Restrictive right ventricular physiology after Tetralogy of Fallot repair is associated with fibrosis of the right ventricular outflow tract visualized on cardiac magnetic resonance imaging

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Aims To determine whether the restrictive physiology seen in Tetralogy of Fallot (TOF) patients can be explained by fibrosis of the right ventricular (RV) outflow tract. The aetiology for restrictive RV physiology after TOF repair is not known.

Methods and results TOF patients (n = 31, 13 girls, 10.2 years ± 2.8) were included 9.2 ± 2.9 years after total correction and examined with cardiac magnetic resonance (CMR) and Doppler echocardiography. Cine, flow, and late gadolinium contrast enhanced (LGE) CMR imaging were performed to quantify RV volumes, pulmonary flow and regurgitation (PR), and fibrosis. Healthy children (n = 12) were investigated with CMR of the pulmonary flow. Forward flow during atrial contraction above mean + 2 SD of healthy subjects was set as a marker of restrictive physiology. Four patients were excluded due to suboptimal LGE-CMR. Fisher’s exact test was used to determine the association between restrictive physiology and fibrosis. Sixteen patients showed fibrosis in the right ventricular outflow tract (RVOT) on LGE-CMR and 14 of them showed restrictive physiology on CMR. Of the 11 patients without fibrosis in the RVOT, 1 showed restrictive physiology. The odds ratio for RVOT fibrosis in patients with restrictive RV physiology was 70.0 (CI: 5.6–882.7, P < 0.001). The transannular patch repair did not differ between the groups (P = 0.37). The degree of RVOT fibrosis correlated positively with PR (r² = 0.38, P < 0.001) and RV volumes (r² = 0.51 for end-diastolic volume and r² = 0.47 for end-systolic volume, P < 0.001).

Conclusion There is a strong association between the restrictive RV physiology detected on CMR and fibrosis of the RVOT in children after TOF repair.

Keywords Cardiac pumping • Heart failure • Ventricular function

Introduction The mid- and long-term outcomes after Tetralogy of Fallot (TOF) repair are nowadays excellent and the quality of life among patients with a repair is generally good.¹-⁴ The age at repair has gradually decreased since the early era of TOF surgery without increase in mortality or the need for re-intervention.⁵-⁶ Furthermore, surgical techniques have improved, now with the ability to reduce the surgical trauma of the right ventricular outflow tract (RVOT) and minimize postoperative pulmonary regurgitation (PR).⁷ In some patients the diastolic properties of the right ventricular (RV) wall allow less compliance to diastolic filling. This RV diastolic dysfunction after TOF repair has been named restrictive RV physiology and is reflected by the occurrence of an end-diastolic forward
flow in the pulmonary artery (PA) detected by Doppler echocardiography. Restrictive RV physiology in the setting of significant PR has been shown to be of positive prognostic value at mid-term follow-up, but the pathophysiological explanation to restrictive physiology is so far unknown.

Cardiac magnetic resonance (CMR) imaging permits three-dimensional visualization of the RV throughout the cardiac cycle, enabling functional assessment with high accuracy. Furthermore, phase velocity-encoded CMR enables flow quantification of the PR with less observer dependency compared with Doppler echocardiography, and CMR can also be used to detect restrictive physiology. Thus, CMR has become a valuable tool in the assessment of patients operated for TOF. Late gadolinium contrast enhancement (LGE) CMR has been used to localize and quantify myocardial infarction and this technique has in recent years been shown to be able to visualize fibrosis of the RV in patients with congenital heart disease. However, no prospective study has used LGE-CMR in children with TOF to investigate the link to restrictive physiology.

Therefore, the aim of this study was to determine whether restrictive physiology seen in children after TOF repair can be associated with fibrosis of the RVOT. Furthermore, we aimed to investigate if there are differences in RV size and function in patients with fibrosis in the RVOT compared with patients without RVOT fibrosis at mid-term follow-up.

Methods

Subjects

Patients who had undergone TOF repair and with measurable PR on Doppler echocardiography were included. Patients with residual pulmonary stenosis pressure gradient > 25 mmHg on Doppler echocardiography, associated atrioventricular septum defect, double outlet right ventricle of Fallot type, pulmonary atresia with ventricular septum defect (VSD), TOF with absent pulmonary valve, or patients who had undergone pulmonary valve replacement (PVR) were excluded. Thirty-one patients (13 girls, mean age at investigation 10.2 ± 2.8 (range 3–16) years) were prospectively included at 9.2 ± 2.9 (range 2–16) years after surgical correction. The median age at repair was 9.5 ± 8.6 months (range 3 weeks to 1 year and 11 months). The study groups included patients surgically corrected < 6 months of age (n = 11) and > 6 months of age (n = 20).

