Flow characteristics of the proximal pulmonary arteries and vena cava in patients with chronic thromboembolic pulmonary hypertension: correlation between 3.0 T phase-contrast MRI and right heart catheterization

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PURPOSE

We aimed to determine the correlation between flow characteristics of the proximal pulmonary arteries and vena cava obtained by 3.0 T phase-contrast magnetic resonance imaging (MRI) and hemodynamic characteristics by right heart catheterization in patients with chronic thromboembolic pulmonary hypertension.

MATERIALS AND METHODS

Twenty consecutive patients with chronic thromboembolic pulmonary hypertension and 20 sex- and age-matched healthy volunteers were included prospectively. All patients and controls underwent phase-contrast MRI to determine the flow characteristics including peak velocity, mean velocity, and mean blood flow of the proximal pulmonary artery and vena cava. All patients underwent right heart catheterization to determine the hemodynamics.

RESULTS

Peak velocity and mean velocity of the proximal pulmonary artery were significantly lower in the patient group. In patients, both peak velocity and mean blood flow were sequentially decreased in the main pulmonary artery, left and right pulmonary arteries, and left and right interlobar pulmonary arteries. Inferior vena cava had higher peak velocity, mean velocity, and mean blood flow than superior vena cava. Peak velocity of the main pulmonary artery correlated with mean and diastolic pulmonary artery pressure. Peak velocity of both inferior and superior vena cava strongly correlated with the pulmonary vascular resistance index (PVRI) ($r=-0.68$, $P < 0.001$ and $r=-0.74$, $P < 0.001$, respectively). Mean velocity of the main pulmonary artery and right pulmonary artery strongly correlated with PVRI and mean pulmonary artery pressure. Mean velocity of the superior vena cava and mean blood flow of the main pulmonary artery strongly correlated with PVRI and right cardiac work index.

CONCLUSION

Blood flow in the proximal pulmonary artery and vena cava evaluated by phase-contrast MRI correlate with hemodynamic parameters of right heart catheterization and can be used to noninvasively evaluate the severity of chronic thromboembolic pulmonary hypertension and, potentially, to follow up the treatment response.

Chronic thromboembolic pulmonary hypertension (CTEPH) develops as a result of obstruction of pulmonary arterial vessels by organized thromboembolic material and subsequent vascular remodeling in small unobstructed vessels, and it is associated with significant morbidity and mortality (1). Right heart catheterization remains the reference standard to diagnose CTEPH, assess the hemodynamic disturbance, and follow up the treatment response; but it is invasive, delivers radiation, and is associated with recognized complications (2, 3).

Magnetic resonance imaging (MRI) is considered not only as the reference standard for evaluation of ventricular function, but it also provides a reproducible and noninvasive assessment of hemodynamics changes in pulmonary hypertension (4). In a separate validation study, cardiac MRI-derived parameters showed a strong correlation with invasive determinations (5).

In a study by Mohiaddin et al. (6), phase-contrast MRI was used to confirm reduced diastolic peak velocity of the inferior vena cava (IVC) in patients with pulmonary hypertension. Only one study reported blood flow conditions in the proximal pulmonary arteries and vena cava in healthy children (7). To our knowledge, no study has assessed proximal pulmonary artery and vena cava flow or evaluated correlation of the flow determined by phase-contrast MRI and hemodynamics by right heart catheterization in CTEPH.

We performed a prospective study to observe the flow change in the proximal pulmonary artery, superior vena cava (SVC) and inferior vena cava (IVC) in patients with CTEPH by phase-contrast MRI, and to evaluate the correlation of the proximal pulmonary artery and vena cava flow with hemodynamics derived by right heart catheterization in patients with CTEPH.

Materials and methods

Subjects

The ethics committee of our institute approved this study, and written informed consent was obtained from all patients.

