An Investigation of Cerebral Perfusion in Aphasia Using Arterial Spin Labeling

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Introduction

- Magnetic Resonance Perfusion Imaging is a neuroradiological technique that can directly measure regional cerebral blood flow (rCBF)
- Previous research has indicated reduced rCBF, or hypoperfusion, among stroke survivors (e.g. Hillis et al., 2001; Fridriksson et al., 2002)
- Hypoperfusion in chronic stroke survivors may correlate more strongly with cognitive and behavioral deficits than standard anatomical neuroimaging (Love et al., 2002)

Questions

- What is the nature of hypoperfusion among stroke survivors, as compared to age-matched and young unimpaired controls?
- What is the time course of rCBF among stroke survivors?

Methods

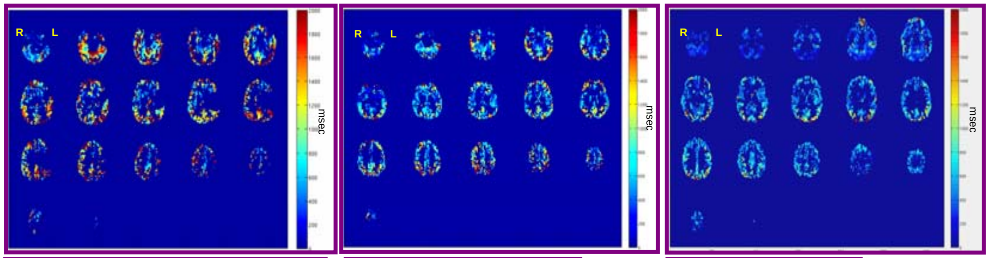
- Images were acquired on a GE Signa EXCITE 3Tesla scanner using an ASL FAIR sequence (scan parameters: TR=2500ms, 16 slices (5mm skip 1 mm), FOV=22) and an 8 gradient head coil
- A Pulsed Arterial Spin Labeling (PASL) Procedure was used; PASL uses radiofrequency pulses to magnetically tag arterial blood water
- Images were acquired following a post-labeling delay (transit delay) during which the tagged blood water is delivered to cerebral areas
- During the patient's first scan, perfusion data were acquired across 8 transit delays (ranging from 300msec to 3 sec) to measure both the time course and localization of rCBF
- Perfusion data were analyzed to find an optimal transit delay TI2 for the stroke patient and a second ASL scan was performed, using the optimal TI time

Participants

- Participants were screened for a negative history of drug or alcohol abuse; all participants were right-handed
- Stroke survivor (45 years old, 5 years post-stroke): damage to left anterior cortical hemisphere; subsequent aphasia; single unilateral stroke, no history of other neurological impairment
- Age-matched unimpaired control, 39 years old
- Younger unimpaired control, 23 years old

Results

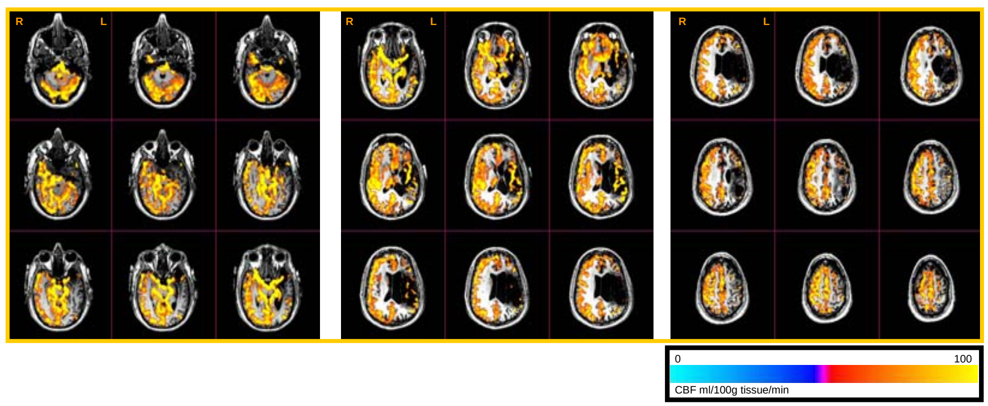
1st Scan: Transit Delay ASL Sequence: Perfusion data acquired across 8 T2 transit times, from 300 msec to 3 sec, to determine optimal T2 transit delay for stroke survivor



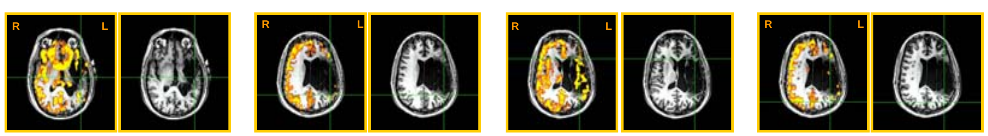
Stroke Survivor, 45 years, 5 years post-stroke | Age-Matched Control, 39 years | Young Control, 23 years

• Transit Delays: Stroke survivor exhibits longer transit delay than age-matched and younger controls: This indicates a slower time course of cerebral perfusion in the stroke survivor

2nd Scan: Optimal Transit Delay ASL Scan for Stroke Survivor: TI1 = 600 msec, TI2 = 2300 msec



Cerebral perfusion within language-related cortical areas



Left Superior Temporal Gyrus | Left Supramarginal Gyrus | Left Inferior Frontal Gyrus | Left Angular Gyrus

**Crosshairs indicate noted areas of interest

Discussion and Conclusions

- The first ASL scan was performed to determine an optimal arterial transit delay for the stroke survivor; this patient exhibited longer arterial blood flow transit times between the tagging region and the imaging regions, as compared to the age-matched and younger controls
- The second ASL scan used a personalized optimal T2 arterial transit time for the stroke patient, and hypoperfusion was still evident, especially in peri-lesional areas. We posit two possible explanations for this finding:
  - A longer T2 transit time may be needed to capture the full extent of cerebral perfusion in people with chronic cerebrovascular abnormalities who have slowed and reduced regional CBF
  - Certain cerebral regions may be chronically hypoperfused and functionally underactive among chronic stroke survivors
- Compromised cerebral perfusion was particularly evident in several cortical regions that have been implicated in various components of language processing: the left superior temporal gyrus, the left supramarginal gyrus, the left inferior frontal gyrus, and the left angular gyrus.
- The finding of increased T2 transit time for the arterial bolus indicates that chronic stroke and cerebrovascular disease may alter the time course of cerebral perfusion
- Future research will examine the correlations between cerebral hypoperfusion, the time course of CBF, and behavioral and cognitive deficits in stroke survivors with aphasia

References

Hillis et al., Ann Neurol 2001; 50: 561-566.
Fridriksson et al., Aphasiology 2002; 16: 859-871.
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Carusone et al., Am J Neuroradiol 2002; 23: 1222-1228.

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