Three-dimensional haemodynamics in patients with obstructive and non-obstructive hypertrophic cardiomyopathy assessed by cardiac magnetic resonance

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Aims
The left ventricular outflow tract (LVOT) peak pressure gradient is an important haemodynamic descriptor in patients with hypertrophic cardiomyopathy (HCM); however, secondary alterations in aortic blood flow have not been well described in these patients. Aortic flow derangement is not easily assessed by traditional imaging methods, but may provide unique characterization of this disease. In this study, we demonstrated how four-dimensional (4D) flow MRI can assess LVOT peak pressure gradients in HCM patients and also evaluated the ascending aorta (AAo) haemodynamic derangement associated with HCM.

Methods and results
Obstructive (n = 12) and non-obstructive (n = 18) HCM patients were included in the study along with 10 normal volunteers. 4D flow MRI was used to visualize three-dimensional (3D) blood flow patterns within the LVOT and AAo, which were graded for the presence of helical flow as a marker of flow derangement (absent = 0, mild/moderate = 1, and severe = 2). MRI-estimated pressure gradient (ΔP MRI) was calculated from the peak systolic 3D blood velocity profile within the LVOT. There was higher grade helical flow in obstructive HCM patients compared with non-obstructive patients (P = 0.04) and volunteers (P < 0.001). Non-obstructive patients also had higher helix grade than volunteers (P = 0.002). There was a significant correlation between helical grade and increasing ΔP MRI (rS = 0.69, P < 0.001). Systolic anterior motion (SAM) of the mitral valve was associated with both increasing helix grade (P < 0.001) and ΔP MRI (P = 0.006).

Conclusions
Ascending aorta flow derangement occurs in both obstructive and non-obstructive HCM patients and can be identified using 4D flow MRI. The degree of flow derangement correlates with LVOT gradient, SAM, and outflow tract geometry.

Keywords
Hypertrophic cardiomyopathy • Cardiac magnetic resonance imaging • Flow imaging

Introduction
Hypertrophic cardiomyopathy (HCM) is a heterogeneous, inherited disease of the myocardium affecting 1:500 people in the general population. The disease manifests as multiple phenotypes characterized by unexplained thickening of the left ventricular (LV) myocardium.1–4 A commonly diagnosed disease subtype is obstructive HCM that is associated with thickening of the interventricular septum and dynamic LV outflow tract (LVOT) obstruction, resulting in increased pressure gradients in the LVOT.5 Dynamic LVOT obstruction is caused by systolic anterior motion (SAM) of the mitral valve apparatus6–8 and is influenced by the complex interaction of a large number of patient-specific variables including outflow tract anatomy, myocardial contractility, and volume status.9,10 Additionally, aortic stiffness has been shown to be increased in HCM patients,10 and LVOT-to-ascending aorta (AAo) diameter ratio...
correlates with the outflow gradient\textsuperscript{11} even though these patients do not have dilated aortas,\textsuperscript{12} but the impact of these AAo changes on patient symptoms and outcomes has not been described.

Current haemodynamic assessment in HCM is limited to the assessment of peak LVOT gradient using echocardiography, two-dimensional cine phase-contrast MRI, or measured directly using cardiac catheterization.\textsuperscript{1,6,13–16} Flow derangement in the AAo is an increasingly recognized disease feature associated with valvular disease,\textsuperscript{17} and the outflow and aortic abnormalities associated with HCM are likely to induce similar AAo bulk flow variations that may relate to disease severity or risk. These aortic flow patterns cannot be well characterized using conventional two-dimensional (2D) imaging approaches, and consequently, the relationship between LVOT obstruction and abnormal three-dimensional (3D) flow features in the LVOT and AAo has not been investigated.

In the current study, we employed time-resolved, 3D, and phase-contrast MRI [four-dimensional (4D) flow MRI] to provide a comprehensive assessment of 3D blood flow and LVOT pressure gradient in HCM patients. This MRI technique can capture the full 3D velocity field in the heart and great vessels over the course of the cardiac cycle, which cannot be achieved using other imaging modalities.\textsuperscript{18} The aims of this study were to demonstrate the ability of 4D flow MRI to measure the peak LVOT pressure gradient in HCM, to explore the relationship between LVOT obstruction in HCM and 3D blood flow patterns in the LVOT and AAo, and to compare these findings to flow patterns in healthy control subjects.

