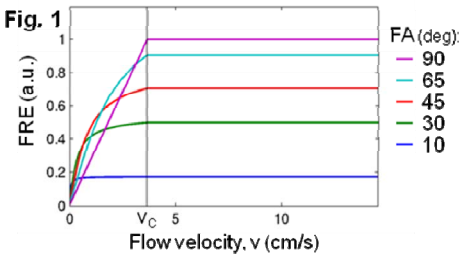
Novel MRI indicators of cerebrovascular compliance in response to cardiac pressure wave

Marta Bianciardi[1], Nicola Toschi[1,2], Jonathan R Polimeni[1], Himanshu Bhat[3], Bruce R Rosen[1], David A Boas[1], and Lawrence L Wald[1]

[1]Department of Radiology, A.A. Martinos Center for Biomedical Imaging, MGH, Harvard Medical School, Boston, MA, United States, [2]Department of Medicine, University of Rome "Tor Vergata", Rome, Italy, [3]Siemens Medical Solutions, Boston, MA, United States

Target Audience: Clinicians and researchers interested in novel MRI indicators of cerebrovascular compliance.

Introduction: Cerebrovascular disease is the second leading cause of mortality worldwide. Cerebrovascular disease risk is not fully explained by currently observable risk factors such as arterial compliance (i.e. the change in blood volume due to cardiac pressure changes) in systemic and in cerebral circulation [1]. Cerebrovascular compliance in the carotid and larger intracranial arteries is currently estimated indirectly through Doppler sonography and MRI measurements of blood flow velocity changes (e.g. pulsatility and resistivity index). In order to meet the need for novel cerebrovascular disease risk factors, the central aim of this research project is to design and validate novel, non-invasive MRI indicators which estimate cerebrovascular compliance through direct measures of blood volume changes in a highly localized and subject-specific manner.



Theory: We employed time-of-flight (TOF) echo-planar-imaging (EPI) MRI, and constrained the sequence parameters to produce an MRI contrast dependent primarily on cerebral blood volume (CBV) changes. In TOF MRI [2], flow related enhancement (FRE) inside the cerebral vessel is weighted by the velocity (v) of flowing spins only for v below or equal to the critical speed v_C (v_C is equal to the ratio of slice thickness - ST - to repetition time - TR). Spins flowing at a velocity above v_C produce the same FRE as spins flowing at v_C. (For a calculation of FRE(v(t)) [2] see Fig. 1 - parameters employed: TR = 33 ms, T_1 = 2.6s; slice thickness ST = 1.2 mm). We therefore hypothesized that by choosing a critical speed (~3.6 cm/s, vertical line in Fig. 1) lower than the blood velocity in large and middle cerebral vessels (where v=10-120 cm/s during the cardiac cycle), the MRI signal change in these vessels over time (S(t)) due to cardiac pressure changes would only depend on blood volume changes since the FRE is constant irrespective of blood velocity changes (here we also assume no changes in oxygen saturation during the cardiac cycle):

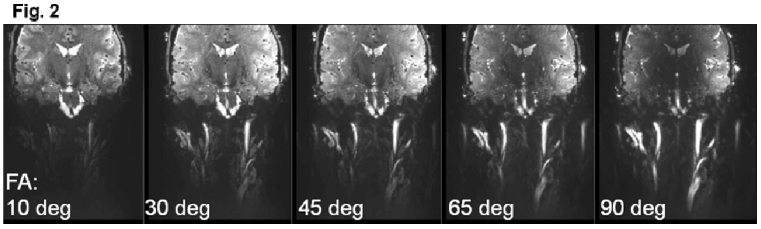
$$S(t)=CBV(t)\cdot FRE\cdot \exp^{-TE/T2^*} \quad \text{Eq. 1}$$

We then defined a pulsatility Volume Index (pVI) as the signal during systole divided by the signal during diastole, either voxel-wise or integrated over a region of interest (e.g. a vessel segment or a vessel cross section):

