

Changes over time of brain perfusion and cerebral vasoreactivity after stroke: preliminary results

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Target audience:

Physicians and physicists interested in recovery after stroke and BOLD fMRI.

Purpose

Depicting cortical reorganization after stroke using BOLD fMRI remains challenging¹. Indeed, long lasting changes of brain basal perfusion and cerebral vasoreactivity have been reported². However, changes over time of these parameters remain unclear³. Because of the biophysical background of BOLD fMRI, these pathological changes may impair the neurovascular coupling, as previously shown in vicinity of stroke² and brain tumors⁴. These changes should be taken into account in order to adjust locally the hemodynamic response to neuronal stimuli. Moreover, functional recovery of the lesioned area after stroke could rely on the restoration of vascular properties⁵. Thus, it is critical to better characterize basal and functional vascular disorders to adjust and estimate therapeutic management⁶.

The aim of this study was to investigate changes over time of cerebral perfusion and CO₂ vasoreactivity (CVR) in patients after stroke using MRI.

Methods

We prospectively examined 16 subjects referred for stroke in the middle cerebral artery (MCA) territory using MRI (Philips Achieva 3T TX) at 3 time points: 35, 50, and 180 days after stroke onset. Beside anatomical images, we measured basal perfusion using dynamic susceptibility contrast during first pass Gd-DTPA bolus (T2*-EG WI : TR/TE=1067/40ms; vox=2x2x4mm; 15 slices; 60 dyn.).

We also measured CVR to a hypercapnic challenge (CO₂ 8%/O₂ 21%/N₂ 71%) using a high concentration face mask and a block-designed paradigm [3 x [air (1') - CO₂ (2') - air (1')], with BOLD contrast (T2*-EG WI: TR/TE=3000/35ms; vox=4x4x4mm; 32 slices; 240 dyn.).

All images were realigned, coregistered, and spatially normalized in MNI space using SPM8. Basal perfusion was computed using deconvolution of the arterial input function to provide common parameters (CBF, CBV, MTT, Tmax). CVR was estimated using GLM approach (SPM8) with the simultaneous end-tidal CO₂ (EtCO₂) time course as a physiological regressor of the BOLD signal changes⁷. Data analyses were conducted on Regions of interest (ROIs), drawn onto both T1- and T2-WI to identify 1°) necrotic core, 2°) perinecrotic rim, and 3°) undamaged MCA territory. Homologous ROIs were mirrored in the healthy hemisphere. We computed laterality indices (LI) to assess interhemispheric differences across parameters with LI = [(lesioned ROI value - healthy ROI value)/(lesioned ROI value + healthy ROI value)]. ANOVAs were conducted using SPSS. Significance was set for p<0.05.

Results

No adverse reaction was detected, including during the hypercapnic challenge.

Perfusion and CVR were decreased in all ROIs in the lesioned hemisphere. Significant changes were detected between 30 and 180 days after stroke (Figures 1 and 2).

In both necrotic core and perinecrotic rim, CBF and CBV decreased whereas MTT increased. In the undamaged MCA territory, perfusion remained stable over time (Figure 1).

CVR increased in the perinecrotic rim but remained stable in the necrosis and the undamaged MCA territory (Figure 2).

Discussion

Our results are in line with previous studies showing important alterations of basal perfusion and CVR in the lesioned hemisphere after stroke, even out of the infarct^{2,8}.

The decrease of basal perfusion between 30 and 180 days after stroke shows that perfusion impairment may worsen over weeks after stroke. However, the improvement of CVR may reveal a prior restoration of CVR after stroke to drive / or follow neural rehabilitation⁶. This also could be a passive consequence of the basal perfusion decrease, suggesting a negative mechanical correlation (i.e. initially reduced vessels caliber would have a broader response when elicited). Furthermore, these changes may affect the hemodynamic response to neuronal stimuli (e.g. neurovascular coupling), and should be taken into account as confounding factor in BOLD fMRI².

Conclusion

These preliminary results showed important basal perfusion and CVR alterations in the lesioned hemisphere. While perfusion decreased over 180 days after stroke, CVR increased in the perinecrotic rim suggesting functional improvement of the perilesional vasculature. Further data are expected at 2 years after stroke in a larger number of patients.

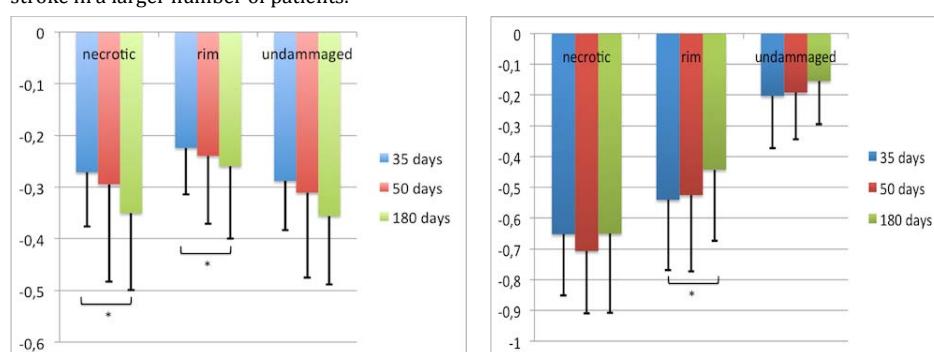


Fig. 1

Decrease of CBF in both necrotic core and perinecrotic rim between 35 and 180 days after stroke.

* p < 0.05

Fig. 2

Increase of CVR in the perinecrotic rim between 35 and 180 days after stroke.

* p < 0.05

References

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