**Methods**

**Study cohort**

We studied a cohort of consecutively recruited patients with HCM ($n = 30$) who had 4D flow MRI included as a part of physician-ordered cardiac MRI at Northwestern Memorial Hospital. Ten healthy control subjects with a similar age range and gender ratio were also included in the study. All patient data were de-identified and included under a protocol approved by the Institutional Review Board (IRB) of Northwestern University, while the control subjects were volunteers who provided signed, informed consent to undergo imaging in an IRB-approved protocol. The patients had all been diagnosed with the basal–septal HCM phenotype on previous clinical echocardiographic assessment using the criteria of LV septal wall thickness of $\geq 1.5$ cm in the absence of another explanation for this hypertrophy.\textsuperscript{2} Patients with any other HCM phenotype were excluded from this study. Included patients were classified as having obstructive HCM if their echocardiographic-assessed peak resting LVOT pressure gradient was $>30$ mmHg or were designated as having non-obstructive HCM if not meeting this gradient threshold. The average time interval between echocardiographic and MRI examinations in the HCM patients was $129 \pm 190$ days, with a median interval of 71 days. Beta-blockers are commonly prescribed to patients with HCM and are likely to influence outflow haemodynamics. Patient use of beta-blocker at the time of both echocardiographic and MRI assessment was assessed.

**Cardiac MRI**

All measurements were performed on 1.5-T ($n = 25$ subjects) or 3-T ($n = 5$ subjects) MR systems (Avanto, Aera, Skyra, Siemens, Germany). All patients underwent a standard-of-care thoracic cardiovascular MRI including ECG-gated time-resolved (CINE) cardiac MRI in multiple orientations (short-axis, LVOT, two-chamber, three-chamber, and four-chamber views) for the evaluation of cardiac function, myocardial wall thickness, SAM, and late gadolinium enhancement (LGE).

For the assessment of LVOT haemodynamics, time-resolved 3D phase-contrast MRI with three-directional velocity encoding (venc) (4D flow MRI) was employed.\textsuperscript{18} ECG and respiratory-gated 4D flow MRI were acquired in a three-dimensional volume angulated in three-chamber orientation to capture the left atrium, left ventricle, and ascending aorta (Figures 1 and 2). Pulse sequence parameters were as follows: spatial resolution $= (2.13–3.84 \times 2.13–4.06 \times 2.40–3.40)$ mm$^3$, temporal resolution $= 36.8–40.0$ ms, venc $= 150–250$ cm/s, echo time $= 2.2–2.5$ ms, repetition time $= 4.6–4.9$ ms, field of view $= 255–380 \times 234–450 \times 65–132$. Depending on heart rate and efficiency of respiratory navigator gating, total 4D flow MRI acquisition time was on the order of 8–15 min.

**Figure 1:** 4D flow visualization in patient with obstructive HCM. (A) 4D flow MRI-generated streamlines co-registered with TrueFISP cine MRI images, showing grade 2 helical flow in the AAo, mitral regurgitation (MR), and a thickened interventricular septum (asterisk) in a patient with obstructive HCM. (B) 4D flow velocity isosurface extracted from a cylindrical analysis volume in the LVOT of HCM patient. The arrow displays a notch in the velocity profile generated by the anterior leaflet of the mitral valve. LV, left ventricle; LVOT, left ventricular outflow tract.
Data collection and analysis
In patients and volunteers, maximal end-diastolic septal thickness and end-diastolic septum-free wall ratio were assessed by the short-axis steady-state free precession cine images. The average outflow diameter was defined as the mean of the end-diastolic LVOT diameter measured on both a three-chamber and LVOT TrueFISP cine MRI image. The presence of SAM was also evaluated by the three-chamber TrueFISP cine MRI. SAM was defined as the anterior systolic motion of the anterior leaflet of the mitral valve out of proportion to the normal motion of the mitral annulus and chordal structures.

