Accordance of ASL delay time and bolus arrival times in parenchyma

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Subject: ASL has become a widely applied tool to non-invasively measure cerebral perfusion. The blood entering the brain through the carotid - and the vertebral arteries is magnetically labelled in the tagging block during a given time period. For technical reasons there has to be a gap between the tagging block and the readout planes (inversion pulse artifacts). Furthermore, the blood needs several seconds to travel the distance between the tagging block to the cortical capillaries (parenchymal phase) where exchange with the tissue takes place [1]. To account for this, the readout begins after a specified delay. However, T1 relaxation causes a fast decay of signal from the labelled blood that limits the time-window of sufficient SNR for ASL acquisition to a few seconds [2]. In patients with steno-occlusive arterial disease (SOAD) above the labeling plane (intracranial stenosis) this time window might be too narrow due to delayed bolus arrival times (BAT) to the parenchyma.. Moreover, differences in BAT would cause signal acquisition from blood in distinct vascular compartments, thus complicating interpretation of ASL CBF maps.

The **aim** of the current ongoing investigation is to estimate to what extent BAT from labelling to the parenchymal phase of cerebral perfusion are delayed in patients with SOAD and from which vascular compartment the ASL signal origins at a given delay time.

Methods: Digital subtraction angiography (DSA; Siemens Axiom Artis zee, Siemens, Erlangen, Germany)) was performed in 10 patients with SOAD and the BAT from labeling block to different vascular compartments was defined for the healthy and the stenotic hemisphere by inspection of the DSA images (GS, MH, FK, ME, KJ). In addition a simulation of T1 decay was computed based on our standard ASL imaging parameters (pseudocontinous ASL[3]; TR 4000ms, label time 1.72s, post labelling delay 1.5 s, blood-brain-partition coefficient 0.9; T1 relaxation 3T: 1490ms) using Bloch's equation [4].
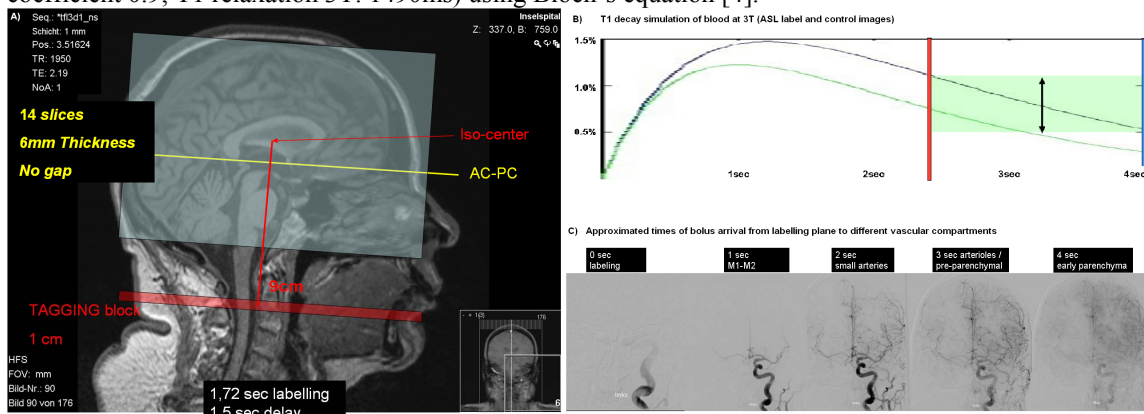

Figure: A) ASL recording setup and parameters. **B)** T1 decay simulation for blood at 3T in % of signal change (control and label images): ASL signal reduction of 50% between acquisitions at 2.48 sec (red bar, current) compared to 4.0sec (blue bar, parenchyma phase) after labelling. **C)** Approximated bolus transit times from tagging plane to different vascular compartments demonstrated by DSA in a view following contrast injection into the left common carotid artery.. Note the collateral flow to the contralateral hemisphere due to occlusion of the right internal carotid artery with a time delay of about 0.3 sec only..

Results: Average BAT from labelling plane to parenchyma was 3.3s±1.0s (mean ± standard deviation) on the healthy hemisphere and 3.8s±2.1s on the hemisphere, supplied by a upstream stenotic artery. Thus, no significant prolongation of transit times can be observed in the group mean (paired t-test p=0.31); however, there are substantial inter-individual differences. Standard ASL acquisition uses labelling and delay times of 1.6-1.72sec and 1-1.5sec, respectively. In this time-window a reasonable SNR can be achieved with 120 images as estimated by the simulation of T1 signal decay for the specified imaging parameters and flow velocities of 90ml/100g/min. However, in this time-window the ASL signal origins from small arteries and not the parenchyma. Prolonging the delay to match the BAT to the parenchyma (4s) reduces the SNR by 50%.

Conclusions: The ASL perfusion values seem to be acquired at a time point where the labelled blood is still in small arteries or arterioles but not yet in the parenchyma. Upstream stenoses from the labelling plane seem not to significantly prolong the BAT, thus a direct comparison of CBF values calculated from healthy and stenotic hemisphere is plausible. However, inter-indiviudal differences have to be considered when interpreting CBF maps from ASL especially in patient populations where blood flow is altered.

References:

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