

# The Relationship Between Perfusion and Diffusion Imaging Parameters in Ischemic Stroke

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**INTRODUCTION** Ischemic stroke occurs when basal cerebral blood flow (CBF) falls below a critical threshold (1-3), resulting in energy failure which subsequently manifests into a reduction in the water apparent diffusion coefficient (ADC) (4). Perfusion- and diffusion-weighted MRI are uniquely sensitive to acute stroke changes and are becoming the method of choice to non-invasively characterize acute ischemic brain injury. However, how the ADC is affected by CBF over time and under different ischemic conditions remains poorly understood. By better understanding relationship between CBF and ADC changes over time, it may be possible to identify potentially salvageable regions and ultimately to determine the fate of tissues subject to ischemic injury.

In this study, ADC and CBF in rat models following permanent and transient (30-min, 60-min and 90-min) middle cerebral artery occlusion (MCAO) were studied to examine the relationship between ADC and CBF at different time after stroke and under different occlusion durations.

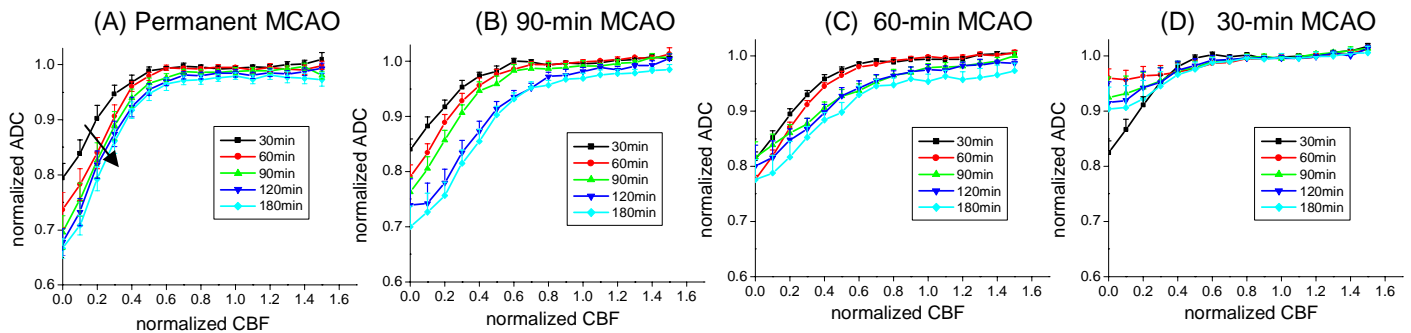
**METHODS** Thirty-six male SD rats (300~350g) were subjected to permanent (n = 12), 30-min (n = 12), 60-min (n = 6) or 90-min (n = 6) intraluminal middle cerebral artery occlusion (5). MRI was performed on a Bruker 4.7-T/40-cm (Billerica, MA) scanner. Imaging was performed every thirty minutes and up to 3 hrs post-occlusion. ADC maps were obtained by averaging three ADC data set acquired with diffusion-sensitive gradients applied along the x, y or z direction. Data were acquired using spin-echo diffusion weighted EPI, TR = 2 s, TE = 37 ms, 64 x 64 matrix, 2.56 x 2.56 cm FOV, eight 1.5 mm slices, b = 10, 1270 s/mm<sup>2</sup>. Quantitative CBF was measured using the continuous arterial spin-labeling technique (6) with gradient-echo EPI and parameters similar to the ADC measurement except TE = 14 ms.

ADC and CBF were normalized with respect to the normal non-ischemic hemisphere. Averaged ADC values of tissues in ischemic hemisphere were plotted at five different time points against 15 CBF bins (bin size= 0.1, from 0 ~1.5).

**RESULTS** For the permanent MCAO (**Figure A**), when CBF was above 50% of its normal value (normal: 1.1±0.2 ml/gram/min), ADC was not affected by changes in CBF and does not significantly deviate from normal (> 95%). Below this threshold, ADC decreased linearly and monotonically with decreasing CBF. ADC values decreased monotonically over time after stroke as expected in permanent MCAO. CBF critical thresholds, which cause ADC decrease, did not change significantly over time (< 5%) and remained at about 50% of normal value. For the 30-min MCAO group (**Figure D**), ADC values transiently returned to close to normal after reperfusion and decreased again over time, suggesting although some tissues were salvaged, there was delayed cell injury. For the 60-min MCAO group (**Figure C**), ADC values only increased slightly after reperfusion. In contrast, for the 90-min MCAO group (**Figure B**), ADC values did not increase, similar to permanent occlusion.

**DISCUSSION** Our study showed that ADC is not affected until CBF drops below a critical value (~50% of normal value). Below this critical threshold value of CBF, the functioning of cells is compromised, as manifested as reduced ADC and ADC decreases linearly and monotonically with decreasing CBF. Our work suggests that sufficiently early treatment of occluded blood flow could lessen or reverse the negative effects of ischemic stroke. The observation of ADC's decrease after reperfusion in 90-min group implies that 90 minutes is already passed treatment window at this rat stroke model.

**CONCLUSIONS** This study demonstrates an alternative and helpful means to analyze the relationship between perfusion and diffusion parameters. The results demonstrated that critical CBF below which ADC decreases is complex, depending on ischemic durations and time after stroke.



**REFERENCES** 1) Astrup et al. Stroke 1981, 12:726. 2) Hossmann K-A. Ann Neurol 1994, 36:557. 3) Lo et al. Stroke 2005, 36:189. 4) Moseley et al. Magn Reson Med 1990, 14:330. 5) Shen et al. JCBFM, 2004, 24:280. 6) Shen et al. JCBFM, 2003, 23:1479.