From August 2011 to February 2012, 30 consecutive patients with confirmed CTEPH were included in this prospective study. CTEPH was defined as mean pulmonary arterial pressure (mPAP) >25 mmHg, pulmonary capillary wedge pressure ≤ 15 mmHg, pulmonary vascular resistance (PVR) >2 Wood units, and a ventilation/perfusion nuclear medicine scan consistent with thromboembolic disease. An investigation protocol to exclude other types of pulmonary hypertension was completed, as described in the European Society of Cardiology/European Respiratory Society guidelines (8). All patients underwent MRI

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and right heart catheterization within three days. Five patients were excluded because of obstructive airway disease, two patients were excluded because of coexisting autoimmune diseases, and three patients were excluded because of poor MRI quality. Twenty patients (13 males and seven females; mean age, 58.1 years; median age, 61 years; range, 33–76 years) were included. Twenty sex- and age-matched healthy volunteers (10 males and 10 females; mean age, 54.2 years; median age, 60.5 years; range, 15–70 years) were included as the control group and underwent phase-contrast MRI.

Magnetic resonance imaging and analysis

MRI was performed with 3.0 Tesla MR scanner (TimTrio, Siemens, Erlangen, Germany) using a four-channel cardiac phased-array surface coil for data acquisition of phase-contrast MRI. For perpendicular pulmonary artery sections, sagittal and oblique coronal images, positioned parallel to the direction of the main pulmonary artery were obtained, using a half-Fourier acquisition single-short turbo spin-echo sequence and true-fast imaging with steady-state precession sequence, during expiratory breath holding. Image sections of phase-contrast MRI for flow measurement were planned in a double oblique section perpendicular to the main pulmonary artery (MPA) and 1.0–1.5 cm above the pulmonary valve, 1.0–1.5 cm after MPA bifurcation for left and right pulmonary arteries (LPA and RPA), and 1.0–1.5 cm after LPA and RPA branches into interlobar pulmonary arteries in each patient (7, 9) (Fig. 1a–1d). The imaging planes for SVC and IVC were superior and inferior to their insertions in the right atrium (6) (Fig. 1e, 1f). Phase-contrast MRI was performed with velocity encoding magnetic resonance phase-contrast cine pulse sequence (TR, 47 ms; TE, 1.99 ms; Flip angle, 30°; matrix, 256×256; field of view, 320 mm; section thickness, 6 mm; NEX, 3) using retrospective electrocardiogram triggering. Sixty frames for each cardiac cycle were obtained. The data acquisition time for each phase-contrast MRI ranged 22–35 s. The velocity encoding gradient was adjusted to 150 cm/s without aliasing and the scan was repeated until the appropriate velocity encoding

Figure 1. a–h. Imaging plane of phase-contrast MRI for flow measurement of each pulmonary artery and vena cava in patients with CTEPH (a–f). Phase-contrast MRI of main pulmonary artery (MPA, outlined in white) showing both anatomy (g) and flow velocity (h). MPA, main pulmonary artery; RPA, right pulmonary artery; LPA, left pulmonary artery; LIPA, left interlobar pulmonary artery; SVC, superior vena cava; IVC, inferior vena cava.

gradient value was determined. The number of scans for each patient ranged from one to three. All examinations and data acquisition were executed by one person in order to set the imaging plane in the same manner and avoid observer dependency.

Phase-contrast MRI was transferred to the workstation (Synago MMWP VE30A, Siemens, Berlin, Germany) and was analyzed using validated software (ARGUS, Siemens Medical System, Erlangen, Germany). An experienced radiologist analyzed the phase-contrast MRI. For flow measurement, all region-of-interests (ROIs) outlining the vessel wall were drawn semiautomatically on anatomy images (Fig. 1g). Three ROIs were selected and the measurements were averaged by the same operator. Each ROI was then copied onto the phase image from the corresponding anatomy image (Fig. 1h).

The mean velocity, peak velocity, and mean blood flow in the ROI on the phase image were automatically calculated throughout one cardiac cycle.

