# Focal brain ischemia in rat: Early changes in Brain tissue $T_1$ reflects early Increase in Brain Tissue Water Content

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## INTRODUCTION

Brain ischemia is considered to cause first a cytotoxic edema and several hours later a vasogenic edema. Vasogenic edema induces an increase of tissue water content which can be detected via an increase in tissue $T_1$ (1). Neither water content nor tissue $T_1$ increases have been observed in a pure model of cytotoxic edema (1). Recent studies have reported an increase in brain tissue $T_1$ during the first minutes after occlusion of the middle cerebral artery (MCA) (2, 3). As this $T_1$ increase occurs prior to the development of vasogenic edema, several assumptions have been proposed to explain the observed variations.

The aim of this study was to assess whether this tissue $T_1$ increase is related to an increase in water content. For this purpose, this study reports $T_1$ and tissue water content measurements obtained with the gravimetric technique on the same animals, after MCA occlusion.

## MATERIAL AND METHODS

A total of 10 adult male Sprague-Dawley rats (318±15g) were used in this study. Rats were tracheostomized and artificially ventilated under isoflurane. Rectal temperature was maintained at 37.0±0.5C throughout the experiment. Focal brain ischemia was induced by occlusion of the right MCA using the intraluminal suture model. Magnetic resonance imaging was performed at 7T. An inversion recovery FLASH sequence was used to produce $T_1$ maps one and two hours after ischemia onset: 22 inversion times between 20 and 9000ms, recovery time=10s, imaging: TR/TE=5.7/3.2ms, field of view= 40mm, matrix= 64², slice thickness= 2mm, 2 averages. Inversion was obtained with a non-selective adiabatic RF pulse to avoid possible flow contributions to the signal. An apparent diffusion coefficient map (ADC) was acquired 1h40min after occlusion (b= 550 s/mm²). Immediately after animal sacrifice, the specific gravity of the tissue was determined in the layered kerosene-bromobenzene linear column (4).

## RESULTS

For all animals, systemic physiological parameters were within normal limits. Fig. 1 shows ADC and $T_1$ values measured in four regions of interest. In the ipsilateral cortex, $T_1$ was 2062±60ms at ischemia onset +1h (contralateral 1811±28ms), 2100±38ms at ischemia onset +2h (contralateral 1807±18ms), and the water content was 81.1±0.7% at ischemia onset +2h15 (contralateral 79.3±0.5%). The $T_1$ and water content values measured in the contralateral area do not differ from the values obtained in the control animals. A significant $T_1$ increase was observed at ischemia onset +1h (+14%) and ischemia onset +2h (+16%), together with a significant increase in tissue water content (+2.3%). Fig. 2 shows the correlation obtained between $1/T_1$ and 1/tissue water content, in agreement with the linear model described by Fatouros (5).

## CONCLUSIONS

In conclusion, this study reports an increase in tissue water content as early as two hours after ischemia onset, correlated with a tissue $T_1$ change, and concomitant with the well-described decrease in ADC. Tissue $T_1$ measurements performed at one hour after ischemia onset also suggest that the tissue water content has already increased during the first hour of ischemia.

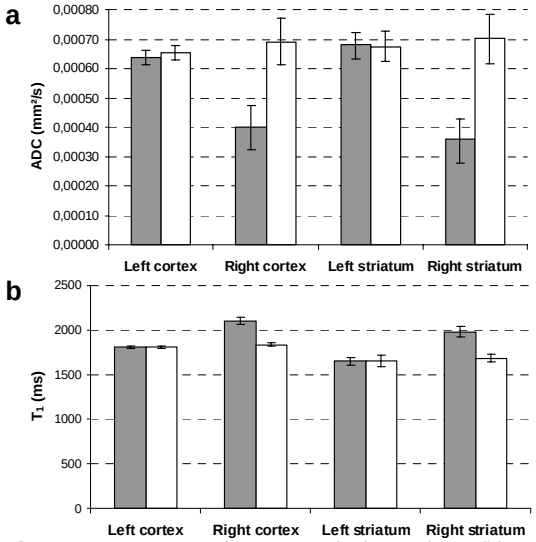


**Fig. 1.** Mean ADC values at occlusion +1h40, (b) mean $T_1$ values at occlusion +2h. Gray bars represent animals with a right MCA occluded, white bars control animals. Error bars represent one standard deviation.

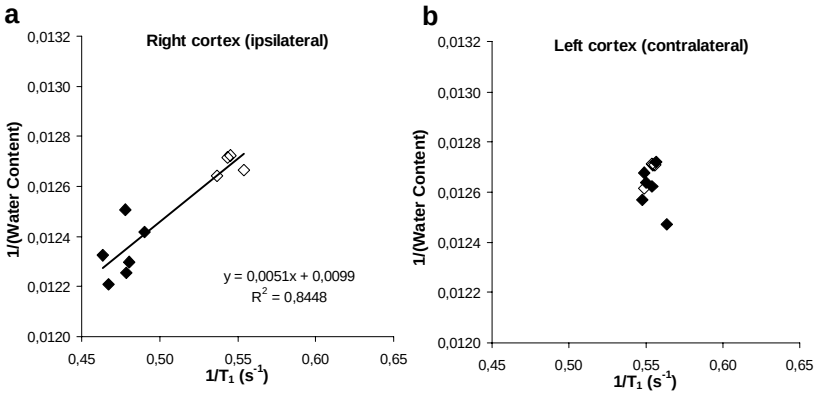


**Fig. 2.** Inverse of tissue water content as a function of $1/T_1$ for (**a**) right cortex, (**b**) left cortex. The equation on (a) describes the linear fit to the data. Black diamonds represent ischemic animals and open diamonds control animals.

## References

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