g, were injected s.c. with 0.54 ml 0.9% NaCl containing 5 μc AIB-1-C^{14} (specific activity 10 mC/mM). 15 min after injection, the rats were exposed in individual lucite chambers to 4 pressures of oxygen, 52, 60, 68 and 75 psig, for 30 min. Rats maintained at ambient pressure served as controls. Animals were decapitated as quickly as possible after decompression, and brain tissue and blood collected and prepared for analysis according to the method of Raisz and O'Brien[10]. Only non-convulsed rats were used. Radioactivity of serum and brain was measured using a Packard Scintillation Spectrometer. Counts were corrected for quenching by internal standardization and AIB-C^{14} content expressed as disintegrations per minute (dpm).

Results and discussion. The results of the experiment are shown in the Table. Exposure to the various pressures of oxygen resulted in significantly decreased ($P < 0.02$) brain concentrations (dpm/g wet wt) of isotope compared to the control value. Serum concentrations (dpm/ml) were similarly decreased. Changes in brain concentrations were directly related to changes in serum concentrations of AIB-1-C^{14} (overall correlation coefficient $r = +0.62$). Comparison of the concentrations in brain to those in serum (T/S) revealed no significant changes with pressure.

It is evident from these results that a flux of AIB into the brain is dependent upon the serum concentration and is not accelerated by HPO. It might be concluded also that the functional integrity of the blood-brain barrier, at least to AIB, remained unaltered during HPO exposure. Although GABA transport may occur via a different pathway to that of AIB, the present findings with AIB and those previously with GABA[5,6] suggest that blood-brain barrier permeability to GABA is not altered by HPO, and that the protective action of GABA against HPO-induced convulsions[1,4] might not be due to an HPO-accelerated transport of GABA into the brain. In view of the simultaneous decreases in AIB levels in brain tissue and serum, it is evident that HPO accelerated the clearance of AIB from the blood. Changes in functional activity of other organs caused by HPO, such as liver and kidney, might be involved in this accelerated clearance[11].

Résumé. L'étude de l'absorption de l'acide α-aminoisobutyric-C^{14}, par les tissus du cerveau des rats exposés à des pressions variées d'oxygène, a suggéré que l'hyperpression d'oxygène n'influence pas la perméabilité de la barrière hémoencéphalique.

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Brain-serum concentrations and tissue/serum ratio (T/S) of AIB-1-C^{14} during hyperbaric oxygen exposure

Groups	Brain (dpm/g wet wt.)	Serum (dpm/ml)	T/S
Control (13)	7,533 ± 172	30,905 ± 743	0.245 ± 0.007
52 psig (10)	6,856[a] ± 190	26,070[a] ± 496	0.264 ± 0.008
60 psig (9)	6,264[a] ± 167	27,405[a] ± 1,094	0.230 ± 0.010
68 psig (8)	5,652[a] ± 314	27,070[a] ± 922	0.210 ± 0.016
75 psig (14)	6,614[a] ± 266	26,804[a] ± 1,157	0.250 ± 0.012

Number of animals in parenthesis. [a] $P < 0.02$, significant difference from the control group.

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