

Comparison of CBF deconvolution techniques using bolus tracking in the presence of delayed tracer arrival in acute stroke patients

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Introduction

Perfusion-weighted MRI (PWI) has been shown to be a sensitive but not specific indicator for tissue at risk of infarction [1]. Moreover, the prevalent method, singular value decomposition (SVD), used to calculate CBF, CBV and mean transit time maps (MTT) has been shown to be sensitive to delay between the arterial input function and the tissue concentration curve [2]. It has been speculated that with flow estimates less contaminated by tracer arrival time differences, assessment of salvageable tissue may be improved. This study compares CBF values in acute stroke patients calculated using SVD and using a delay-insensitive technique to determine if decoupling delay from flow estimates is clinically beneficial.

Patients and Methods:

Patients with large-vessel atherothrombotic (n=8) and cardioembolic strokes (n=18) imaged on a 1.5 T clinical scanner (General Electric) with both diffusion-weighted MRI (DWI) and PWI <12 hr from symptom onset were retrospectively analyzed. Median time to scan from symptom onset was 5.1 h. Patients were selected from a non-consecutive cohort of patients admitted between 1996 and 2003 who were not treated with thrombolysis and who received follow-up imaging (F/U) at least 5 days after initial event. Apparent diffusion coefficient (ADC) maps were calculated from the DWI. Dynamic susceptibility-weighted contrast-enhanced images were acquired during the first pass of a bolus of 0.2 mmol/kg of gadolinium-based contrast agent. CBF and MTT were calculated by deconvolution of tissue concentration curves with an arterial input function selected from the ipsilateral hemisphere using standard SVD [3] (sCBF and sMTT) and using SVD with a block-circulant matrix (cSVD) and minimization of oscillation index [4] (cCBF and cMTT). Tracer arrival differences (Delay maps) were estimated as the time of the residue function peak in the cSVD [4].

All imaging studies were coregistered using an automated linear registration tool (FLIRT) [5]. Infarct volumes were outlined on the F/U. The ischemic core was operationally defined as the initial DWI abnormality (Core), and perfusion lesion as an area of increased delay on the Delay maps. All images except MTT and Delay maps were normalized with respect to normal contralateral gray matter. Relative MTT and Delay maps were calculated by subtracting mean values in normal contralateral gray matter. Regions with delayed flow that did not infarct (Non-infarct) were defined as the difference between perfusion lesion and F/U lesion. Regions that were normal on the initial DWI and abnormal on F/U was considered recruited infarct volume (New-infarct). Mean values were calculated and compared (paired Student t-tests) in patients exhibiting large mismatches between DWI and PWI (>10 cm³).

Results

Out of the 26 patients, 19 exhibited large mismatches. Fig 1 shows an example of acute DWI and PWI lesion mismatch along with corresponding sCBF and cCBF images for a patient imaged 5.1 h after symptom onset. A much larger PWI lesion is evident (arrows) than becomes infarcted as seen on F/U. Table 1 shows mean±SD of relative values measured in the different regions for all 19 patients. sCBF and cCBF were significantly lower in Core than in New-infarct (p=.004 and p<.001 respectively) and in Non-infarct (p=.002 and p<.001 respectively) tissue regions. No significant difference was found in sCBF values between New-infarct and Non-infarct regions (p=.3) while a significant difference was found in cCBF values (p=.04). For sMTT, no significant difference was found between the three regions (p>.05). In contrast, cMTT was significantly greater in core than in New-infarct (p=.005) and Non-infarct (p=.02) tissue regions.

Discussion

sCBF was found to be uniformly reduced and sMTT uniformly increased in areas showing delayed tracer arrival not originally infarcted. However, using a delay-insensitive technique, cCBF was found significantly higher in tissue that did not infarct compared to tissue that became infarcted. These results suggest that the lack of specificity of existing CBF estimates in predicting infarct volume [6] are potentially due to the presence of delayed tracer arrival. Using delay-insensitive CBF estimation techniques may therefore provide greater insight into tissue salvageability in acute stroke.

References

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Fig 1. Example acute DWI and PWI lesion mismatch. Much larger area of tissue appears hypoperfused in sCBF (arrows) than cCBF. This region does not infarct according to F/U imaging.

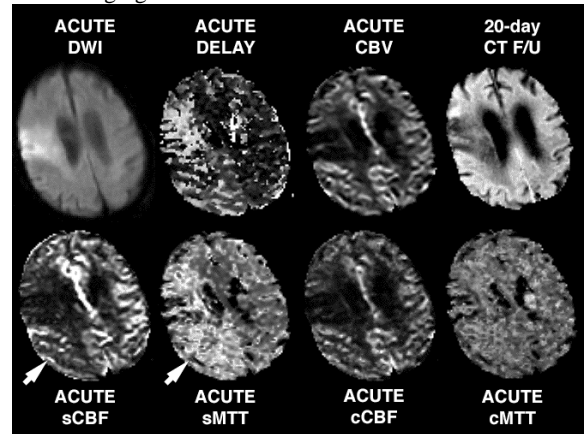


Table 1: Mean relative sCBF and cCBF

	Core	New-infarct	Non-infarct
sCBF	.43±.24	.54±.18	.57±.17
cCBF	.46±.21	.64±.17	.71±.16
sMTT	2.6±1.6	2.4±1.7	2.8±1.4
cMTT	2.9±2.0	2.1±1.8	2.0±1.2