Original Research

Estimation of Pulse Wave Velocity in Main Pulmonary Artery With Phase Contrast MRI: Preliminary Investigation

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Purpose: To assess the feasibility and reproducibility of a noninvasive MRI method to measure pulse wave velocity (PWV) in the main pulmonary artery (MPA).

Materials and Methods: A total of 17 subjects without history of pulmonary diseases (38.2 ± 18.4 years) participated in this study. Series of MR velocity maps of the MPA were acquired at 2 cm above the pulmonary valves using a two-dimensional phase-contrast sequence. Effective temporal resolution was 11 msec after interleaving two dynamic series with different values of electrocardiograph (ECG) trigger delay. PWV was derived as the rate of MPA flow variations per unit change in MPA cross-sectional area, during early systole. Seven healthy subjects underwent three repetitive examinations to investigate intrascan and interscan reproducibility.

Results: Flow vs. area was highly linear in the MPA during early systole, with Pearson's coefficients ranging from 0.982 to 0.999, rendering derivation of PWV with little difficulty. Average value of PWV in MPA was 1.96 ± 0.27 m/second, in good agreement with literature values measured using invasive means. The percentage intra- and interscan differences were 5.46% and –10.86%, respectively.

Conclusion: Phase-contrast MRI to noninvasively measure PWV in the MPA is feasible with good reproducibility.

Key Words: pulse wave velocity; main pulmonary artery; phase contrast imaging; pulmonary arterial hypertension; noninvasive measurement.
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THE REPEATED EJECTION of the blood by the heart generates pressure waves in the aorta and the main pulmonary artery (MPA), which are then transmitted through the arterial trees (1). The propagation velocity of the pressure waves depends primarily on the distensibility and the compliance of the vessel wall (2,3). Therefore, a measurement of the pulse wave velocity (PWV) may be helpful in assessing the elastic properties of the vessel wall, which in turn may increase understanding of certain vessel pathophysiology. As an example, pulmonary arterial hypertension (PAH) is a challenging pathological condition characterized by progressive elevation in the blood pressure of the arteries of the lung, which could lead to substantial morbidity and mortality from failure of the right ventricle if not treated properly (4). It has been shown that the pulmonary artery distensibility decreases significantly in patients with familial or idiopathic PAH (5) or secondary PAH associated with congenital heart disease (6). Hence, a noninvasive PWV assessment in the MPA may have potential providing assistance in the screening for PAH.

The assessment of PWV in the aorta has been demonstrated using catheter insertion (7), Doppler ultrasound (8,9), and MR phase-contrast imaging (10–13). Compared with PWV in the aorta, however, PWV in the MPA is much less documented (5,14), despite its potential importance in the screening of PAH. In addition to very early works employing either theoretical models of vascular trees (15) or highly invasive measurement techniques (16), only scarce reports are found in the literature (14,17,18). The lack of investigative studies on PWV in the MPA can be understood because a measurement of arterial PWV usually involves the determination of the transit time of the pressure pulse over a known distance (19), while the short length of the MPA (about 5 cm) (20) imposes a practical difficulty as it demands a high temporal resolution.

Recently, a research group proposed a single-slice MR imaging method to measure PWV in the aorta using the phase-contrast technique, which we shall term the "QA method" in this study (21). The QA method is based on the incrementally linear relationship between aortic flow (Q) and vessel cross-sectional area (A) during early systole, for which the slope was shown equal to the PWV, as validated by other means (10,11,21). Due to

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Table 1
Clinical Data and Main Pulmonary Artery Pulse Wave Velocity of the 17 Subjects

Subjects	Sex	Age (year)	Pulse wave velocity (m/second)	Diagnosis
1	M	23	1.88	Healthy
2	M	23	1.86	Healthy
3	M	23	1.40	Healthy
4	M	24	2.12	Healthy
5	M	23	1.90	Healthy
6	F	26	1.51	Healthy
7	M	25	2.11	Healthy
8	M	23	1.97	Healthy
9	M	57	2.17	DM, HT
10	M	17	1.65	Marfan syndrome
11	M	51	2.01	HIVD, alpha-thalassemia
12	M	62	1.88	CAD, HT, CABG
13	M	52	2.45	DAA
14	M	76	2.00	CAD, AMI
15	F	62	2.32	HT
16	F	40	2.20	Liver tumor
17	F	42	1.81	Acute myocarditis
Mean ± SD		38.2 ± 18.4	1.96 ± 0.27	

