Use of the Talairach proportional grid system for ROI quantification of Cerebral Blood Volume maps of the brain

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Introduction

Functional neuroimaging has proved to be of great help in the early detection of subjects with possible or probable Alzheimer disease (AD). Structural and functional alterations, including cerebral blood volume, in specific areas of temporal, parietal and frontal cortex, and a relative sparing of primary motor and sensitive regions, have been observed in AD patients. For quantitative analysis of brain CBV maps, the main methodological problem is the low resolution of the images, which makes it difficult the anatomical localization of brain structures. In this study we propose a new method for regional quantification of brain CBV maps using the anatomical information of structural MRI images and the Talairach proportional grid system.

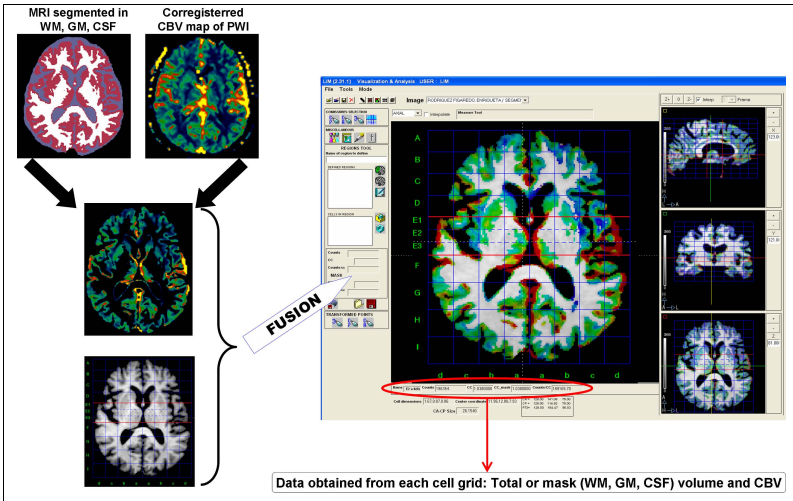
Method

The image protocol included a volumetric T1 weighted 3D gradient echo (voxel size 1 x 1 x 1.5 mm. Parametric coloured maps of PWI images were obtained from DSC images using EPI sequence after the injection of a bolus of gadolinium chelate. The method proposed is a multimodal application where the anatomical information of the T1W MRI is used to build the Talairach grid and a co-registered CBV image is superimposed on the same grid (Figure, left). By doing so, the Talairach-normalized tessellation of the brain is directly extended to CBV images, allowing for a convenient regional analysis of volume and blood volume rates of brain structures, defined in the Talairach Atlas as sets of cells. This procedure requires minimal manipulation of brain geometry, thus fully preserving individual brain morphology, which is important in neurodegenerative diseases that may cause brain atrophy.

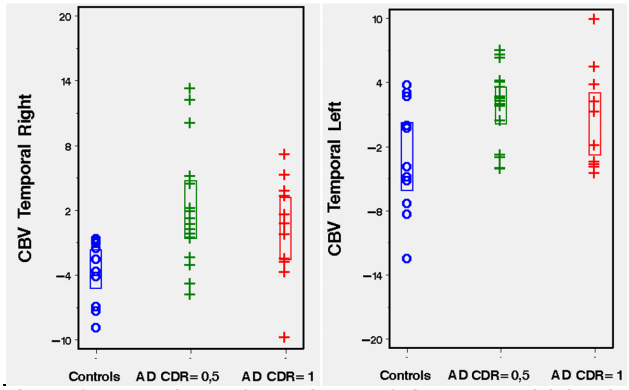
This method allowed us to obtain volume and CBV data for the whole brain and for the frontal, parietal, occipital and temporal lobes, for each hemisphere. To avoid the confounding effect of overall brain atrophy, ROI CBV values were obtained per unit of brain GM tissue (in cc) and proportionally normalized to the whole brain value of CBV for each subject. To illustrate the potential of the method, we performed a comparison of CBV maps in a sample of 17 mild (Clinical Dementia Rating (CDR) score =1.0) and 11 very mild CDR=0.5) cognitive impaired Alzheimer patients and 12 controls, aged 50 to 85.

Results

Using an ANCOVA model that included total brain volume and age as covariates, we observed significant differences in the CBV of GM tissue in the temporal lobe when comparing the three groups (Figure right). A significant reduction of the CBV values was observed between the AD group with CDR=0,5 and those with CDR=1,0 (n=11) in the temporal lobes (right: $F_{2, 36} = 4.6$; p=0.0168; left: $F_{2, 36} = 3.8$; p=0.0310). Control subjects show significantly lower values than both patient groups. This pattern of increased hyperperfusion of CBV values in AD patients has also been observed in other brain areas known to be altered in AD, like the hippocampus (Alsop et al. 2008). These findings support the claim of compensatory elevation of neural activity, inflammation or elevated production of vasodilators in early stages of the disease (Nagata et al. 2000).



Overview of the method and a screen capture showing the interface of the software tool developed to implement the method.



Plots of CBV values of GM tissue of the temporal lobe, in controls and AD patients with mild (CDR=1) and very mild (CDR=0,5) cognitive impairement. Data points represent residuals for total volume and age from the model used in the ANCOVA Box shows 2 STD around the mean.

Conclusions

- Perfusion Weighted MR imaging to estimate CBV maps is a promising alternative to nuclear medicine for functional evaluation of patients with dementia.
- The Talairach grid system enables regional quantification of CBV maps.
- The results showed a regional hypoperfusion in the left temporal lobes in AD patients with mild cognitive impairment compared with those with very mild cognitive impairment. Overall, AD patients show hyperferfusion relative to controls, possibly as a compensatory mechanism of the brain at an early stage of the disease.

References

Nagata, K., Kondoh, Y., Atchison, R., Sato, et al., 2000. Neurobiol Aging 21, 301-307.
Alsop, D.C., Casement, M., de Bazelaire, et al., 2008. Neuroimage 42, 1267-1274.