Right heart catheterization

All patients underwent right heart catheterization within three days after MRI. The catheter (8 F Swan-Ganz, Baxter Healthcare, Illinois, USA) was introduced using the Seldinger technique through a femoral or right internal jugular vein and positioned under fluoroscopic guidance in a pulmonary artery. After a 10-min rest for stabilization, hemodynamic parameters including systolic pulmonary arterial pressure (sPAP), diastolic pulmonary artery pressure (dPAP), and pulmonary capillary wedge pressure etc., were acquired by a monitor (M1165A, Hewlett-Packard Co, Fullerton, California, USA). Then, calculations in-

cluding pulmonary vascular resistance index (PVRI), right cardiac work index (RCWI) were performed.

Statistical analysis

Data was analyzed using commercially available software (Statistical Package for Social Sciences, version 13.0, SPSS Inc, Chicago, Illinois, USA). All data were expressed as mean±standard deviation or median, unless otherwise specified. Age and gender distributions were compared between groups using Mann-Whitney U and chi-square tests, respectively. The peak velocity and mean velocity between MPA and branching pulmonary arteries were compared by an independent t-test, respectively. The correlation between peak velocity, mean velocity, mean blood flow of pulmonary artery and hemodynamics by right heart catheterization were tested by Pearson or Spearman correlation coefficient. A significant difference was considered at $P < 0.05$.

Results

As shown in Table 1, all patients with CTEPH had a clear sign of pulmonary hypertension. The range of their pathogenesis was one month to seventeen years and a median of two years. The embolus distribution was shown in Fig. 2. There were no significant differences of gender ($P = 0.337$) and age ($P = 0.402$) between the CTEPH group and the control group.

As demonstrated in Table 2, there were significant differences in the peak velocity and mean velocity of CTEPH patients and the control group. CTEPH patients had significantly lower mean blood flow of right interlobar pulmonary artery (RIPA) than the control group ($P < 0.001$). As shown in Fig. 3, CTEPH patients had lower peak velocity, mean velocity, and mean blood flow in MPA, LPA, RPA, left interlobar pulmonary artery (LIPA), and RIPA compared with the control group. In CTEPH patients, both peak velocity and mean blood flow were sequentially decreased in MPA, LPA, RPA, LIPA, and RIPA ($P < 0.001$). In the control group, both mean velocity and mean blood flow were sequentially decreased in MPA, LPA, RPA, LIPA, and RIPA ($P < 0.001$). IVC had higher peak velocity compared with SVC (52.91 ± 13.75 vs. 39.67 ± 17.08 , $P = 0.010$). The mean velocity did not dif-

Table 1. Baseline parameters in patients with CTEPH

Baseline parameters	CTEPH patients (n=20)
Sex (male/female)	13/7
Age (years)	58.10±11.83
6MWD (m)	343.50±116.32
NYHA (II/III)	17/3
Hemodynamics	
sPAP (mmHg)	85.55±17.00
dPAP (mmHg)	29.65±6.15
mPAP (mmHg)	47.85±9.14
CVP (mmHg)	5.30±5.40
PCWP (mmHg)	9.00±3.01
PVRI (dyn·s/cm ⁵)	1070.75±437.44
RCWI (kg/min/m ²)	1.07±0.34
RVSWI (g/min/m ² /beat)	13.55±4.48

CTEPH, chronic thromboembolic pulmonary hypertension; 6MWD, 6-minute walking distance; NYHA, New York Heart Association; sPAP, systolic pulmonary artery pressure; dPAP, diastolic pulmonary artery pressure; mPAP, mean pulmonary artery pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; PVRI, pulmonary vascular resistance index; RCWI, right cardiac work index; RVSWI, right ventricular stroke work index.

fer significantly between IVC and SVC (8.37 ± 6.57 vs. 6.78 ± 4.82 , $P = 0.389$). The mean blood flow ratio of SVC to IVC was approximately 45% (19.77 ± 7.51 , 43.87 ± 20.62).

