

A Kinetic Model for Vessel-Encoded Dynamic Angiography with Arterial Spin Labeling

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Introduction: Artery-selective angiography provides vital information in a variety of cerebrovascular diseases, such as the source and extent of collateral blood flow, which can maintain vital brain tissues when the primary feeding artery is compromised. Conventional x-ray digital subtraction angiography (DSA) is invasive and carries risks to the patient [1]. Recently, we proposed a non-invasive, non-contrast MRI-based method [2] for artery-selective time-resolved angiography using vessel-encoded pseudocontinuous arterial spin labeling (VEPCASL) [3] which gives qualitative artery-specific information about blood flow patterns, vessel morphology and hemodynamics. However, image interpretation may be biased by signal attenuation from T1 decay and imaging RF pulses and only outflow of the labeled bolus is visualized, unlike the more intuitive inflow images achieved with DSA. In this work we develop a kinetic model to describe the signal in VEPCASL dynamic angiography [4], accurately accounting for bolus dispersion and signal attenuation. This model is applicable to other techniques based on continuous or pseudocontinuous ASL. The fitted model parameters are used to: i) synthesize more intuitive inflow images unbiased by signal attenuation, and ii) calculate relative blood volume flow rates in downstream vessels from each feeding artery. These methods are applied in healthy volunteers and a patient with Moyamoya disease.

Theory: Unlike conventional ASL, the signal in VEPCASL angiography is proportional to blood volume, rather than blood flow [4]. The "ideal" signal profile in a single voxel is a rect (box-car) function characterized by blood arrival time δ and duration equal to that of the VEPCASL pulse train, τ . Bolus dispersion can be modelled by convolution of the ideal signal profile with a dispersion kernel, $D(t_d)$, describing the fraction of blood delayed by time t_d due to dispersion. We choose a gamma variate function for $D(t_d)$, characterised by sharpness, s , and time to peak, p [5]. The signal is attenuated by T1 decay and imaging RF pulses, described by factors $T(\delta, t_d)$ and $R(t, \delta, t_d)$ respectively. These can be written as $T(\delta, t_d) = \exp(-(\delta + t_d)/T_{1b})$, where T_{1b} is the T1 of arterial blood, and $R(t, \delta, t_d) = \cos^N \alpha$ where α is the RF excitation flip angle and $N(t, t_d, \delta_{min})$ is the previous number of RF pulses experienced by the blood. N depends on the time at which imaging commences, t_0 , and the earliest arrival of blood from the artery of interest into the imaging region, δ_{min} . Making a continuous approximation for N gives: $N \approx \min\{(t - t_0)/T_R, (\delta - \delta_{min} + t_d)/T_R\}$ where T_R is the imaging pulse repetition time. Putting this together yields the final model:

$$S(t) = A \int_{t-\delta}^{t-\delta+\tau} dt_d D(t_d) T(\delta, t_d) R(t, \delta, t_d) \quad (1)$$

where A is a scaling factor proportional to blood volume. For well-mixed downstream vessels the relative contribution to the blood volume flow rate from each feeding artery is equal to the relative blood volume within that artery which can be calculated simply by summing A over the vessel segment of interest.

Methods: Six healthy volunteers and one patient with Moyamoya disease were recruited and scanned on a 3T Siemens scanner under protocols approved by local ethics and institutional committees. VEPCASL dynamic angiography was performed in transverse and coronal views centered on the circle of Willis (sequence parameters as per [2]). Artery specific images were produced using a maximum *a posteriori* (MAP) Bayesian inference method [6,7]. Estimation of δ_{min} for each feeding artery was performed by fitting Eq. 1 to the large proximal arteries to estimate the earliest blood arrival. The five-parameter fit (A, δ, s, p, σ), including the noise standard deviation, σ , was performed at each voxel within a vessel mask for each feeding artery using a MAP Bayesian method with Gaussian priors on each parameter. Inflow visualization was achieved by using the fitted parameter values to calculate the expected signal at a range of time points during blood inflow with $\tau \rightarrow \infty, T_{1b} \rightarrow \infty$ and $\alpha = 0$.

