Carotid pulse wave velocity quantification with non-gated, velocity-encoded projections

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INTRODUCTION: Pulse wave velocity (PWV) is a measure of arterial stiffness equal to the propagation velocity of the arterial flow waveform along a segment of the arterial tree. Elevated aortic PWV has been shown to correlate with aging, but is also independently associated with cardiovascular disease, including atherosclerosis, and all-cause mortality,[1] making PWV an important marker of vascular health. In contrast to the aorta, PWV in the iliofemoral arteries has been found to decrease with age.[2] These findings imply that segmental PWV can distinguish between different normal physiologic and pathologic processes, and suggest the utility of PWV measurement in different arterial locations. Carotid artery PWV (cPWV) is of particular interest due to the significant cardiovascular and neurovascular morbidity arising from carotid artery disease and the need for better non-invasive markers of carotid disease progression. However, existing MRI-based PWV techniques have insufficient temporal resolution to resolve pulse arrival times over the short distances spanned by smaller arteries such as the carotid. Here we present a method for resolving flow waveforms at two user-defined slice locations with 5 ms temporal resolution, permitting quantification of cPWV in seconds, non-invasively, and without gating or breath-hold.

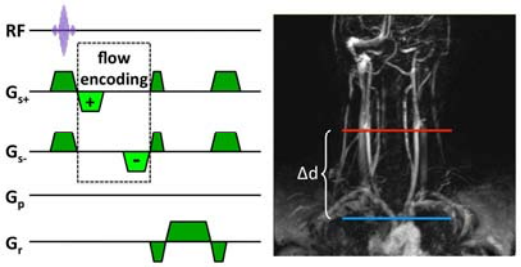

Figure 1: (left) cPWV pulse sequence diagram illustrates the phase-modulated sinc pulse used for dual-slice excitation and acquisition of velocity-encoded projections; **(right)** Coronal MIP illustrating corresponding slice locations in the carotid artery.

METHODS: The proposed method is a modification of the velocity-encoded projection technique previously used to measure iliofemoral PWV.[2]

Pulse Sequence – A phase-modulated sinc pulse is used to simultaneously excite two slices at the inferior and superior portion of the carotid artery with the bandwidth of the carrier wave determining the slice separation distance (Fig. 1). Non-phase encoded projections with toggled velocity encoding and 5 ms TR are acquired continuously for 10 s. Care is taken to ensure that there is no vessel overlap in the projection direction within each slice, and use of body array, spine, and neck coils enables separation of signal from the lower slice (body and spine coils) and upper slice (neck coil). Sequence parameters are flip angle = 20°, VENC = 100 m/s, TE/TR = 3.2/5.0 ms, dwell time = 10 μs, $FOV_{Readout}$ = 150 mm, and number of readout points = 100.

In Vivo Experiments – Three healthy males (ages: 25, 27, & 41 y.o.) were scanned at 3T (Siemens, Erlanger, Germany). For each, cPWV was measured over four spans: from the lower common carotid artery (CCA) on the left or lower brachiocephalic artery (BCA) on the right, to both the upper CCA and internal carotid artery (ICA).

Data Analysis – Taking the complex difference between adjacent pairs of velocity-encoded projections yields temporally resolved flow waveforms, acquired simultaneously at each slice location. An in-house-written ChainLink (© Magland, 2008) analysis tool is used to automatically quantify the temporal separation of the waveform pair at the foot, which is averaged over all waveform pairs in the 10 s acquisition window to determine the arrival separation time (Δt). The vessel path length (Δd) is determined from the same axial stack used for slice planning. cPWV is then quantified as Δd/Δt.

RESULTS: Figure 2 shows a characteristic dataset. Upper (2a) and lower (2b) reference images, complex difference projection data (2c), and velocity waveforms (2d) are shown. The temporal separation between the lower and upper slice waveforms is used to calculate Δt. Table 1 shows cPWV for each subject and segment. Mean (SD) cPWV was 6.2 (1.9) m/s, and mean (SD) path length was 12.4 (2.2) cm.

Table 1. cPWV (m/s) for each segment & subject.

	Subject 1	Subject 2	Subject 3
Left CCA→CCA	5.9	5.8	11.5
Left CCA→ICA	5.3	5.0	4.6
Right BCA→CCA	5.4	7.1	7.2
Right BCA→ICA	5.0	6.6	4.9

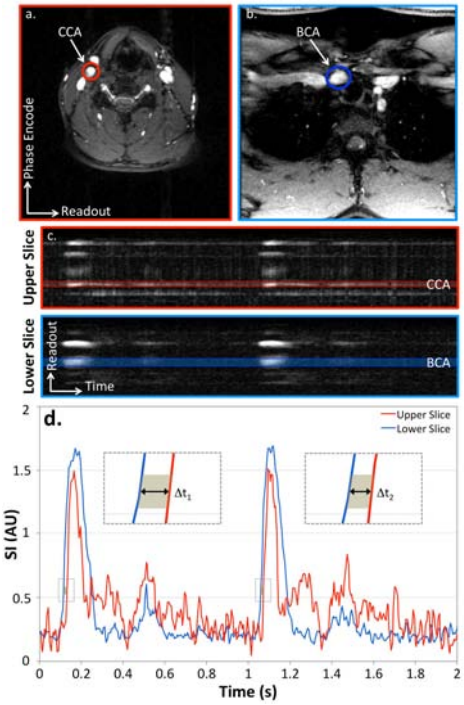

Figure 2: Magnitude images corresponding to the upper **(a)** and lower **(b)** slices with CCA and BCA indicated; **(c)** Complex difference between velocity-encoded projections in both the upper and lower slices; **(d)** Average signal from ROIs shown in upper and lower slices is used to calculate the waveform separation, Δt.

DISCUSSION: Measured cPWV values are similar to those reported for aortic PWV,[3] and agree with literature reported values for cPWV.[4,5] Subject 3's left CCA to CCA cPWV is quite high, likely a result of insufficient slice separation causing the neck coil to acquire data from both the upper and lower slices, contaminating the waveform with signal from the lower slice vessels. Interestingly, cPWV decreases when using the ICA rather than CCA for the upper slice, suggesting a slowing of the waveform as it progresses up the arterial tree. However, it is necessary to scan additional subjects to determine whether this effect is real.

CONCLUSIONS: We have demonstrated feasibility of an MR-method for quantification of cPWV based on velocity-encoded projections. Proper coil placement is essential for separation of signal from the two slice locations and presents a challenge to this otherwise rapid, robust, and reproducible technique. This limitation is being addressed through investigation of different coils/placement as well as post-processing methods to deconvolve the signal from the two slices based on measured coil sensitivity profiles.[6] Given the scarcity of cPWV data in the literature, current studies are focused on applying the technique in healthy individuals to establish cPWV values over a range of ages, determining intra-subject variability and method reproducibility, as well as investigating the relationship between cPWV and aortic PWV. As there is a significant association between aortic PWV and cardiovascular morbidity, we are eager to investigate the potential relationship between cPWV and cardiovascular/neurovascular disease progression, and to evaluate the utility of cPWV as an early marker of carotid artery disease.

REFERENCES: [1] Vlachopoulos, et al., *JACC* 55(13):1318-27 (2010); [2] Langham, et al., *JCMR* 13:81 (2011); [3] Langham, et al., MRM 65:750-5 (2011); [4] Dizaji, et al., J Teh U Heart Ctr: 91-96 (2009); [5] Hardy, et al., *Proc. ISMRM* (2008); [6] Larkman, et al, *JMRI* 13:313-17 (2001).

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