3D blood flow visualization and quantification
Data preprocessing was performed and included noise filtering and correction for eddy currents, Maxwell terms, and velocity aliasing as previously described by Bock et al. Three-dimensional blood flow visualization and flow quantification were performed using the dedicated software (EnSight, CEI, Apex, NC). Peak flow velocity, defined as the maximum flow velocity over the cardiac cycle, was measured in a user-generated cylindrical 3D analysis volume covering the entire LVOT. Maximum velocity within the volume was queried at each time step throughout the cardiac cycle (Figure 1B). Peak MRI-estimated LVOT pressure gradient (ΔP MRI) was calculated based on the peak systolic velocity inside the 3D LVOT volume using the simplified Bernoulli equation: ΔP = 4v^2 (ΔP = peak pressure gradient and v = peak velocity).

Two-dimensional planes were positioned perpendicular to the direction of blood flow at the level of the mitral valve, in the LV apex, within the LVOT, and in the proximal AAo. Time-resolved pathlines were emitted from these planes to depict the three-dimensional path of blood flow over one cardiac cycle in the left ventricle, LVOT, and AAo (Figure 1A). The resulting 3D cine dataset was graded by two independent observers blinded to each other’s results (B.D.A. and P.v.O) for the presence of helical flow in the AAo using the scale for helix grade: no helical flow (grade 0), mild/moderate helical flow (grade 1), and severe helical flow (grade 2). All reported helix grades are the average of the two independent observers.

Results
Subject characteristics
Of the 30 HCM patients, 12 patients (40%) had outflow obstruction. Demographics of all patients and healthy controls are summarized in Table 1. Fifteen HCM patients (50%, non-obstructive: 9 patients and obstructive: 6 patients) were treated with a beta-blocker at the time of echocardiography, and two additional obstructive patients were started on a beta-blocker prior to MRI assessment for a total of 17 patients (57%) being treated at the time of MRI. As anticipated from experimental design, both obstructive and non-obstructive HCM patients had greater septal thickness (2.1 ± 0.4 and 1.9 ± 0.6 cm, respectively) relative to the normal volunteers (1.0 ± 0.1 cm, P < 0.001), but there was no difference between patient groups (P = 1.0). Non-obstructive patients had a greater septum/free wall ratio compared with volunteers (1.9 ± 0.9 vs. 1.1 ± 0.1, P = 0.03), whereas obstructive patients showed a non-significant trend towards an increased septum/free wall ratio compared with volunteers (1.8 ± 0.6, P = 0.09). There was no statistically significant difference in the average LVOT diameter between obstructive patients, non-obstructive patients, and volunteers (20.1 ± 3 vs. 21.8 ± 4 vs. 22.7 ± 2 mm, P = 0.22). In all subjects, 4D flow MRI...
datasets were of sufficient quality to allow for haemodynamic quantification and flow visualization.

**Haemodynamic quantification**

As anticipated, the obstructive cohort (defined by echocardiography) had higher $\Delta P_{MRI}$ relative to the volunteers ($54 \pm 29$ vs. $11 \pm 6$ mmHg, $P = 0.001$). However, the obstructive group was not significantly different from non-obstructive patients ($34 \pm 29$ mmHg, $P = 0.12$; Table 2). In patients, there was a moderate correlation of $\Delta P_{MRI}$ with average outflow diameter ($r = -0.43$, $P = 0.03$). No correlation was demonstrated with septal thickness ($r = 0.03$, $P = 0.86$) or septum/free wall ratio ($r = -0.22$, $P = 0.25$; Figure 3C). Including volunteers in the analysis resulted in a weak but significant correlation of $\Delta P_{MRI}$ with septal thickness ($r = 0.33$, $P = 0.04$).