Table 3 shows the correlation between peak and mean velocities and hemodynamics in CTEPH patients. The peak velocity of MPA correlated negatively with mPAP and dPAP. The peak velocity of SVC strongly correlated with PVRI ($r = -0.74$, $P < 0.001$). The peak velocity of IVC also had a strong correlation with PVRI ($r = -0.68$, $P < 0.001$). The mean velocity of MPA and RPA strongly correlated with PVRI and mPAP. The mean velocity of SVC strongly correlated with PVRI and RCWI.

Table 4 shows the correlation of mean blood flow and hemodynamics in patients with CTEPH. The mean blood flow of MPA strongly correlated with PVRI and RCWI. The mean blood flow of RPA and SVC moderately correlated with PVRI. The mean blood flow of IVC moderately correlated with PVRI and RCWI.

Discussion

In this study, we showed that proximal pulmonary artery hemodynamic parameters obtained by phase-contrast MRI are good indicators for predicting

Figure 2. Embolus distribution feature in patients with CTEPH.

PAP and PVR in patients with CTEPH. In addition, SVC and IVC hemodynamic changes are also valuable predictors.

Phase-contrast MRI provides a non-invasive measurement of hemodynamic parameters in pulmonary hypertension patients (10). Prior studies showed good prospects for a variety of phase-contrast MRI-derived parameters in the evaluation of pulmonary hypertension and clinical follow-up (4, 11, 12). Patients with CTEPH will often require repeated studies to assess the surgical suitability and to monitor the outcome. At present, only limited evidence exists for separate evaluation of hemodynamics in CTEPH patients. Our study systematically evaluated the hemodynamics in MPA, RPA, LPA,

Table 2. Comparison of pulmonary artery hemodynamic parameters derived by phase-contrast MRI between CTEPH patients and healthy controls

	Peak velocity (mean±SD)			Mean velocity (mean±SD)			Mean blood flow (mean±SD)		
	CTEPH (n=20)	Control (n=20)	P	CTEPH (n=20)	Control (n=20)	P	CTEPH (n=20)	Control (n=20)	P
MPA	52.55±14.70	79.91±16.75	< 0.001	7.10±2.48	15.38±3.13	< 0.001	74.31±20.75	72.80±17.77	0.430
RPA	31.97±7.85	78.48±12.28	< 0.001	6.57±1.94	13.88±2.75	< 0.001	43.55±17.41	38.67±6.18	0.440
LPA	30.83±9.22	77.34±15.21	< 0.001	6.15±4.13	12.77±3.02	< 0.001	29.30±21.67	33.82±4.16	0.220
RIPA	25.05±7.97	73.55±16.00	< 0.001	6.51±3.50	11.36±1.84	< 0.001	14.02±7.41	29.06±8.27	< 0.001
LIPA	27.23±6.43	71.27±14.29	< 0.001	6.19±2.17	11.14±2.45	< 0.001	19.21±7.97	20.29±7.59	0.887

SD, standard deviation; CTEPH, chronic thromboembolic pulmonary hypertension; MPA, main pulmonary artery; RPA, right pulmonary artery; LPA, left pulmonary artery; RIPA, right interlobar pulmonary artery; LIPA, left interlobar pulmonary artery.

Table 3. Correlation coefficients between peak and mean velocity of pulmonary arteries derived by phase-contrast MRI and right heart catheterization hemodynamic characteristics (n=20)

