

DEVELOPMENT OF A METHOD FOR MEASURING THE DYNAMICS OF OXYGEN CONSUMPTION IN THE KIDNEY USING BLOOD OXYGENATION LEVEL-DEPENDENT MAGNETIC RESONANCE IMAGING

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Purpose

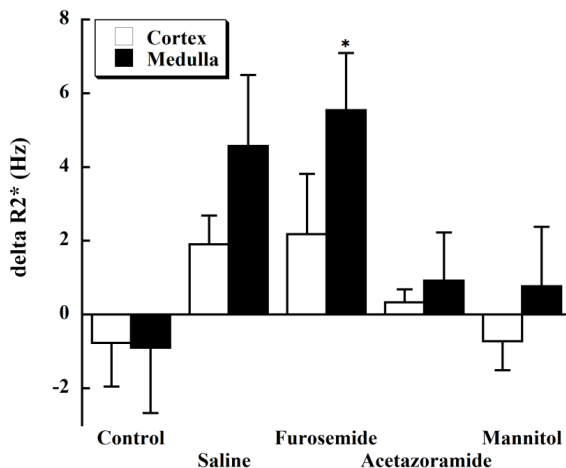
To develop a method for measuring the dynamics of oxygen consumption in the kidney using blood oxygenation level-dependent (BOLD) magnetic resonance imaging (MRI) and to investigate the usefulness and feasibility of this method by measuring the changes in the renal oxygen consumption induced by various diuretics using animal experiments.

Materials and Methods

The BOLD MRI studies were performed in anesthetized Sprague-Dawley rats ($n = 35$) using an MRI system for animal experiments consisting of a 1.5-tesla permanent magnet. To avoid the movement of the kidney during the experiments, we immobilized the rat kidney using a plastic holder of our own manufacture. For BOLD MRI, we used a gradient-echo sequence with a repetition time of 150 ms, echo times (TEs) of 6.5 ms, 15 ms, and 30 ms, a excitation pulse flip angle of 28 degree, a matrix size of 256×128 , a field of view of $60 \text{ mm} \times 30 \text{ mm}$, a number of excitations of 3, a slice number of 4, and slice thickness of 3.2 mm. In addition to the BOLD images, the T_2 -weighted images with the same geometry were acquired before the acquisition of BOLD MRI data as anatomical images. To investigate the effects of diuretics on the oxygen consumption in the kidney, we administered saline, furosemide, acetazolamide, or mannitol as diuretics. We generated the R_2^* maps from the BOLD images with 3 different TEs using the linear least-squares method. The mean \pm standard error of the ΔR_2^* values within the inner compartments of the rat kidney were calculated and compared with those obtained from a control group in which saline or any diuretic was not administered. The ΔR_2^* value was calculated from $\Delta R_2^* = R_{2, \text{before}}^* - R_{2, \text{after}}^*$, where $R_{2, \text{before}}^*$ and $R_{2, \text{after}}^*$ represent the R_2^* value before and after the administration of saline or diuretic, respectively.

Results

Figure 1 shows the changes of R_2^* in the renal cortex and medulla at 24 min after administration of saline or diuretic. For comparison, those in the control group were also shown. As shown in Fig. 1, there was no significant change in R_2^* for approximately 30 min in the control group. When saline, acetazolamide, or mannitol was administered, there were also no significant changes in R_2^* in the cortex and medulla compared to those in the control group. When furosemide was administered, there was a significant change in R_2^* in the medulla compared to that in the control group, supporting the fact that furosemide acts as an inhibitor of the $\text{Na}^+\text{-K}^+\text{-2Cl}^-$ cotransporter existing in the thick ascending limb. When saline was administered, the change of R_2^* in the medulla was greater than that in the cortex.



Conclusion

Our method appears to be useful for measuring the dynamics of oxygen consumption in the kidney. This technique may be useful in the evaluation of therapeutic strategies in animal models of pathophysiological states such as acute renal failure or diabetic nephropathy.

Fig. 1 Changes of R_2^* in the renal cortex and medulla at 24 min after administration of saline or diuretic. For comparison, those in the control group were also shown. Data are represented as mean \pm standard error. * $P < 0.05$ compared to the control group.