**3D flow visualization and helix grading**

Obstructive HCM patients had higher grade helical flow than both non-obstructive patients ($1.6 \pm 0.4$ vs. $1.1 \pm 0.6$, $P = 0.04$) and volunteers ($0.3 \pm 0.48$, $P < 0.001$). Non-obstructive HCM patients also had higher grade helical flow than volunteers ($P = 0.001$; Table 2). Figure 4 displays the distribution of helical flow in the three subject groups. Considering only the HCM patients, there was a moderate, significant correlation between deranged LVOT flow (increased helical grade) and increasing $\Delta P_{MRI}$ ($r = 0.58$, $P = 0.001$). When including the volunteers, the correlation became even stronger ($r = 0.69$, $P < 0.001$). Figure 5 shows the distributions of $\Delta P_{MRI}$ within each helical grade that illustrates the correlation of flow derangement with $\Delta P_{MRI}$. Figure 3A shows the distribution of anatomical measurements within each subject subgroup, whereas Figure 3B shows the relationship of helix grade with anatomical features for the entire study population. In the combined cohort, a significant correlation between helical grade and septal thickness was observed ($r = 0.58$, $P < 0.001$), as well as a correlation between helical grade and septum/free wall ratio ($r = 0.38$, $P = 0.02$). There was a weak trend towards an increasing helical grade with a reduced average outflow diameter, but no significant correlation was found ($r = -0.29$, $P = 0.09$). No significant correlations of anatomical disease features with the helix grade were found in the patient-only group.

Interobserver variability for helix grading was moderate with an ICC of $0.62$ ($P < 0.001$).

**SAM subgroup analysis**

The presence of SAM with both $\Delta P_{MRI}$ ($r = 0.54$, $P = 0.002$) and helix grade ($r = 0.49$, $P = 0.006$) in the HCM patients. As a result, a

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**Table 1** Patient and volunteer characteristics

<table>
<thead>
<tr>
<th></th>
<th>Obstructive HCM [echo gradient $&gt; 30$ mmHg ($n = 12$, 9 males and 3 females)]</th>
<th>Non-obstructive HCM [echo gradient $&lt; 30$ mmHg ($n = 18$, 13 males and 5 females)]</th>
<th>Volunteers ($n = 10$, 6 males and 4 females)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>$56.7 \pm 12.7$</td>
<td>$51.1 \pm 16.1$</td>
<td>$54.8 \pm 8.96$</td>
<td>$0.03^{{}\dagger}$</td>
</tr>
<tr>
<td>Septal thickness (cm)</td>
<td>$2.1 \pm 0.35$</td>
<td>$1.9 \pm 0.61$</td>
<td>$1.03 \pm 0.1$</td>
<td>$0.09$</td>
</tr>
<tr>
<td>Septum/free wall ratio</td>
<td>$1.8 \pm 0.63$</td>
<td>$1.9 \pm 0.91$</td>
<td>$1.1 \pm 0.11$</td>
<td>$0.09$</td>
</tr>
<tr>
<td>Average outflow diameter (mm)</td>
<td>$20.1 \pm 2.65$</td>
<td>$21.8 \pm 3.9$</td>
<td>$22.66 \pm 1.78$</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>SAM positive (total numbers)</td>
<td>12</td>
<td>9</td>
<td>0</td>
<td>$&lt; 0.001$</td>
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$^{{}\dagger}$-values from ANOVA, all other intragroup $P$-values generated by the post hoc Bonferroni test.