		MPA	RPA	RIPA	LPA	LIPA	SVC	IVC
mPAP vs. PV	r	-0.48 ^a	-0.37	0.13	-0.09	-0.10	-0.41	-0.47 ^a
	P	0.031	0.114	0.584	0.700	0.662	0.076	0.035
vs. MV	r	-0.47 ^a	-0.58 ^b	0.05	-0.40	-0.44	-0.17	-0.51 ^a
	P	0.035	0.008	0.835	0.081	0.053	0.473	0.023
sPAP vs. PV	r	-0.40	-0.27	0.07	-0.12	-0.05	-0.35	-0.53 ^a
	P	0.179	0.221	0.774	0.607	0.828	0.127	0.017
vs. MV	r	-0.41 ^a	-0.49 ^a	-0.02	-0.35	-0.44 ^a	-0.19	-0.50 ^a
	P	0.042	0.028	0.922	0.132	0.040	0.433	0.026
dPAP vs. PV	r	-0.54 ^a	-0.42	-0.20	-0.36	-0.15	-0.42	-0.49 ^a
	P	0.016	0.068	0.406	0.117	0.523	0.066	0.030
vs. MV	r	-0.41	-0.51 ^a	-0.14	-0.47 ^a	-0.27	-0.21	-0.46 ^a
	P	0.075	0.023	0.549	0.035	0.245	0.370	0.042
CVP vs. PV	r	-0.37	0.01	-0.24	-0.18	0.03	-0.49 ^a	-0.56 ^b
	P	0.092	0.963	0.310	0.441	0.888	0.021	0.010
vs. MV	r	-0.08	0.24	-0.35	-0.10	0.06	-0.46 ^a	-0.57 ^b
	P	0.739	0.304	0.128	0.662	0.807	0.043	0.009
PVRI vs. PV	r	-0.41	-0.41	-0.16	-0.25	-0.12	-0.74 ^b	-0.68 ^b
	P	0.071	0.069	0.510	0.282	0.613	0.000	0.001
vs. MV	r	-0.62 ^b	-0.47 ^a	0.17	-0.34	-0.32	-0.65 ^b	-0.59 ^b
	P	0.004	0.036	0.476	0.148	0.173	0.002	0.007
RCWI vs. PV	r	0.04	0.11	0.02	0.22	-0.12	0.63 ^b	0.53 ^a
	P	0.860	0.643	0.950	0.354	0.621	0.003	0.017
vs. MV	r	0.38	0.17	-0.03	-0.18	-0.15	0.74 ^b	0.46 ^a
	P	0.097	0.464	0.910	0.449	0.529	0.000	0.040
PCWP vs. PV	r	0.08	0.05	0.28	0.08	-0.18	-0.04	-0.29
	P	0.732	0.832	0.225	0.745	0.442	0.877	0.222
vs. MV	r	-0.01	-0.04	-0.37	-0.11	-0.33	-0.09	-0.28
	P	0.976	0.885	0.105	0.639	0.155	0.696	0.227

MPA, main pulmonary artery; RPA, right pulmonary artery; RIPA, right interlobar pulmonary artery; LPA, left pulmonary artery; LIPA, left interlobar pulmonary artery; SVC, superior vena cava; IVC, inferior vena cava; mPAP, mean pulmonary artery pressure; PV, peak velocity; MV, mean velocity; dPAP, diastolic pulmonary artery pressure; sPAP, systolic pulmonary artery pressure; CVP, central venous pressure; PVRI, pulmonary vascular resistance index; RCWI, right cardiac work index; PCWP, pulmonary capillary wedge pressure.

^aP < 0.05, ^bP < 0.01.

Table 4. Correlation coefficients between mean blood flow of pulmonary arteries derived by phase-contrast MRI and right heart catheterization hemodynamic characteristics (n=20)

