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Assessing intracranial vascular compliance using dynamic arterial spin labeling

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ABSTRACT

Vascular compliance (VC) is an important marker for a number of cardiovascular diseases and dementia, which is typically assessed in the central and peripheral arteries indirectly by quantifying pulse wave velocity (PWV), and/or pulse pressure waveform. To date, very few methods are available for the quantification of intracranial VC. In the present study, a novel MRI technique for in-vivo assessment of intracranial VC was introduced, where dynamic arterial spin labeling (ASL) scans were synchronized with the systolic and diastolic phases of the cardiac cycle. VC is defined as the ratio of change in arterial cerebral blood volume (ΔCBV) and change in arterial pressure (ΔBP). Intracranial VC was assessed in different vascular components using the proposed dynamic ASL method. Our results show that VC mainly occurs in large arteries, and gradually decreases in small arteries and arterioles. The comparison of intracranial VC between young and elderly subjects shows that aging is accompanied by a reduction of intracranial VC, in good agreement with the literature. Furthermore, a positive association between intracranial VC and cerebral perfusion measured using pseudo-continuous ASL with 3D GRASE MRI was observed independent of aging effects, suggesting loss of VC is associated with a decline in perfusion. Finally, a significant positive correlation between intracranial and central (aortic arch) VC was observed using an ungated phase-contrast 1D projection PWV technique. The proposed dynamic ASL method offers a promising approach for assessing intracranial VC in a range of cardiovascular diseases and dementia.

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Introduction

Vascular compliance (VC) represents the ability of a vessel to distend or increase volume in response to an increase in blood pressure (Kelly and Chowienczyk, 2002). This buffering mechanism plays an essential role in the protection of the vascular bed by converting the pulsatile flow in arteries to continuous flow into the capillaries. Aging is accompanied by loss of elastin and increased collagen deposition in vessel walls, which result in the loss of aortic compliance (Lee and Oh, 2010; Mohiaddin et al., 1993; Van Bortel and Spek, 1998). Pathologic changes in the blood vessels can also lead to loss of vascular compliance. The reduction of VC has been regarded as a potentially important marker of a number of diseases with high social and economic impact, such as cardio- and cerebrovascular diseases, hypertension, diabetes, and Alzheimer's disease (AD). For instance, studies have reported that both central VC and peripheral VC provide a sensitive marker for essential hypertension (Laurent et al., 2003; McVeigh et al., 1991). A positive association between arterial stiffness and atherosclerosis was found at various sites along the vascular tree (van Popele et al., 2001). It was also reported that the reduction of VC of both large and small arteries occurs at an early time point in patients with diabetes (Romney and Lewanczuk, 2001). Studies have shown that decrease in aortic VC is an independent predictor of cardiovascular events and Alzheimer's disease (Blacher et al., 1999; Laurent et al., 2001; Meaume et al., 2001a; Sutton-Tyrrell et al., 2005). Therefore, the assessment of VC is useful for a number of clinical indications.

In clinical practice, vascular compliance can be indirectly estimated by the measurement of brachial pulse pressure (i.e. systolic–diastolic blood pressure) (Mackenzie et al., 2002). Applanation tonometry (Nelson et al., 2010; Wilkinson et al., 1998) is a noninvasive and accurate representation of the aortic pressure waveform, which can be

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used to estimate arterial stiffness with the measurement of central pressures and augmentation index. Pulse wave velocity (PWV) has emerged as another commonly used method to assess VC, which can be measured by Doppler ultrasound (Sutton-Tyrrell et al., 2005) and MRI (Boese et al., 2000; Laffon et al., 2005; Langham et al., 2011a; Mohiaddin et al., 1993; Wentland et al., 2014). The flow pulse waveforms at two artery sites (such as carotid and femoral arteries) are measured. PWV is then calculated by dividing the path length of the pressure wave between the two sites of arteries by the propagation time of the pulse wave. PWV is considered the best surrogate marker of arterial stiffness which is inversely related to VC. However, PWV quantification is limited to large arterial segments such as the carotid arteries, aortic arch or iliofemoral arteries (Boese et al., 2000; Langham et al., 2011a,b; Mohiaddin, 1992; Mohiaddin et al., 1993; Sutton-Tyrrell et al., 2005; Tanaka et al., 2009).

Mounting evidence suggests that intracranial vascular pathology may be associated with the pathogenesis and progression of cerebrovascular disorders and neurodegenerative diseases. Particularly, in Alzheimer's disease, it has been hypothesized that cerebrovascular dysfunctions may precede and cause amyloid accumulation in the vessel walls as well as impaired clearance of amyloid-β peptide 42 across the blood–brain barrier (BBB) (Hughes et al., 2013; Zlokovic, 2011). Therefore, intracranial VC may offer an important marker for vascular pathology of earliest stages of cerebrovascular diseases and AD. To date, however, very few options exist for non-invasive estimation of intracranial VC. Transcranial Doppler ultrasound (TCD) has been applied for assessing cerebral VC through measuring pulsatile blood flow velocities in large cerebral arteries (e.g., middle cerebral artery (MCA)) (Zhang et al., 2009). However, TCD is operator dependent, and has limited capability for assessing changes in arterial geometry and cerebral blood volume. Furthermore, the MCA may not be accessible to TCD in a considerable fraction of the population.