**Table 2** Patient and volunteer haemodynamic comparison

<table>
<thead>
<tr>
<th>Results</th>
<th>Obstructive HCM [echo gradient $&gt; 30$ mmHg ($n = 12$, 9 males and 3 females)]</th>
<th>Non-obstructive HCM [echo gradient $&lt; 30$ mmHg ($n = 18$, 13 males and 5 females)]</th>
<th>Volunteers ($n = 10$, 6 males and 4 females)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI-estimated LVOT gradient (mmHg)</td>
<td>$53.8 \pm 29.2$</td>
<td>$33.6 \pm 28.6$</td>
<td>$10.5 \pm 5.58$</td>
<td>$0.12$</td>
</tr>
<tr>
<td>Helical grade</td>
<td>$1.6 \pm 0.38$</td>
<td>$1.1 \pm 0.64$</td>
<td>$0.3 \pm 0.48$</td>
<td>$0.04$</td>
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<td>$&lt; 0.001$</td>
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Figure 3. (A) Distribution of anatomical measurements in each cohort of the study. Horizontal black line—P < 0.05 between groups. (B) Distribution of anatomical measurements relative to the MRI-estimated gradient (ΔP). (C) Scatter plot of anatomical measurements relative to the MRI-estimated gradient. Asterisks denote statistically significant correlation.
subgroup analysis was performed comparing patients with SAM (SAM+) and without SAM (SAM−). Twenty-one patients were SAM+, whereas nine patients were SAM− (Table 3). The SAM+ group included all 12 patients in the obstructive HCM group and 9 (50%) in the non-obstructive HCM group. SAM+ patients had a higher $\Delta P_{MRI}$ (52 ± 28 mmHg vs. 17 ± 18, $P = 0.001$) and helix grade (1.5 ± 0.5 vs. 0.9 ± 0.6, $P = 0.009$) compared with SAM− patients and volunteers (gradient: 10.5 ± 6 mmHg, $P < 0.001$ and helix grade: 0.3 ± 0.48, $P < 0.001$). Of note, a trend suggesting a difference in helix grade was also observed between SAM− patients and volunteers ($P = 0.05$), even though no difference was found in $\Delta P_{MRI}$ between these groups ($P = 1.0$, but with a power of only 19.3% to detect this difference).

Discussion

In the current study, haemodynamic assessment using 4D flow MRI revealed deranged flow patterns in the AAo of both obstructive and non-obstructive HCM patients. Our findings also suggest that haemodynamic derangement in basal–septal HCM manifested by helical flow in the AAo is likely related to pressure changes across the LVOT, but also linked with SAM and, to a lesser degree, outflow tract geometry. It is important to note, however, that the modest correlations in our results do not support a causative relationship between any of the observed HCM disease features and flow derangement, suggesting that 3D haemodynamic alteration may reflect the combined effect of multiple disease characteristics.

Cardiac MRI has been shown to be useful for the assessment of the morphological and haemodynamic derangements secondary to obstruction as markers of disease, and also permits unique identification and quantification of myocardial scar using late gadolinium enhancement. Our current findings indicate that MRI has the additional unique potential to investigate the abnormal flow patterns that develop in both obstructive and non-obstructive HCM patients, opening up new areas for further research.

Given the complexity of the LVOT geometry and the dynamic nature of the obstruction secondary to SAM, techniques such as Doppler echocardiography or planar 2D phase-contrast MRI are less ideally suited for comprehensive haemodynamic assessment in patients with HCM. The advantage of 4D flow MRI in evaluating the LVOT and AAo of these patients is the ability of the technique to capture the complete haemodynamic environment within a user-prescribed 3D volume of interest. In the current study, this capability allowed for extensive haemodynamic assessment at an offline workstation after the scan was complete.

Our results suggest that the abnormal flow patterns observed in the LVOT and AAo are present in the majority of basal–septal HCM patients even without a significant LVOT gradient, although derangement is worse as the gradient increases. The idea of linking flow derangement with vascular or valvular disease is not new and has been invigorated by the increasing application of 4D flow MRI. Many studies have looked at qualitative and quantitative measures of helical flow as a marker of flow derangement in the ascending aorta of patients with bicuspid aortic valves, in ascending aortic aneurysms, as a result of surgical intervention, and in patients with congenital malformations. Our study builds on this body of work by applying 4D flow MRI flow assessment in HCM patients.