Mean blood flow		MPA	RPA	RIPA	LPA	LIPA	SVC	IVC
mPAP	r	-0.38	-0.43 ^a	-0.08	-0.38	0.06	0.31	-0.37
	P	0.094	0.046	0.741	0.098	0.817	0.183	0.112
sPAP	r	-0.41	-0.49 ^a	0.01	-0.28	0.08	0.27	-0.41
	P	0.075	0.029	0.950	0.239	0.746	0.157	0.076
dPAP	r	-0.31	-0.31	-0.20	-0.27	0.003	0.30	-0.28
	P	0.188	0.192	0.399	0.250	0.989	0.233	0.233
CVP	r	-0.48 ^a	-0.25	0.19	-0.15	0.09	0.24	-0.37
	P	0.030	0.289	0.412	0.523	0.706	0.309	0.105
PVRI	r	-0.73 ^b	-0.59 ^b	-0.01	-0.41 ^a	0.21	0.52 ^a	-0.53 ^a
	P	0.000	0.006	0.975	0.037	0.368	0.020	0.016
RCWI	r	0.64 ^a	-0.39	-0.26	-0.06	-0.40	-0.25	0.54 ^a
	P	0.002	0.091	0.274	0.797	0.079	0.075	0.015
PCWP	r	-0.11	-0.01	-0.59	-0.19	-0.44	-0.18	-0.19
	P	0.652	0.966	0.056	0.435	0.050	0.442	0.413

MPA, main pulmonary artery; RPA, right pulmonary artery; RIPA, right interlobar pulmonary artery; LPA, left pulmonary artery; LIPA, left interlobar pulmonary artery; SVC, superior vena cava; IVC, inferior vena cava; mPAP, mean pulmonary artery pressure; sPAP, systolic pulmonary artery pressure; dPAP, diastolic pulmonary artery pressure; CVP, central venous pressure; PVRI, pulmonary vascular resistance index; RCWI, right cardiac work index; PCWP, pulmonary capillary wedge pressure.

^a $P < 0.05$, ^b $P < 0.01$.

Figure 3. a–c. Comparison of peak velocity between CTEPH patients and healthy controls (a). Comparison of mean velocity between CTEPH patients and healthy controls (b). Comparison of mean blood flow between CTEPH patients and healthy controls (c).

RIPA, and LIPA, and their relationship with hemodynamics measured by right heart catheterization.

Firstly, we found that in CTEPH group MPA had a higher peak velocity than RPA and LPA, and those in turn had a higher peak velocity than RIPA and LIPA. But in the control group, no significant differences were found in the peak velocity of these arteries. On the contrary, in the control group, MPA had a higher mean velocity than the other proximal pulmonary arteries. Secondly, patients with CTEPH had

significantly slower peak and mean velocities of pulmonary artery compared with the healthy population. The peak velocity decreased more rapidly with the increasing vessel grade in CTEPH patients compared with the control. Thirdly, we found that IVC had significantly higher peak and mean velocities compared with SVC. This is consistent with previous study results (13).

Sridharan et al. (14) reported that phase-contrast MRI could assess differential branch pulmonary blood flow accurately. In this study, we showed that

the mean blood flow of RPA was approximately 1.5 times more compared with the LPA, and the right to left pulmonary flow distribution would be 59% to 41%. These ratios are consistent with the fact that the right lung is somewhat larger than the left lung because of the position of the heart on the left (9). We also found that the embolus burden did not affect pulmonary flow distribution. The reason may be that most clots are found in the proximal arteries, but pulmonary flow is mainly modulated by the distal pulmonary vasculature. The

31% to 69% SVC to IVC flow ratio in our study was consistent with the quoted 35% to 65% ratio in the literature (15). It is hypothesized that vena cava blood flow distribution is not changed in CTEPH patients.

Sanz et al. (16) reported that the average blood velocity throughout the cardiac cycle was strongly correlated with pulmonary pressures and resistance. Garcia-Alvarez et al. (17) demonstrated a noninvasive method for the quantification of PVR, based on the measurement of right ventricular ejection fraction and average pulmonary artery velocity. In our study, not only mean velocity, but also peak velocity and mean blood flow of MPA were useful parameters in the evaluation of CTEPH. The progressive reduction in blood flow velocity through the pulmonary vascular bed with increasing of pulmonary artery pressures and PVR observed in the present study may be interpreted as an adaptive mechanism triggered to keep a constant pulmonary blood flow. So PVR is a sensitive parameter reflecting pulmonary artery pressures, especially critical in the assessment of patients with CTEPH, because of its importance in the prediction of potential candidates for pulmonary endarterectomy and postoperative outcome. In this study the mean flow of MPA demonstrated a higher negative correlation with PVRI than the peak and mean velocities. We speculated that MPA flow adaptation to chronic pressure overload was more sensitively determined by resistance. In addition, it was a good parameter reflecting right ventricular function. Our study also showed that the peak velocity of vena cava correlated negatively with PVRI, suggesting that PVRI was not only affected by obstruction of the pulmonary artery but was also associated with complicated pathophysiology secondary to chronic pulmonary embolism which was reflected by systemic circulation change.

