Cerebral arterial blood volume and blood flow in hypertensive and normotensive rats

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Introduction

In chronic hypertension, the structure of cerebral arterial vessels becomes altered to cope with increased arterial vessel resistance. It produces a narrowed lumen of arterial vessels (1,2), reducing arterial cerebral blood volume (CBV<sub>a</sub>). These alterations in arterial blood vessels are concurrently associated with cerebrovascular diseases, such as stroke, vascular dementia, and cognitive impairment. Since hypertension most alters the arterial side of vessels, noninvasive imaging assessment of CBV<sub>a</sub> is of great clinical relevance for the early diagnosis of hypertension vascular dysfunction. In this study, we applied magnetization transfer (MT)-varied arterial spin labeling (ASL) technique (3) to a well-established hypertensive animal model (spontaneously hypertensive rats, SHR) and a control model (Wistar Kyoto rats, WKY) to assess quantitative CBV<sub>a</sub> and cerebral blood flow (CBF) mapping for regional detection of hypertensive cerebrovascular morphological changes in the baseline condition.

Methods

3 - 5 months old of eight SHR and nine WKY were used. The animals were anesthetized with ~ 1.3 % isoflurane with air supplemented with O<sub>2</sub> to attain a total O<sub>2</sub> level of ~30%. Rectal temperature, blood pressure and blood gases were maintained within normal physiological ranges. Five 2-mm thick coronal slices were acquired on a 9.4 T/31 cm Varian NMR system using the single-shot spin-echo echo planar imaging (EPI) technique with matrix size of 64 (readout) × 32 (phase-encoding) and FOV = 3.0 × 1.5 cm<sup>2</sup>. Two actively detunable RF coils were used; a neck coil provided arterial blood signals; pairs of interleaved ASL and MT pulses were repeated during the spin preparation period (2.75s). TR = 3 s and TE of 18 ms were used. CBV<sub>a</sub> and CBF were determined from the slope and intercept of the linear fit of normalized ASL (ΔS<sub>sat</sub>/S<sub>0</sub>) vs. control (S<sub>sat</sub>/S<sub>0</sub>) values at the two MT levels. Since steady state was not achieved during the relatively short spin preparation period, CBF values were corrected by multiplication with [1-exp(-TR/T<sub>1app</sub>)], where T<sub>1app</sub> (apparent T<sub>1</sub>) = 1.9 s.

Results and Discussion

Quantitative multi-slice maps of CBV<sub>a</sub> (units of ml/100 g) and CBF (units of ml/100 g/min), with T<sub>2</sub>-weighted anatomical images were obtained for SHR (MABP ~140 mmHg) and WKY (MABP ~90 mmHg) rats. Fig.1 shows CBF and CBV<sub>a</sub> maps from a representative animal of each group, and Fig.2 shows regional baseline values for SHR vs. WKY. Quantified CBV<sub>a</sub> values from SHR were generally smaller than those from WKY, while CBF values were less different agree with previous finding (4). Hippocampal values of CBV<sub>a</sub> in SHR are significantly lower than those of SHR (*p < 0.01), indicating that region-dependent hypertension-induced cerebrovascular changes may indeed occur. The smaller CBV<sub>a</sub> of hippocampus in SHR is possibly related to vascular dementia of this model (5). This study shows CBV<sub>a</sub> can be a key indicator to detect regional cerebrovascular impairment in hypertension and follow its progression as hypertension advances.