DM = diabetes mellitus, HIVD = hernia of intervertebral disk, HT = hypertension, CAD = coronary artery disease, CABG = coronary artery bypass grafting, DAA = dissecting aneurysm of the aorta, AMI = acute myocardial infarction, M = male, F = female.

the short length of the MPA, the single-slice QA method seems to be inherently suitable for measuring PWV in the MPA. As will become clear in a later section, however, application of the QA method to the measurement of PWV in the MPA is more challenging than in the aorta due to irregular shape of the MPA cross-sectional area. In this study, therefore, we aimed to study the reproducibility of the QA method in the estimation of PWV in MPA, using images acquired from healthy young subjects. We further recruited patients (i.e., supposedly less cooperative subjects than healthy volunteers) without apparent symptoms of PAH to investigate the applicability of this method in clinical routine examinations.

MATERIALS AND METHODS

Subjects

MR imaging was performed on 17 subjects (four women and 13 men) aged 17–76 years (38.2 ± 18.4 years). Eight were healthy young volunteers (23.8 ± 1.2 years) who were normotensive, free from cardiovascular, respiratory, or central nervous system disease, and without alcohol, tobacco, or drug abuse. Seven of the eight healthy volunteers participated in the experiments on reproducibility test. The other nine subjects (51.0 ± 16.8 years) were patients without PAH symptoms, who were admitted to the hospital and referred to MR examinations for causes other than pulmonary disease. They were randomly recruited in this study merely because the torso coil array was used, which facilitated insertion of our sequence protocol with minimum interference to the clinical routine. Basic data and clinical diagnosis of these subjects are summarized in Table 1, along with their PWV values measured using the method as described later. All subjects provided written informed consents before scanning.

Imaging Experiments

MR imaging was performed on a 1.5-T clinical imager (Siemens Sonata, Erlangen, Germany) using the torso coil array. A three-plane localizer was first obtained, following which another nearly-sagittal oblique slice whose orientation was identified from the transaxial and coronal localizers was acquired at the location of the MPA using a two-dimensional true fast imaging with steady precession (TrueFISP) sequence (Fig. 1a). The image slice from which the PWV would be derived (Fig. 1b) was then determined as the plane perpendicular to the long axis of MPA at about 2 cm above the pulmonary valves (white solid line in Fig. 1a). The rationale for choosing 2 cm above the pulmonary valves was due to a balance between two requirements: The measurement site should be as close as possible to the right ventricle to maximize the available measurement time window without interference by the reflection wave, while the distance to the pulmonary valves should simultaneously be sufficiently far in order to avoid the presence of turbulent flow.

A two-dimensional single-slice multiphase phase-contrast fast low-angle shot (FLASH) sequence (TR/TE = 22/4.8 msec with electrocardiograph (ECG) triggering, flip angle = 15°, voxel size = 1.17 × 1.17 × 5 mm^3, one signal average, acquisition time <3 minutes) with ± 150 cm/second velocity-encoding sensitivity (Venc) was used to acquire velocity maps across the MPA at different cardiac phases, sampling 85% to 90% of the cardiac cycle. The sequence was repeated twice with trigger delays of 0 and 11 msec from the R-wave trigger pulse, respectively, such that an effective temporal resolution of 11 msec could be achieved by subsequently interleaving the data obtained from two successive phase-contrast imaging acquisitions. Each complete series of dynamic velocity maps hence took about six minutes to acquire. For the reproducibility

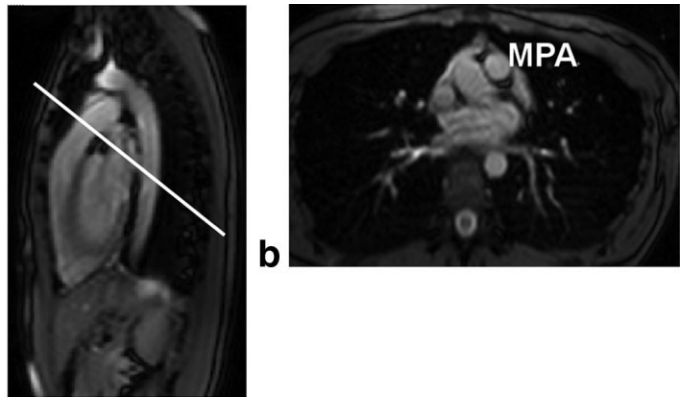

Figure 1. Slice location for derivation of pulse wave velocity from main pulmonary artery. **a:** Sagittal image shows the prescription of oblique slice perpendicular to the main pulmonary artery (white solid line). **b:** The resultant image.

investigation, the same examination was repeated thrice: the first two with identical localizer to assess intrascan reproducibility, and the last one with the subject coming out from the scanner and repositioned to examine interscan reproducibility. The total examination time in the reproducibility experiments was approximately 20 minutes for each subject, depending on the heart rate, including the localizing scans. For clinical patients recruited in this study, the phase-contrast study alone took about 10 minutes or less, again depending on the heart rate, out of a 40-minute clinical protocol.