Results: The kinetic model fitted the data well in all subjects and produced parameter maps with the expected patterns in healthy volunteers: high blood volume (A) in large proximal vessels, and later blood arrival (δ) and greater dispersion (s, p) in distal vessels. Fig. 1 shows parameter maps and example model fits in the patient with Moyamoya disease. Delayed blood arrival in collateral vessels and post-stenosis in the right middle cerebral artery are evident. Fig. 2 shows an example of synthesized inflow visualization in a healthy volunteer with non-standard flow patterns. Relative flow rate calculations indicate that the left posterior cerebral artery (PCA) obtains 94% of its blood supply from the left internal carotid artery and the remaining 6% from the right vertebral artery in this subject. This is unlike the majority (4 of 6) of the healthy volunteers scanned for this study where the PCAs are fed almost exclusively by the two vertebral arteries. In the figures color is used to represent the origin of the blood signal (red = right internal carotid, green = left internal carotid, blue = right vertebral, magenta = left vertebral).

Discussion: The kinetic model described here accurately fits the data acquired thus far. The resulting parameter maps show delayed blood transit and increased dispersion (not shown) distal to diseased vessels in the patient with Moyamoya disease, suggesting that they may prove useful as biomarkers of disease. Inflow images give an intuitive visualization of the data without biases from T1 decay and RF effects which may appeal to clinicians familiar with x-ray DSA. Relative flow rate calculations show the importance of each feeding artery to the blood supply in downstream vessels, which is likely to be of use in the assessment of collateral flow for therapeutic planning.

References:

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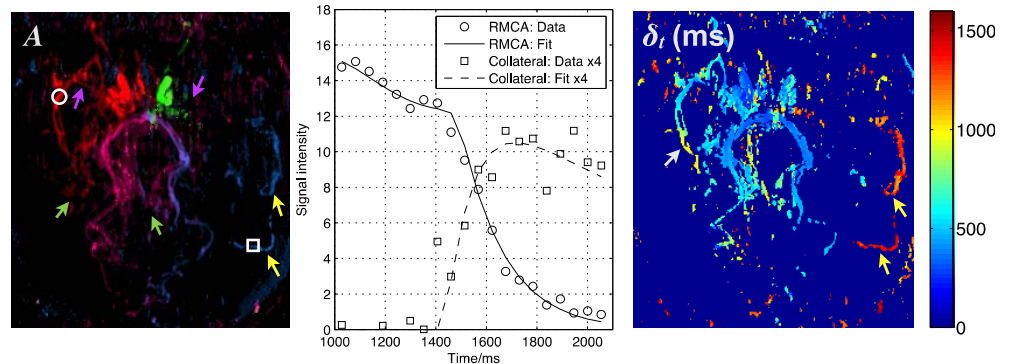


Figure 1: Modelling fitting results in a patient with Moyamoya disease. The color coded map of parameter A (left), which relates to blood volume, demonstrates severe stenoses (purple arrows), extensive small collateral vessel formation (green arrows), and a larger collateral vessel (yellow arrows). Delayed transit to the collateral vessel and right middle cerebral artery (white arrow) are evident in the map of δ (right). Example model fits in two voxels (RMCA and large collateral vessel) are also shown (middle), corresponding to those highlighted in the map of A .

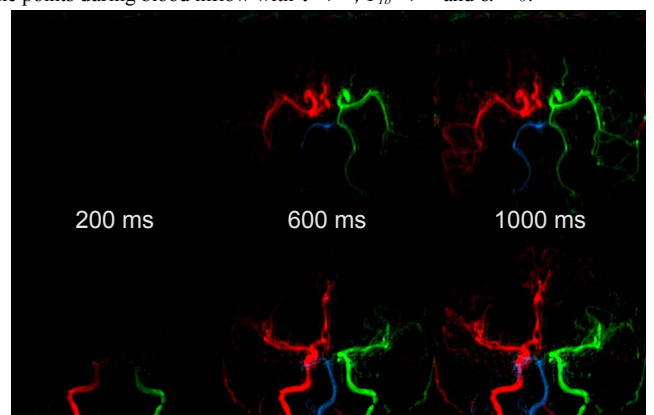


Figure 2: Example time frames from inflow visualization in a healthy volunteer with non-standard flow patterns in transverse (top) and coronal (bottom) views.

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