The local institutional ethics committee approved the study, and parents gave informed consent to the children’s participation in the study. CMR and echocardiography examinations were performed within 2 days.

Echocardiography

Transthoracic echocardiography was performed by one observer (P.M.) using a GE Vingmed Vivid Five system with FPA 3.5, 5, and 10 MHz transducers. Echocardiographic measurements of restrictive RV physiology were performed as previously described. In short, restrictive RV physiology was defined as forward pulmonary flow in late diastole present throughout the respiratory cycle (Figure 1).

CMR

The CMR protocol and parameters are listed in the Appendix. CMR was performed on a 1.5 T Philips Intera CV (n = 27) and a 1.5 T Siemens Magnetom Vision (n = 4). Cine, pulmonary flow velocity mapping, and LGE CMR images were acquired. Cine images were acquired in the RVOT plane, oblique transverse plane, and/or the left ventricular short-axis plane in order to determine the RV end-diastolic volume (EDV) and end-systolic volume (ESV) as well as the RV ejection fraction (EF). Flow velocity mapping CMR was performed during free breathing perpendicular to the plane of the flow in the PA to determine the regurgitant volume and regurgitant fraction (RF).

Restrictive physiology using CMR

Forward flow during atrial contraction was used to detect restrictive physiology. In normal subjects, there is a slight forward flow during late diastole in flow quantification using CMR, caused by the movement of the pulmonary valve during atrioventricular displacement. Therefore, forward flow as a percentage of the net forward flow during the cardiac cycle (Figure 1) was calculated in 12 healthy children (15 ± 3 years) with normal CMR referred for screening of arrhythmogenic right ventricular cardiomyopathy because of a family history of this disease. A threshold of mean ± 2 SD of the percentage of forward flow during atrial contraction in healthy subjects was set as normal and values above this as restrictive physiology. The percentage of forward flow in TOF patients was defined as a forward flow in the PA during atrial contraction divided by the net (effective) flow (forward minus backward flow) (Figure 1). In a separate analysis, patients were classified as having restrictive and non-restrictive physiology using visual assessment where any peak of forward flow prior to the systolic forward flow in the PA was considered as restrictive physiology.

LGE-CMR, was obtained for fibrosis visualization in the same plane as for cine CMR. Images were acquired 10–20 min after i.v. administration of Gadolinium (Gd)-based contrast agent (Gd-DOTA or Gd-DTPA, 0.2 mmol/kg body weight).

Image analysis

Echocardiographic images were analysed without the knowledge of CMR findings and vice versa. The echocardiographic images were evaluated by vendor-provided software in the scanner. CMR data were analysed using the software Segment v1.8 (http://segment.heiberg.se). The endocardial contours were drawn on cine CMR to calculate the RV end-diastolic volume (RVEDV), RV end-systolic volume (RVESV), and RV ejection fraction (RVEF). The right atrial (RA) size in end-systole was delineated in cine CMR to provide an indication of RV diastolic pressures. The PA was segmented on flow CMR to quantify the pulmonary regurgitant volume as the backward volume in diastole and the RA as a per cent of forward flow. LGE-CMR was visually scored by two experienced observers (M.C. and H.A.) in consensus. The RV was divided into three short-axis and three transverse regions giving a total of nine segments as previously described. Each segment was given a score from 0 to 4: no fibrosis was given the score 0, 1–25% segmental fibrosis 1, 26–50% 2, 51–75% 3, and 76–100% 4. Fibrosis had to be present in two different imaging planes to be considered a true finding. Per cent fibrosis of the RV was calculated as (total score/36) × 100%.

Statistical analysis

All continuous variables were expressed as mean ± SD. The Mann–Whitney test was used to compare continuous variables in the restrictive vs. non-restrictive RV physiology groups and fibrotic vs. non-fibrotic groups. Fisher’s exact test was used when comparing non-continuous variables between the groups and the odds ratio (OR) with confidence interval (CI) was calculated. Pearson’s correlation analysis was used.
performed between the degree of RVOT fibrosis and the RVEDV, RVESV, RVEF, and regurgitant volume. Results with a P-value <0.05 were considered significant.