Arterial spin labeling (ASL) is a noninvasive MRI technique for quantitative measurement of cerebral blood flow (CBF) by magnetically labeling the upstream arterial blood water as an endogenous tracer (Detre et al., 2009; Golay et al., 2004). Recently, a novel non-invasive MRI technique for in-vivo estimation of arterial cerebral blood volume (CBV) has been introduced using dynamic ASL by combining ASL with a cine segmented multi-phase balanced steady-state free precession (bSSFP) sequence (Yan et al., 2012). According to the definition of compliance, VC can be calculated by the change of blood volume due to a given change of blood pressure. In the present study, we propose a novel technique for in vivo assessment of intracranial VC, where dynamic ASL scans were synchronized with the systolic and diastolic phases of the cardiac cycle respectively, and VC can be estimated by changes in arterial CBV (ΔBV) in response to changes in arterial pressure (ΔBP). We evaluated the feasibility of this dynamic ASL technique for assessing intracranial VC in two vascular components of large arteries, small arteries and arterioles. The aging effect on VC and the relationships between intracranial VC with cerebral perfusion as well as aortic PWV were subsequently explored as an initial validation of the proposed technique.

Methods

Assessment of intracranial vascular compliance

A dynamic ASL technique has been developed recently for in-vivo estimation of intracranial arterial CBV, by combining ASL with a cine multi-phase bSSFP sequence which was originally developed for cine cardiac imaging. Technical details of this dynamic ASL technique can be found in Yan et al. (2012). This sequence takes advantage of the phenomenon that the longitudinal magnetization of flowing blood is not or only marginally disturbed by the bSSFP pulse train, given the high contrast of blood signal and inherent flow compensation of the bSSFP sequence (Wu et al., 2010; Yan et al., 2012). Once the blood exchanges into tissue, it becomes quickly saturated by the bSSFP readout as the T2/T1 ratio is much lower in the tissue than in the arterial blood at 3 T. Therefore, the labeled blood behaves like an intravascular contrast agent in the dynamic ASL scan, and arterial CBV can be quantified using the standard tracer kinetic model (Ostergaard et al., 1996) similar to dynamic susceptibility contrast MRI.

Fig. 1 shows the diagram of the proposed dynamic ASL technique. By synchronizing ASL with the peak systolic and early diastolic phases of the cardiac cycle, this technique can measure the arterial CBV at systole and diastole, respectively. VC can be subsequently calculated as the ratio of the change of CBV between systole and diastole and the change in arterial pressure, which was approximated by brachial pulse pressure. Since dynamic ASL is able to estimate arterial CBV in both large and small arteries and even in arterioles, this technique can provide in vivo estimation of both global and regional intracranial VC in different components of the cerebrovascular bed.

MRI experiments

All experiments were carried out on a Siemens Tim Trio 3 T scanner using the product 12-channel head coil. A total of 48 subjects (48.6 ± 18.9 years, 21 males) were recruited in this study, and 12 were excluded due to head motion (see below), resulting in a total of 36 subjects (48.6 ± 20 years, 15 males) that were included in data analyses. All subjects provided written informed consents before they participated in the study. Following standard scout and anatomical MRI, an ECG-triggered time-resolved phase contrast (PC) MRI was performed to measure the blood flow velocity in the internal carotid arteries (ICA), as shown in Fig. 1, with the following parameters: FOV = 220 × 220 mm^2, matrix = 192 × 192, FA = 15°, TE = 5.23 ms, VENC = 100 cm/s, 23 phases with an interval of 50 ms, a single axial slice of 5-mm thickness at the level of C1/C2 was imaged with a scan time of 2 min. A typical profile of mean flow velocity in ICA across a cardiac cycle is shown in Fig. 1. The time delays at peak systole and early diastole were identified in each individual subject (on average 150 ms and 400 ms following the trigger, respectively). Two separate ECG-triggered multi-phase bSSFP ASL scans (Control at systole-Tag at systole; Control at diastole-Tag at diastole) were performed with pulsed spin labeling synchronized with the peak systolic and early diastolic phases by an ECG trigger, respectively. The flow-sensitive alternating inversion recovery (FAIR) scheme was implemented for spin labeling, which was immediately followed by a cine bSSFP readout train. The imaging parameters were: FOV = 220 × 220 mm^2, matrix = 96 × 96, FA = 40°, TE/TR = 1.87/3.74 ms, partial Fourier in the phase encoding direction = 6/8, centric ordering k-space acquisition with 20 lines per segment, and 29 phases from 150 to 2250 ms with an interval of 75 ms. The interval between inversion pulses was approximately 3 s covering 3 to 4 heartbeats. An oblique axial slice of 5-mm thickness (Parallel to AC–PC) at the level of internal capsule was imaged. In each cine bSSFP ASL scan, 8 pairs of label/control acquisitions were collected which took approximately 3 min. The two bSSFP ASL scans at systole and diastole took approximately 6 min. To minimize the head motion during scan, paddings were placed around the head. Before and after the MRI scans, brachial blood pressure (BP) was recorded using a MR compatible cuff sphygmomanometer. ECG leads were placed on subjects' chest to trigger VC scans which were performed in the following 4 experiments.

Exp. 1: assessment of VC in different vascular components

Nine young healthy subjects (21.8 ± 2.6 years, 6 males) were included in Exp. 1. Based on the Bloch equation simulation and experimental data, cine bSSFP based dynamic ASL scans can only measure CBV in arteries and arterioles. Therefore, two ECG-triggered bSSFP ASL scans were performed to assess VC in arteries and arterioles. For comparison, two ECG-triggered Look-locker (LL) echo-planar imaging (EPI) ASL

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