Data Processing and Statistical Analysis

After obtaining a series of dynamic velocity maps of each subject, the derivation of PWV followed the QA method proposed by Vulliemoz et al (21). In brief, a transmission of the pressure pulse along a certain artery is accompanied by a change in the blood flow (Q), which in turn is associated with an alteration in the cross-sectional area (A) of the vessel. For vessel wall having a higher distensibility, it is expected that an increase in the blood flow is partly absorbed by the vessel wall dilatation, resulting in a slower propagation of the pressure pulse along the vessel and a simultaneous greater enlargement in the vessel cross-sectional area (10). The PWV is hence related to the amount of incremental flow per unit change in the vessel cross-sectional area (dQ/dA) as the pressure pulse propagates (21). Note that the above argument holds true only when there is no reflection wave superimposed on the forward-transmitting pressure pulse. Thus, the derivation of the PWV in the MPA involved measurements of the MPA cross-sectional area and instantaneous flow as a function of time at one slice location, yet only during early systolic phases where an approximately linear relationship between dQ and dA could be found.

For each cardiac phase, the region of interest (ROI) corresponding to the MPA was manually outlined in the absolute phase image by one operator (H.H.P.) (Fig. 2a). The rationale for using the absolute phase images was that the MPA exhibited higher contrast from adjacent vessels and tissues than in the magnitude images (Fig. 2b) and phase images (Fig. 2c). The cross-sectional area of the MPA was then determined. Note that the procedure of manual outlining for the MPA is obviously a potential source of error. Hence the imprecision of MPA area measurement and its resulting disagreement in PWV derivation were also assessed by repeated manual outlining of the MPA area on the early systole images for 10 randomly selected subjects (five healthy subjects and five patients, totally more than 50 images), by the same operator (H.H.P.) on two different days, two to six months apart from each other, without identification of the subject names beforehand. Interobserver variability was not assessed in this preliminary proof-of-principle investigation, because subjective outlining of the MPA by different operators is inevitably prone to errors caused by training factors and empirical rules among different individuals. Following area determination, flow was computed as the integration of the velocity derived from the phase images (Fig. 2c) over the entire MPA area. Data for the instantaneous MPA flow were subsequently plotted against the MPA cross-sectional area, from which the linear region corresponding to early systole was identified. The early systolic phases of our study population were about 10–60 msec after the R-wave trigger pulse. The actual time points chosen to derive the PWV depended on the duration of the linear upslope of flow profile in each case. The slope of regression (i.e., dQ/dA) was then derived to give PWV (21).

Imprecision of the MPA cross-sectional area, mean velocity, instantaneous flow rate, and the consequent PWV derivation due to manual outlining of the MPA was assessed and expressed in percentage deviation from their average values (i.e., the unbiased estimates). Intrascan and interscan reproducibility of the PWV estimations was evaluated using Bland-Altman analysis for pairs of measurements (22). Possible difference in PWV between healthy subjects and patients was investigated using the Student's t-test. A P value < 0.05 was considered statistically significant.

RESULTS

Figure 3a and b show the instantaneous flow rate plotted as a function of the MPA area (QA plot) at different cardiac phases for a 24-year-old healthy male and a 42-year-old female patient, respectively. Only the data points during early systole are shown. The fitting of the linear segment in the two plots to derive the PWV of