In addition, the mean velocity of RPA correlated negatively with mPAP, sPAP, dPAP, and PVRI and the mean velocity of LPA had moderate negative correlation with dPAP. Compared with MPA and LPA, the mean velocity and mean blood flow of RPA may reflect pulmonary artery pressure changes all

around. This suggests that right pulmonary vessels are affected by pulmonary pressures and resistance more than the left pulmonary vessels, in patients with CTEPH. We estimated that the embolus incidence of right pulmonary artery was higher than left pulmonary artery, and our study also confirmed this.

Although the peak velocity of the proximal pulmonary artery could not reflect pulmonary pressures except for MPA, the peak velocity difference between RPA and RIPA may be a good parameter manifesting dPAP changes in CTEPH patients. The peak and mean velocities of IVC demonstrated moderate correlation with all the right heart catheterization-derived hemodynamic characteristics. So IVC hemodynamic characteristics may be an independent predictor for pulmonary artery pressure and resistance. Compared with IVC, moderate correlation between SVC velocity and central venous pressure suggests that central venous pressure changes could be reflected by SVC hemodynamic characteristics. Moreover, SVC mean velocity correlated with mPAP and dPAP, suggesting that SVC hemodynamic changes are more sensitive to dPAP than sPAP. This may be due to SVC filling the right atrium with blood during the diastole.

In our study, compared to the left pulmonary, the peak velocity difference between RPA and RIPA stayed inversely with dPAP, suggesting that right pulmonary arterial compliance decreases more apparently than that of left pulmonary (18). The respective correlation between the mean velocity difference of LPA, and LIPA, mPAP, sPAP demonstrated that mPAP, sPAP variability was mainly reflected in the lung with less embolus burden. This was consistent with CTEPH pathophysiology showing that resistance results from small vessel arteriopathy (1).

There are several limitations in our study. Only patients with CTEPH were included; thus, whether these results apply in other types of pulmonary hypertension requires further research. The examinations were not performed on the same day, which might influence the strength of the observed correlations. In order to reduce motion artifacts and acquisition time, we acquired phase-contrast MR images

during a breath-hold, whereas right heart catheterization was performed during free breathing. The possibility of magnetic field intensity affecting flow velocity could not be assessed. Total venous flow was noted to be less than total pulmonary flow. In addition to breath holding influence, this is probably the result of IVC flow underestimation, given the very small distance in the IVC between the right atrium and the insertion of the hepatic vein, and our inability to consistently acquire an MRI slice superior to the hepatic vein.

In conclusion, in patients with CTEPH, blood flow condition in the proximal pulmonary artery evaluated by phase-contrast MRI correlates with hemodynamic parameters of right heart catheterization; hemodynamic parameters of IVC are more useful indicators for estimating PVR and PAP than SVC; and the velocity of SVC is a better index reflecting right ventricular function. We believe these parameters can be used to noninvasively evaluate CTEPH severity and, potentially, to follow up the treatment response.

Conflict of interest disclosure

The authors declared no conflicts of interest.

