Simultaneous imaging of cerebral perfusion and glucose metabolism by PET/MRI

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Introduction The concept of combining positron emission tomography (PET) with magnetic resonance imaging (MRI) was initially conceived to provide the high soft-tissue contrast of MRI with the functional information of PET. The potential of this hybrid approach extends beyond this concept considering the variety of MRI methods for assessing function, diffusion, and metabolite concentrations (1). In the neurosciences, PET/MRI provides the ability to study the relationship between cerebral perfusion, as measured by arterial spin labelling (ASL), and cerebral energy metabolism or neurotransmitter systems. Applications of PET/MRI could include investigating potential perfusion/metabolism mismatches associated with cerebrovascular disease and studying how alterations in neurotransmitters affect brain function. The objective of this study is to present preliminary data demonstrating simultaneous acquisition of ASL and PET images, which were acquired as part of an on-going feasibility study involving fluorine-18 fluoro-deoxyglucose (FDG) imaging.

Materials and Methods Data were acquired on a Siemens Biograph mMR PET/MR scanner. The PET system consisted of 8 rings of 56 detector blocks, each housing 8x8 lutetium oxyorthosilicate crystals coupled to avalanche photodiodes. The rings are located between the gradient and RF coils of a 3 T magnet. The scanner includes a whole-body gradient coil with a maximum strength of 45 mT/m and a slew rate of 200 T/m/s. All MR images were acquired using a product 16-channel receive-only head/neck RF coil. Simultaneous PET/MRI data were acquired from four patents: three with a history of cancer undergoing whole-body FDG examinations and one with frontotemporal dementia. The brain scan (12 min) was conducted 2.5 hours after injecting 182-555MBq of FDG. During this time, TI-weighted MPRAGE images were acquired (1 mm isotropic resolution), followed by pseudo-continuous (pCASL) imaging (1.5 s labelling duration, 24 cm FOV, 64x64 matrix, 16 axial slices 7.5 imaging duration) (2).

The FDG data were attenuation corrected using an ultra-short TE sequence and standared uptake value (SUV) generated by iterative reconstruction for a resolution of 1.2 x 1.2 x 2 mm. Cerebral blood flow (CBF) image were generated by pairwise subtraction using ASLtbx (3). The SUV and CBF images were normalized to the standard brain using SPM8, and a regression analysis conducted pixel by pixel to investigate the relationship between CBV and SUV.

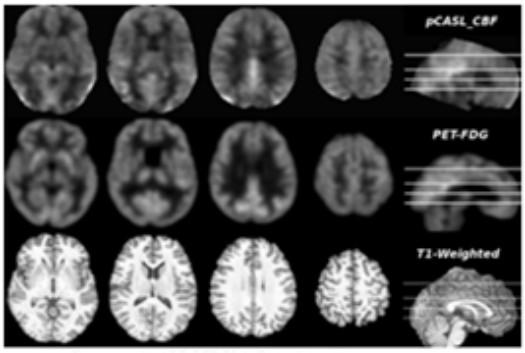

Fig.1: CBF, FDG and anatomical images of 4 slices from one patient

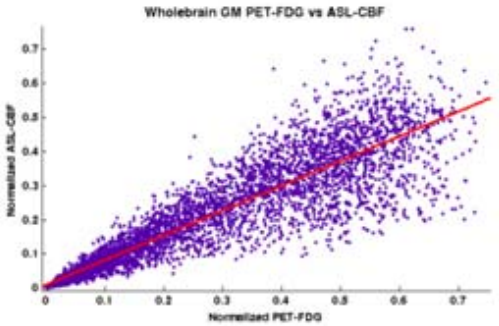

Fig.2: Correlation between CBF and FDG uptake across all patients (R^2 = 0.85).

Results Fig. 1 displays ASL-CBF, FDG-SUV and T1-weighted images for four slices from one whole-brain data sets. The overall correlation between CBF and SUV across all 4 patients is shown in Fig. 2. However, a perfusion-metabolism mismatch in the frontal lobe was observed in the dementia patient, as well as a 27% decrease in whole-brain CBF. The functional images from one cancer patient exhibited hypo metabolism/perfusion due to a pre-existing traumatic brain injury (TBI) (Fig. 3).

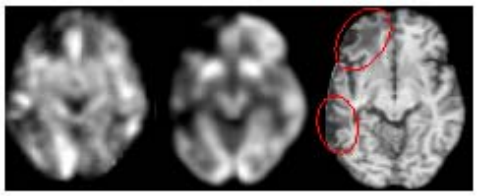

Fig.3: ASL, FDG and anatomical imaging from patient with TBI(injury sites indicated by the cirdes).

Discussion The PET/MRI RF coil provided good spatial coverage; however, a 50% reduction in the temporal signal-to-noise ratio was found compared to data acquired with a 32-channel coil (4). This reduction was offset by the use of pCASL, and the SNR was sufficient to reliability map regional CBF. The strong correlation between regional CBF and FDG uptake was expected considering the tight coupling of CBF to energy demands (5). This agreement extended to the injured brain regions in the TBI patient (Fig. 3). Hypometabolism was observed in the frontotemporal dementia patient, but there was no concurrent hypoperfusion seen in the ASL images. This mismatch is similar to frontal lobe hypometabolism reported with Alzheimer's patients (6,7).

Reference [1] Pichler, *J Nucl Med* 2010;51:333-336. [2] Wang, *Proc ISMRM*, 2007;15:2974. [3] [3] Wang Z, et al., *JMRI*, 2008;26:261-9. [4] Owen, *Pain* 2008;136:85-96. [5] Siesjo Brain Energy Metabolism 1978. [6] Musieket *Alzheimers Dement.* 2012;8:51-59. [7] Chen *Neurology* 2011;77:1977-1985.