$$pVI = \int_{ROI} S(t_{systole})/S(t_{diastole})\, dr^3 \quad \text{Eq. 2}$$

Methods: Four subjects (1m/3f, age 24± 2) participated in this IRB-approved study. Gradient echo EPI was performed at 7 Tesla using a 32 receive-only RF array and the following parameters: TR/TE/flip angle (FA) = 33 ms/18 ms/90°, slice orientation: coronal, in plane voxel-size = 1.2 × 1.2 mm^2, ST = 1.2 mm, N_{scans} = 625, GRAPPA factor = 5, variable number of slices (up to 40 slices, TA/slice = 20 s; ~ 6 to cover the carotid arteries). EPI allowed to speed up the acquisition by a factor of ~ 5 as compared to CINE acquisitions. By employing a FA of 90° instead of the Ernst angle (~10°), we were able to obtain higher FRE for moving spins (see Fig. 1, v > 0 cm/s) as well as improved suppression of stationary spins (see Fig. 1, v = 0 cm/s). As validation, we repeatedly acquired the same slice while varying FA ([10 30 45 65 90]°). Note also, that we were able to employ a coronal slice orientation, because for high flow velocity significant in-plane flow attenuation occurs after spins have traveled in-plane for several cm. Cardiac pulsation was recorded by a piezoelectric finger pulse sensor (1 kHz sampling rate) and used for detecting the timing of cardiac peaks. EPI time-courses were band-pass filtered (0.9-1.4 Hz). For each voxel, and for each cardiac peak, we considered a window of 1 cardiac cycle (~1 s) of the EPI time-course, and then averaged this window across peaks to obtain an average cardiac EPI waveform (the number of consecutive averaged peaks, Nav varied between 15 and 20 across subjects and slices). From each average EPI waveform and each voxel, we obtained voxel-wise estimates of $S(t_{systole})$ and $S(t_{diastole})$ as the maximum and minimum signal of the average EPI waveform, respectively. We then computed the integral of $S(t_{systole})/S(t_{diastole})$ on a voxel-by-voxel basis (pVI_{VOXEL}), across a vessel cross-section ($pVI_{CROSS\text{-}SECTION}$) or across an entire segmented vessel ($pVI_{SEGMENT}$).

Fig. 2



Results & Discussion: Fig. 2 shows an EPI image acquired with several FAs (1 time-point), demonstrating a ~10-fold gain in contrast of flowing versus stationary spins for FA = 90° compared to FA = 10°, in agreement with the predicted FRE (see Fig. 1). Fig. 2 also shows the feasibility of mapping in-plane flow in long segments of the carotid arteries (coronal acquisition). Fig. 3 shows pVI_{VOXEL} for an example data-set (MIP across six slices). Interestingly, pVI_{VOXEL} was lower for voxels inside or containing the arterial lumen (note a darker stripe inside the arteries, red arrows), and higher for voxels covering the arterial wall, as expected for a blood volume effect. The computed $pVI_{CROSS\text{-}SECTION}$ (for an example data-set) and $pVI_{SEGMENT}$ (mean ± s.e. across subjects) for the carotid arteries are shown in Figs. 4 and 5, respectively. pVI varied across the arterial axis (Fig. 4), between arteries (*, p <0.02, Fig. 5), and displayed a trend of left/right asymmetry. Extraction of pVI indices was feasible also for other arteries (e.g. MCA, ACA, basilar artery, scalp arteries) and in the sagittal sinus, which displayed marked pulsatility (results not shown).

Fig. 3 pVI_{VOXEL}

Fig. 4 $pVI_{CROSS\text{-}SECTION}$

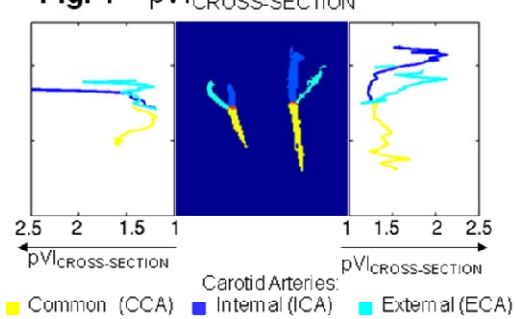
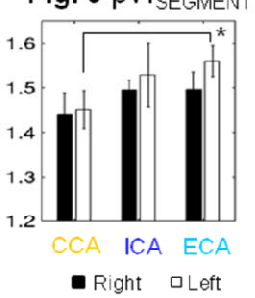


Fig. 5 $pVI_{SEGMENT}$



Conclusion: pVI seems a promising MRI indicator, which can potentially measure cerebrovascular compliance in a highly localized and subject-specific manner. We envisage whole brain coverage in < 15 minutes through the use of slice acceleration EPI techniques, as well as using of pVI to assess the compliance also of smaller vessels.

References: [1] Laurent and Boutouyrie, *CNS Drugs*,19:1-11, 2005. [2] Haacke et al, MRI, Chap. 24, John Wiley & Sons, 1999.