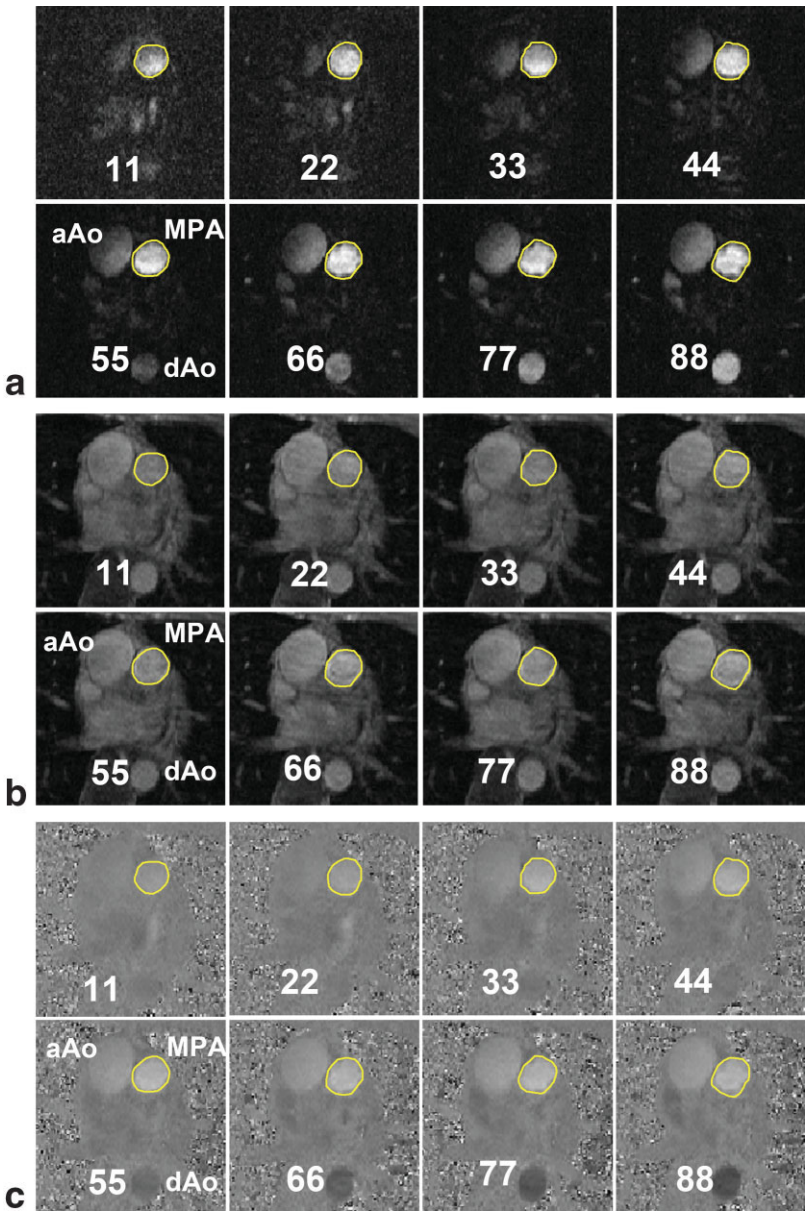

Figure 2. Velocity maps at eight cardiac phases in a 23-year-old healthy male subject. **a:** Absolute phase MR images are used to determine the cross-sectional area of the main pulmonary artery (yellow) due to conspicuous distinction between the vessel and background. Cardiac phases are indicated at the bottom of the images as the time after the R-wave trigger pulse. Abbreviations: MPA: main pulmonary artery; aAo: ascending aorta; dAo: descending aorta. **b:** Magnitude MR images show clear identification of main pulmonary artery from the anatomic locations, although with less contrast between the MPA and adjacent vessels or tissues. **c:** Phase MR images are used to measure the flow during early systole.

these subjects was seen to exhibit no difficulty, as reflected by the high Pearson's correlation coefficients of 0.998 or higher. In our study population, the Pearson's correlation coefficients ranged from 0.982 to 0.999 in both the healthy volunteers and patients, showing good linearity of the regression curves. Therefore, the assumption of no reflection wave during early systole as demonstrated for the aorta by Vulliemoz et al (21) seems to be valid for the MPA as well.

Figure 4 shows the precision of intraobserver PWV derivations with manual MPA area estimation, analyzed using Bland-Altman analysis (22). It is seen from the dotted lines that the standard deviation (SD) of the disagreement was 0.08 m/second for the 10 subjects, suggesting high precision. Mean percentage deviations from the averages were 2.97%, 6.22%, 9.53%, and 5.66% for MPA cross-sectional areas, mean velocity within the chosen ROI, instantaneous flow rate, and PWV, respectively.

The PWV values measured from the seven healthy subjects in the reproducibility tests are shown in Table 2. Bland-Altman plots for intrascan and interscan agreement are given in Fig. 5a and b, respectively. Note from Fig. 5b that the interscan agreement was assessed using all available pairs of data, hence resulting in 11 data points (cf. Table 2). Intrascan disagreement was found to be 0.03 ± 0.17 m/second (mean ± SD; mean percentage difference 5.46%), whereas interscan disagreement was somewhat larger, at −0.05 ± 0.25 m/second (mean percentage difference 10.86%).