References

1. Moser KM, Bloor CM. Pulmonary vascular lesions occurring in patients with chronic major vessel thromboembolic pulmonary hypertension. *Chest* 1993; 103:685–692.
2. Hoeper MM, Lee SH, Voswinckel R, et al. Complications of right heart catheterization procedures in patients with pulmonary hypertension in experienced centers. *J Am Coll Cardiol* 2006; 48:2546–2452.
3. Abel E, Jankowski A, Pison C, Luc Bosson J, Bouvaist H, Ferretti GR. Pulmonary artery and right ventricle assessment in pulmonary hypertension: correlation between functional parameters of ECG-gated CT and right-side heart catheterization. *Acta Radiol* 2012; 53:720–727.
4. Nogami M, Ohno Y, Koyama H, et al. Utility of phase contrast MR imaging for assessment of pulmonary flow and pressure estimation in patients with pulmonary hypertension: comparison with right heart catheterization and echocardiography. *J Magn Reson Imaging* 2009; 30:973–980.
5. Stevens GR, Fida N, Sanz J. Computed tomography and cardiac magnetic resonance imaging in pulmonary hypertension. *Prog Cardiovasc Dis* 2012; 55:161–171.
6. Mohiaddin RH, Wann SL, Underwood R, Firmin DN, Rees S, Longmore DB. Vena

- cava flow: assessment with cine MR velocity mapping. *Radiology* 1990; 177:537–541.
7. Cheng CP, Herfkens RJ, Lightner AL, Taylor CA, Feinstein JA. Blood flow conditions in the proximal pulmonary arteries and vena cavae: healthy children during upright cycling exercise. *Am J Physiol Heart Circ Physiol* 2004; 287:921–926.
 8. Galiè N, Hoeper MM, Humbert M, et al. Guidelines for the diagnosis and treatment of pulmonary hypertension: the task force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). *Eur Heart J* 2009; 30:2493–2537.
 9. Higgins CB, Roos AD. Cardiovascular MRI and MRA. Philadelphia: Lippincott Williams and Wilkins, 2003; 496.
 10. Mousseaux E, Tasu JP, Jolivet O, Simonneau G, Bittoun J, Gaux JC. Pulmonary arterial resistance: noninvasive measurement with indexes of pulmonary flow estimated at velocity encoded MR imaging—preliminary experience. *Radiology* 1999; 212:896–902.
 11. Jan S, Monda LS, Stephen M, et al. Pulmonary arterial hypertension: MR imaging-derived first-pass bolus kinetic parameters are biomarkers for pulmonary hemodynamics, cardiac function, and ventricular remodeling. *Radiology* 2012; 263:678–687.
 12. Dellegrottaglie S, Perrone-Filardi P, García-Alvarez A, et al. Serial phase-contrast MRI for prediction of pulmonary hemodynamic changes in patients with pulmonary arterial hypertension. *Int J Cardiol* 2012; 157:140–142.
 13. Markl M, Geiger J, Philip JK, et al. Time-resolved three-dimensional magnetic resonance velocity mapping of cardiovascular flow paths in volunteers and patients with Fontan circulation. *Eur J Cardiothorac Surg* 2011; 39:206–212.
 14. Sridharan S, Derrick G, Deanfield J, Taylor AM. Assessment of differential branch pulmonary blood flow: a comprehensive study of phase contrast magnetic resonance imaging and radionuclide lung perfusion imaging. *Heart* 2006; 92:963–968.
 15. Salim MA, DiSessa TG, Arheart KL, Alpert BS. Contribution of superior vena caval flow to total cardiac output in children: a Doppler echocardiographic study. *Circulation* 1995; 92:1860–1865.
 16. Sanz J, Kuschner P, Rius T, et al. Pulmonary arterial hypertension: noninvasive detection with phase-contrast MR imaging. *Radiology* 2007; 243:70–79.
 17. García-Alvarez A, Fernández-Friera L, Miralis JG, et al. Noninvasive estimation of pulmonary vascular resistance with cardiac magnetic resonance. *Eur Heart J* 2011; 32:2438–2445.
 18. Lankhaar JW, Westerhof N, Faes TJ, et al. Pulmonary vascular resistance and compliance stay inversely related during treatment of pulmonary hypertension. *Eur Heart J* 2008; 29:1688–1695.