The observed helical flow pattern is likely secondary to an asymmetric high-velocity flow jet resulting from LVOT abnormalities, but the pathophysiological significance of this flow characteristic and how it may impact the aorta in HCM patients is uncertain. It is interesting to note that weak but significant correlations were observed between helical flow and outflow tract anatomical features (septal thickness, septum/free wall ratio, and outflow area) in the combined patient/volunteer cohort, but were no longer seen in the patient-only cohort. These anatomical features are used to diagnose HCM, and thus are fairly consistent across the patient group regardless of obstructive/non-obstructive status, and have not been shown to correlate with the LVOT pressure gradient in other studies. This finding further supports the limitations of these common structural measures for comprehensive disease characterization and highlights the
Although patients without SAM did not have elevated required to appropriately validate 4D flow MRI for this application. Techniques for the assessment of LVOT peak pressure gradient is measurements only as the diagnostic classification of obstructive or and our 4D flow results, and as such we used the clinical echo based on direct quantitative comparisons between echo findings and disease description, a quantitative measure of flow derangement may represent a unique marker of disease in HCM patients. While there is a strong interest in correlating our 4D flow findings with quantitative results from echocardiography, we intentionally did not include this analysis here because of the retrospective nature of comparison of echocardiographic and MRI data. It is well established that there is a high degree of variation between measured outflow tract gradients in patients depending on the time interval between measurements. In our study, we included the most recent echocardiographic data, but the median interval between scans was still over 2 months. Thus, we were not comfortable drawing conclusions based on direct quantitative comparisons between echo findings and our 4D flow results, and as such we used the clinical echo measurements only as the diagnostic classification of obstructive or non-obstructive HCM. A study prospectively comparing these two techniques for the assessment of LVOT peak pressure gradient is required to appropriately validate 4D flow MRI for this application.

Limitations
The current analysis is subject to several limitations. First, our definition of obstructive and non-obstructive HCM relied on a single echocardiographic measure of the peak LVOT gradient, and we did not consider additional data that can be used in diagnosing obstructive disease such as provokable gradients. We also did not attempt to control for medication use when selecting patients and two of the obstructive patients were started on a beta-blocker between echo and MRI assessment. Taken along with the haemodynamic variability associated with the large average time interval between echocardiography and MRI assessment, these potential confounders could lead to potential misclassification of obstructive and non-obstructive patients in our analysis and again necessitates a prospective study in this population. Also, the numbers of patients within each group are relatively small, but groups were large enough to detect statistically significant differences for the major outcomes of interest. A larger cohort would allow improved resolution of the data, and also allow for estimation of the sensitivity and specificity of our techniques.

The semi-quantitative nature of the helix grading system is subjective, and thus not ideal. Other studies have shown good inter- and intraobserver correlation for both quantitative and semi-quantitative 4D flow analyses, and our study shows moderate agreement between observers. However, for the purposes of risk stratification and disease description, a quantitative measure of flow derangement is needed. An advantage of 4D flow MRI is the ability to calculate velocity-related haemodynamic parameters such as helicity, wall shear stress, or energy loss. Correlating a repeatable, quantitative measure of flow disturbance with symptoms and disease characteristics in obstructive HCM should be the aim of future studies.

It is also important to recognize some limitations associated with the 4D flow technique. One drawback is the scan time required to acquire 4D flow data, as well as the post-processing required to allow for meaningful visualization and quantification. The technique uses a free-breathing, ECG- and respiratory-gated approach, so depending on the patient and volume of data being acquired, scan times can range from 5 to 20 min with post-processing times of 30–60 min. Also, particle trace visualization and flow quantification are highly dependent on the discretization that occurs based on the spatial and temporal resolution of the MRI acquisition. It is imperative to consider the impact of the trade-off between scan time and spatial/temporal resolution on the quality and accuracy of 4D flow MRI data.

**Conclusion**

Ascending aorta flow derangement occurs in patients with both obstructive and non-obstructive HCM, and can be identified using 4D flow MRI. This result is an initial step in developing a MRI protocol for HCM imaging that can provide comprehensive anatomical and haemodynamic data in a single study, but the clinical significance of

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<th>Table 3 SAM of the mitral valve subgroup analysis</th>
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<td><strong>SAM+ (n = 21, 12 males and 3 females)</strong></td>
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*P-values from ANOVA, all other intragroup P-values generated by the post hoc Bonferroni test.
the observed flow derangement in this cohort is currently unknown. Further studies exploring the relationships between 3D flow characteristics and patient symptoms and outcomes are warranted in order to establish the added value of 4D flow MRI in the evaluation of patients with HCM.

Conflict of interest: none declared.

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References