Figure 6 shows the distribution of PWV of MPA for the 17 subjects. Overall, the mean PWV was found to be 1.96 ± 0.27 m/second. When divided into two groups, the healthy volunteers and the patients showed average PWV values of 1.84 ± 0.26 m/second and 2.05 ± 0.26 m/second, respectively. For these subjects having neither pulmonary disease nor PAH symptoms, the group

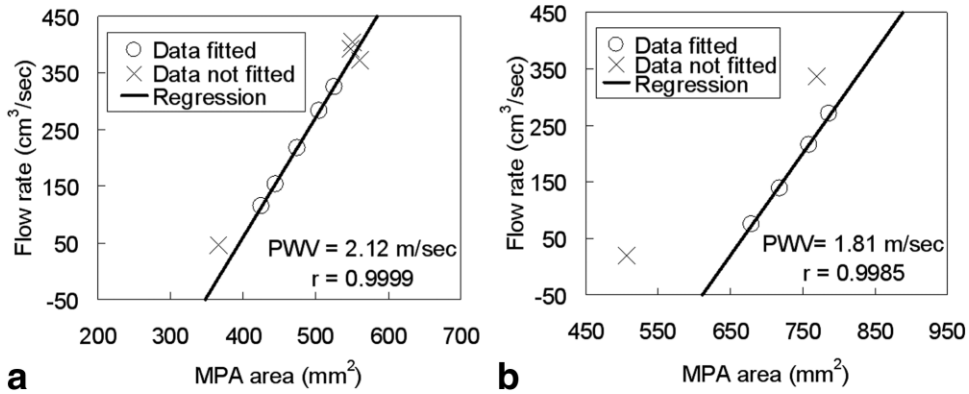

Figure 3. Plots show flow rate vs. cross-sectional area of the pulmonary artery in a 24-year-old male subject at nine cardiac phases (**a**) and in a 42-year-old female patient at six cardiac phases (**b**), respectively. The pulse wave velocity values as derived from the linear region (circles) was 2.12 and 1.81 m/second, respectively. The linearity of the relationship between flow rate and vessel area, which justified the appropriateness of linear regression to estimate pulse wave velocity during early systole, was evidenced from the high Pearson's correlation coefficients (r = 0.998 or higher). The data points obtained from outside the early systole region (crosses) were seen to deviate from the regression line.

difference in PWV of the MPA was statistically insignificant (Student's *t*-test, $P > 0.05$).

DISCUSSION

PWV has been regarded as an important physiological parameter for the aorta in systemic circulation diseases (2,7,9,10,12,21). Reports on the measurements of PWV in the MPA, however, are rarely found (14–16). In this study, we demonstrated the feasibility of a single-slice approach, namely the QA method (21), to measure PWV in the MPA using noninvasive phase-contrast MR imaging. Mean percentage differences in repeated PWV measurements were 5.46% and 10.86% for intrascan and interscan, respectively.

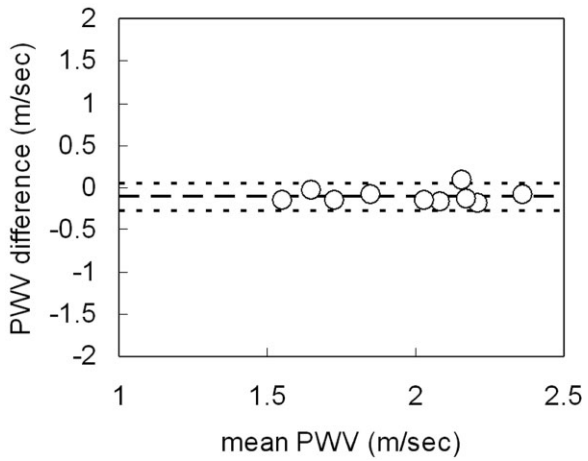

Figure 4. Bland-Altman plot shows difference in PWV derivations obtained using two manually estimated cross-sectional areas of the main pulmonary artery, plotted vs. their average values for 10 subjects (five healthy volunteers and five patients). The disagreement in PWV from manual area measurements was found to be −0.05 ± 0.04 m/second, suggesting good precision. The dashed line indicates the mean difference, whereas the dotted lines represent mean difference ±2 SD.

The PWV measured from the MPA of the 17 subjects in our work was 1.96 ± 0.27 m/second, which is close to literature values (15,16,23). In 1967, Caro et al (16) employed an invasive foot-to-foot method to measure PWV in the MPA and obtained a value of 1.82 m/second, in good agreement with our results. In 1969, Milnor et al (15) reported a PWV value of about 1.68 m/second in three subjects (21, 22, and 53 years of age, respectively) with normal cardiovascular physiology using a method involving averaging of higher frequency harmonics, which is also consistent with the data from the young healthy subjects recruited in our study. While being able to obtain the same quantity as these reports, the MR method chosen in our study here has the important advantage of non-invasiveness and hence is potentially suitable for routine clinical examinations.

