Combined Vessel Size and Blood Flow Imaging with Hyperoxia

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Introduction

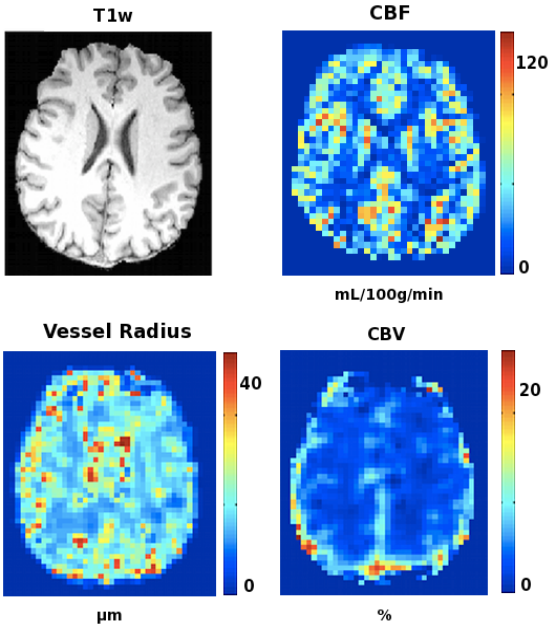
MRI studies are able to provide a wide range of physiological information, including quantitative measures of perfusion and vessel size. The combination of such information may help to elucidate the complex physiological interdependence of these measures and how they evolve post stroke[1], helping to tease apart contributions from neovascularization, vasodilatation of existing vessels and collateral flow. Recent MRI studies have shown the feasibility of performing vessel size imaging (VSI) without the use of intravenous paramagnetic contrast agents. To accomplish this, either carbogen[2] or oxygen[3] can be used to induce changes in blood oxygenation. Here we present an imaging protocol that is able to provide simultaneous estimates of mean vessel radius, resting cerebral blood flow (CBF) and non-arterial cerebral blood volume (CBV). The technique uses a hyperoxia paradigm with a multi-TI pseudo-continuous ASL (pCASL) sequence, modified to include a gradient echo and spin echo EPI readout.

Theory

VSI is based on the vessel size dependence of gradient echo (GE) and spin echo (SE) acquisitions[4]. Changes in tissue susceptibility during breathing challenges result in differential changes in GE and SE signals from which estimates of mean vessel radius can be made using appropriate biophysical models. Periods of mild hyperoxia may be used to modulate the BOLD signal, while producing minimal changes in CBF[5]. The paradigm allows for simultaneous acquisition of perfusion and vessel size dependent data, while the GE data[5] or a combination of GE and SE data[2] may be used to estimate CBV.

Methods

4 healthy volunteers were scanned on a 3 Tesla Siemens Verio with a 32-channel head coil, using a pCASL sequence with a dual GE-SE EPI readout (TR = 4.5s, TE=22/81ms). In order to improve the BOLD contrast the pCASL sequence did not include any background saturation of brain tissue. 16 axial slices were acquired in ascending order (4x4x4.5mm voxels, 0.9mm inter-slice gap). The labelling duration was 1.4s with 5 post labelling delay times (0.2, 0.4, 0.6, 0.8 and 1.0s). The imaging paradigm consisted of one 27-minute acquisition, comprised of 4x3minute hyperoxic periods interleaved with 5x3minute periods of normal air. During periods of hyperoxia, 100% oxygen was delivered to the volunteers via a nasal cannula. The flow rate of oxygen was adjusted to deliver an inspired oxygen fraction of 0.5. Resting CBF images were calculated by fitting the GE data to the ASL kinetic model[6]. BOLD weighted GE and SE images were produced by averaging the tag and control images. GE and SE images were pre-processed with FSL. The resulting images were converted to ΔR_2^* and ΔR_2, where $\Delta R2^* = -\log(S_{GE}(t)/S_{0,GE})/TE_{GE}$ and $\Delta R2 = -\log(S_{SE}(t)/S_{0,SE})/TE_{SE}$. S(t) is the time course of the magnitude GE or SE data and S_0 is the average value during baseline. A linear regression was used to calculate the ratio of $\Delta R2$ and $\Delta R2^*$. The ratio maps were transferred to maps of mean vessel radius via a polynomial fit to Monte-Carlo model data[2], evaluated at 3T. Maps of $\Delta R2^*$ and vessel radius were then used to calculate CBV[2].



Results

The figure shows example parameter maps calculated from an individual subject, with a structural image included for comparison. The group averaged gray matter (GM) values are given in the table below.

	CBF (mL/100g/min)	Vessel Radius (µm)	CBV (%)
Mean ± s.d.	50.56 ± 5.33	16.20 ± 0.91	4.06 ± 1.46

Mean gray matter values measured in four volunteers

Discussion and Conclusions

The protocol presented here provides a means of measuring a range of clinically relevant parameters from a single acquisition. The technique uses oxygen to modulate the BOLD signal and therefore should be well tolerated. The group average GM values agree well with previously published results[2,5,7].

References

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