Vascular territory mapping using ASL measurements of arterial transit time

M. F. Lythgoe[1], D. L. Thomas[2], D. G. Gadian[1]

[1]RCS Unit of Biophysics, Institute of Child Health, London, United Kingdom, [2]Department of Medical Physics and Bioengineering, University College London, London, United Kingdom

Introduction

The ability to identify the regional distribution of vascular supply in the brain may provide an additional measure of cerebral haemodynamics for the assessment of cerebrovascular disease. Currently the two MRI techniques that can provide various measures of cerebral perfusion are dynamic susceptibility contrast imaging and arterial spin labelling (ASL). Although neither of the methods can provide an assessment of the intracerebral contributions to vascular distribution, recently Davies et al. have selectively labelled the major feeding vessels to the brain (1). The aim of this study was to acquire ASL measurements of arterial transit time to estimate vascular territory maps (VTM) in the rat brain.

Methods

Sprague Dawley rats (n=6) (200-260g) were anaesthetised with 2.5% halothane and maintained on 1.25% halothane with 70/30% nitrous oxide/oxygen. Middle cerebral artery occlusion (MCAO) was performed using a 4/0 suture inserted in the CCA . **Pre-occlusion:** Coronal SE-EPI images were obtained on a 2.35T horizontal bore SMIS MR scanner approximately 0.5 mm from bregma with FOV 40x20 mm, a 2 mm slice, and 128 × 64 pixels. CASL: interleaved adiabatic inversion labelling and control measurements, 10 different post-labelling delay times from 50 to 2000 ms (2), and 88 averages. T_1: IR-EPI with 8 TI, ranging from 284ms to 3734ms. CBF and transit time: All data were fitted on a pixel-by-pixel basis in IDL. T_1 and M_0 are fitted using the IR data; subsequently these values are used to fit the magnetisation difference to the equation in Alsop *et al.* (1) for CBF and arterial transit time. Assumptions: T_{1a} = 1.5 s, λ = 0.9, efficiency of the spin labelling pulse = 0.9 (previous results; data not shown), tissue transit time = 1 s. **Post-occlusion:** CBF maps were calculated using a single post-labelling delay time (500ms) and 22 averages. ADC maps were calculated from trace-weighted single shot spin-echo EPI images with b=38 and b=1181 s/mm^2. ROIs: 3 ROIs used in figure 2: the motor cortex, somatosensory cortex and caudate putamen, which approximately correspond to the middle cerebral artery (MCA), anterior cerebral artery (ACA) and lenticulostriate (LSt) vascular territories.

Fig 1

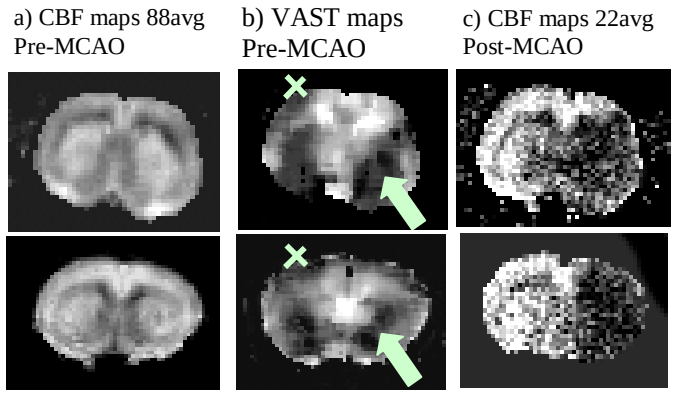
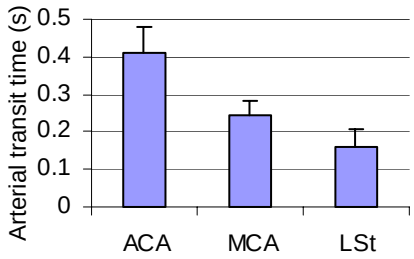


Fig 2. Arterial transit times from 3 regions in control rats (n=6)



Results and discussion

Figure 1 demonstrates the distribution of CBF values pre-occlusion (a) and the associated decrease in CBF in the regions supplied by the middle cerebral artery (MCA) and lenticulostriate artery (LSt) following occlusion (c). Transit time maps calculated pre-occlusion (b) indicated a shorter transit time in the area supplied by the LSt (filled arrow) in all animals (Fig 1b), but differentiation of the MCA and anterior cerebral artery (ACA) border was not always possible (Fig 1b, Rat B, cross) and was only clearly defined in one rat (Fig 1b, Rat A, cross). However, transit times averaged over selected ROIs demonstrated significant differences between the motor cortex (ACA), somatosensory cortex (MCA) and caudate putamen (LSt) (Figure 2). These three regions are, in general, supplied by the MCA, ACA and LSt (3,4). Some rats had larger areas of diffusion/perfusion mismatch, possibly reflecting larger regions of collateral flow, which may account for why there was not a discrete MCA/ACA border in some rats (Rat B, Fig 1b). Transit time measurements in humans have previously indicated an anterior to posterior graduation, which was in agreement with geometry of the blood supply (5). As far as we are aware, this is the first attempt to use ASL transit time measurements to identify specific intracerebral vascular territories in the rat brain. Future work aims to clarify the contribution of collateral supply to the VTM.

(1) Davies, N. (2003) MRM, 49:1133-1142
(2) Alsop DC, Detre JA. (1996) J. Cereb. Blood Flow Metab. 16, 1236-1249
(3) Ginsburg MD, Busto R. (1989) Stroke, 20, 1627-1642
(4) McAuley MA. (1995) Cerbrovasc and Brain Metab Rev
(5) Figueiredo P et al. (2002) Proc ISMRM, 623