Laffon et al (14) also used phase-contrast MRI to investigate the tuning of pulmonary arterial circulation, that is, the adaptive optimization of coupling between the right ventricle and the pulmonary arterial tree in order to reduce right ventricle loading. They found the PWV in MPA to be 3.08 ± 0.56 m/second for 12 healthy subjects (14), much higher than the 1.96 ± 0.27 m/second reported in the current study. There are several possibilities that could explain the data inconsistency. In particular, the theoretical model proposed by Laffon et al (14) was based on several important assumptions that have to be validated before being put into practice. One assumption is that the reflection coefficient for the pressure pulse is global, meaning that the individual backward pressure waves generated from arterial branches of the lungs are indistinguishable. In other words, Laffon's model did not take into account the nearest reflections at the bifurcation of the two large pulmonary arteries. Another assumption is that the pulmonary arterial system has reached resonance (or being "tuned"), satisfying the quarter-wavelength condition of a sinusoidal pressure wave (14). While these assumptions await experimental verification, it should be further noted that Laffon's model to compute PWV

Table 2
Pulse Wave Velocity Values in Main Pulmonary Artery for the Reproducibility Experiments

Subject number	Sex	Age (years)	Identical localizer		Different localizer
			PWV1 (m/second)	PWV2 (m/second)	PWV (m/second)
1	M	23	2.19	1.83	1.88
2	M	23	1.86	–	2.35
3	M	23	1.37	1.40	–
4	M	24	2.12	2.24	2.19
5	M	23	1.95	1.90	1.95
6	F	26	1.47	1.51	1.24
7	M	25	2.11	2.15	2.52
Mean ± SD		23.9 ± 1.2	1.87 ± 0.36	1.84 ± 0.34	2.02 ± 0.46

M = male, F = female, – = no measurement or poor image quality.

involves rather laborious iteration processes, with initial parameters such as the difference between systolic and diastolic pressures set at subject-independent values. In a separate study comparing five MR approaches estimating pulmonary arterial pressure, the Laffon model was found to exhibit very limited accuracy when a good correlation is lacking between pulse pressure and mean arterial pressure in the MPA (18). Over- and underestimations of different ranges of data are reported, which are attributed to some of the key steps to restrict the range of parameter correlations that appear to be highly subjective (18). These aforementioned issues may lead to questionable reliability of the 3.08 ± 0.56 m/second PWV in the MPA.

Compared with the theoretical model used by Laffon et al (14), the QA method employed in our study is advantageous in that fewer assumptions are needed. The most critical assumption needed in the QA method is that there should be no reflection wave at the measurement site during early systole, such that the incremental variation in blood flow is proportional to the change in MPA cross-sectional area. Validity of this assumption is supported by two pieces of evidence. First, the QA plots for our subjects showed high linearity for the data points measured during early systole (usually about 11–55 msec after the R-wave trigger pulse), suggesting an absence of interference from reflection waves during that period (21). Second, for average PWV of the order of 2.0 m/second as found in our study, it takes about 10 msec for the pressure pulse wave to originate from the right ventricle after end-diastole (i.e., appearance of R wave) to the MPA site of PWV measurement (Fig. 7). Assuming identical PWV for the transmitting pulse and the reflection pulse, the time needed for the reflection pulse wave to reach the same site is about 40 msec after end-diastole for an MPA 5.0 cm in length (20). That is to say, for data points measured between 10 and 40 msec after the R wave, the assumption of no reflection wave should be valid. It is noticed that for the subjects recruited in our study, the QA data measured between 11 and 55 msec after the R-wave trigger pulse followed a linear trend closely, whereas those measured at 0 msec and after 66 msec after the R-wave trigger pulse deviated substantially from the regression line (cf. Fig. 3), consistent with the estimated PWV values on the order of 2.0 m/second. As to why the QA value at 55 msec after the R wave also followed the regression line in some cases, we think that it might be due to either somewhat slower wave propagation for the reflection pulse than the forward-transmitting pulse, or weakened reflection wave at the MPA bifurcation from nonideal termination (23). Note that although it is an oversimplification to assume identical PWV for the transmitting and the reflection pulses, the assumption is likely to be largely valid. The velocity of wave propagation in blood vessels depends on the compliance, mean cross-sectional area of vessel wall, and blood volume mass (24,25). Since these parameters do not change instantaneously during a short period, the PWV is expected to be relatively constant for the MPA. Furthermore, if PWV were to be altered during reflection, the reflection pulse is more likely to travel slowly rather than being accelerated. While these uncertain issues certainly warrant further investigations, the validity of the experimental linear relationship between flow rate and cross sectional area in the MPA is relatively unaffected.

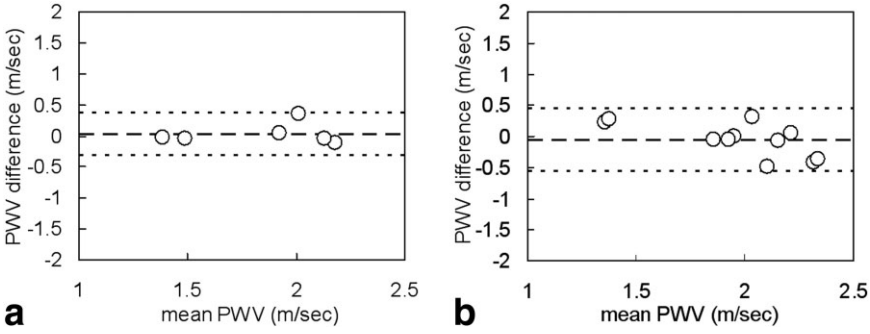

Figure 5. Bland-Altman plots for the intrascan and interscan comparison of pulse wave velocity measurements. **a:** Intrascan (i.e., identical localizer) disagreement of six subjects was 0.03 ± 0.17 m/second (mean percentage difference 5.46%). Similar to Fig. 4, the dashed line indicates the mean disagreement. Dotted lines represent mean disagreement ±2 SD. **b:** Interscan (i.e., subject repositioned) disagreement of 11 pairs of data was –0.05 ± 0.25 m/second (mean percentage difference 10.86%).

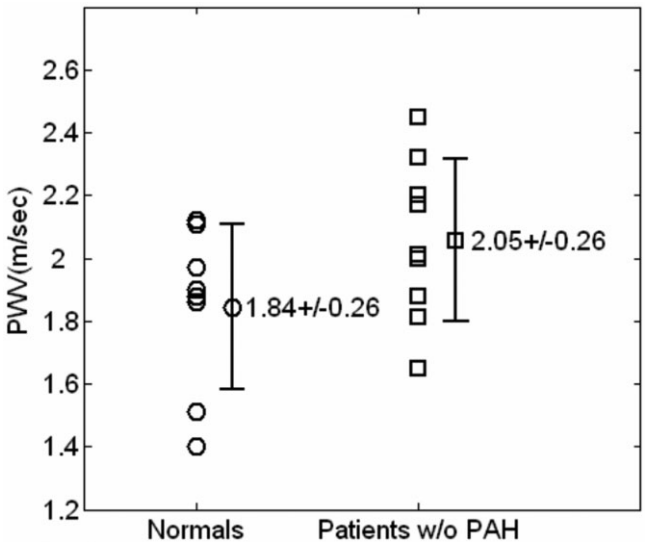

Figure 6. Plot shows distribution of PWV in MPA of eight normal volunteers and nine patients without apparent symptoms of PAH. The values of mean PWV from these two groups were found to be 1.84 ± 0.26 m/second and 2.05 ± 0.26 m/second, respectively. The difference was statistically insignificant (Student's *t*-test, $P > 0.05$).

The arguments regarding QA data availability as stated above could also help in consolidating the PWV values of the order of 2.0 m/second found in our study. If PWV in the MPA were about 3.0 m/second as reported by Laffon et al (14), the number of data points following a linear trend in the QA plot would be substantially less because they would need to be measured between 7 and 27 msec after the R wave. In contrast, the fact that we were able to find good data linearity between 11 and 55 msec supports the order of 2.0 m/second PWV (15,16,23).

Temporal resolution is an important factor for reliable PWV estimations, considering that the time window after the R wave for valid QA data points is quite limited in the MPA. The phase-contrast sequence used in our current MR system allows a minimum TR of 22 msec at a velocity encoding value of ±150 cm/second. To increase the number of time points, we performed two consecutive scans at different ECG trigger delay values (0 and 11 msec after the R-wave trigger pulse, respectively) and interleaved the two sets of data. Involuntary motions between the two interleaving scans may cause jagged profiles of the time course data, particularly for patients who are supposedly less cooperative than the healthy subjects recruited in this study. In addition, momentary changes in physiology may alter pulmonary PWV continuously, hence potentially invalidating PWV derivations using two separate acquisitions when the total scan time becomes long. Possible remedies include choices of higher readout bandwidth or fractional echo such that a TR of about 10 msec or less could be achieved, or to interleave the two scans within the phase encoding loop. These await continuous advances in imaging gradient hardware and sequence software.

There are several limitations that should be mentioned about this study. First, manual outlining of the MPA remains subjective and is prone to inaccuracy, particularly when the blood flow velocity is small. Sequences that better delineate the vessel wall, such as the balanced steady-state free precession imaging combined with phase-contrast capability (26), could be used for this purpose. However, a larger scale of technical development would be needed, which is beyond the scope of this study. In order to minimize operator inaccuracy, we investigated the reproducibility of manual area measurements, which at least provided an estimate of the precision level to be expected for PWV in the MPA. If this method were to be executed at different medical centers, an automatic MPA-outlining tool such as those based on active contouring (27) would be highly desirable to minimize operator dependency. A second limitation is that the QA method is unable to estimate PWV reliably when there is severe flow reversal, as might be encountered in patients with late-stage PAH. Hence, we anticipate that only subjects with normal pulmonary circulation and patients having moderate PAH with mild flow reverse are suitable candidates for PWV estimations in the MPA using the current QA method employed in this study. Therefore, the PWV measurement should be regarded as a method that could potentially assist screening and early diagnosis of PAH, rather than being used for continually monitoring

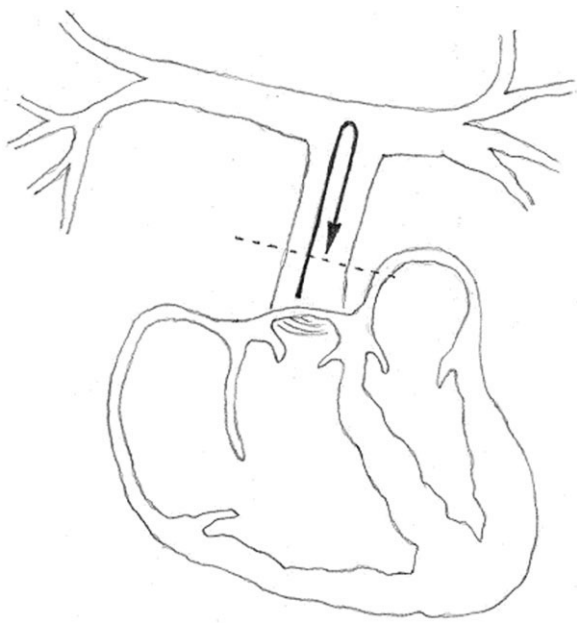

Figure 7. Schematic drawing of pulse wave propagation in the main pulmonary artery. Curved arrow indicates the propagation of transmitting and reflecting pulse wave originating from the right ventricle. Since the imaging slice was acquired at 2 cm above the pulmonary valves (dotted line), the total path of pulse wave travel was about 8 cm in length for the transmitting pulse to reach the bifurcation and then return to the imaging slice, assuming a 5-cm-long main pulmonary artery. For pulse wave velocity of the order of 2.0 m/second, the data points during early systole (i.e., in the absence of reflecting wave) were hence valid between 10 and 40 msec after the R-wave (i.e., end diastole). If the reflection wave propagates at a slower speed than the transmitting wave, the available data points could be further extended.

the progress of PAH severity. In fact, it is for this reason that we included only healthy volunteers and patients without pulmonary diseases to examine the feasibility of the QA method and the resulting PWV ranges in the MPA. The statistically insignificant difference in mean PWV obtained from the two groups should not be interpreted with any physiological meaning at the moment, because age-matched recruitment was not performed and some of the diseases might lead to changes in pulmonary pressure. Another weakness of this study is that we did not compare our results to an independent standard of reference. For these subjects having no pulmonary disease, invasive catheter insertion is certainly impossible. Doppler ultrasound might be one possibility. However, the accuracy of noninvasive PWV measurements in the MPA is somewhat limited due to short penetrating depth (18,28,29). Therefore, a reliable external reference standard of PWV measurements in the MPA is impractical at this moment, if ever available. Further research on patients with proven PAH, where catheter insertion is clinically indicated, is highly desirable to resolve the validation issue.

In conclusion, our work demonstrated that a noninvasive measurement of PWV in the MPA is feasible using phase-contrast MR imaging. Our approach was shown to be highly reproducible with good precision, with the estimated PWV values in agreement with literature data obtained using invasive measurements. The methodology developed in our study and the reported baseline PWV for healthy subjects may be potentially helpful in future clinical applications involving pathological changes in the distensibility of the MPA vessel wall.

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