Results

Restrictive RV physiology
The percentage of end-diastolic forward flow of the total flow was 1.10 ± 0.71% in healthy subjects and therefore the upper normal limit was 2.5%. Sixteen patients had end-diastolic forward flow ≥2.5% and was considered to have restrictive physiology using CMR. The diastolic forward flow as a percentage of stroke volume in all patients was 4.7 ± 6.3%, in patients with restrictive physiology 9.1 ± 5.4%, and in patients with non-restrictive physiology −0.5 ± 2.9%. Most patients with restrictive physiology (n = 14) demonstrated a negative PA flow at the onset of the distinct end-diastolic flow wave as exemplified in Figure 1C. On Doppler echocardiography 15 patients were found to be restrictive. Ten of these were restrictive also on CMR. On Doppler echocardiography 16 patients were non-restrictive. Ten of these were non-restrictive on CMR. The kappa value between CMR and Doppler echocardiography for restrictive physiology was 0.29; the OR was not significant (OR: 3.3, CI: 0.8–14.6, P = 0.16).

Restrictive RV physiology and RV myocardial fibrosis
Four patients were excluded from the fibrotic vs. non-fibrotic analysis due to inadequate or incomplete LGE-CMR acquisition.
Fibrosis of the RVOT on LGE-CMR (Figures 2 and 3) was found in 16 out of 27 (59%) investigated patients. There was a strong association between RVOT fibrosis on LGE-CMR and restrictive RV physiology on CMR (OR: 70, CI: 5.6–882.7, \( P < 0.001 \)) (Table 1). Fourteen of the 16 patients with RVOT fibrosis showed restrictive RV physiology and only one of the 11 patients without RVOT fibrosis showed restrictive RV physiology. Fifteen of the patients with fibrosis had restrictive physiology when using visual assessment and five of the non-fibrotic patients had non-restrictive physiology. The OR for visual assessment of restrictive physiology on echocardiography (OR: 4.4, CI: 0.8–24.0, \( P = 0.12 \)). Three of the patients without fibrosis showed restrictive physiology on CMR (OR: 70, CI: 5.6–882.7, \( P < 0.001 \)) (Figure 4). The RA size was larger in the group with fibrosis (\( P = 0.046 \)), indicating higher RV diastolic pressure (Table 2).

Patients with restrictive physiology had larger RVEDV/BSA (159 ± 49 mL/m², \( P = 0.003 \)) and RVESV/BSA (83 ± 34 mL/m², \( P = 0.008 \)), and higher RF (45 ± 9%, \( P = 0.003 \)) compared with patients with non-restrictive physiology on CMR (111 ± 29 mL/m², 52 ± 18 mL/m², 23 ± 19%) (Table 3).

**Discussion**

This study is the first to show that there is a strong association between restrictive RV physiology and fibrosis of the RVOT in children after TOF repair with residual postoperative PR. RVOT fibrosis correlated positively to RV volume and PR but did not correlate to TAP repair.

**Fibrosis and restrictive physiology**

Fibrosis of the RVOT was associated with restrictive physiology assessed by CMR and the link may be that the fibrosis decreases the RV compliance. In the case of an RV with low compliance, at atrial systole blood will be pumped against a stiff RV, resulting in forward pulmonary flow in ventricular diastole. Only 1 out of 15 patients with restrictive physiology showed no sign of fibrosis in the RV. Fibrosis was mainly found in the anterior free wall of the RVOT. Furthermore, fibrosis in the region of the VSD repair was seen in most patients and in the inferoseptal RV insertion points or in RV trabeculae in a minority of patients. Previous studies in adults have found RVOT fibrosis in 71–99% after primary repair of TOF.21,22 in line with our findings. Extensive RV fibrosis (≥75th percentile) has earlier been associated with restrictive RV physiology in adults.17 In our study in children, we found a clear association with restrictive RV physiology even when including milder degrees of RVOT fibrosis. This difference may be attributed to the younger population in our material as has been previously proposed.17 In our population patients with fibrosis also had a larger RA size, indicating higher RA pressure and therefore higher RV diastolic pressure. Restrictive physiology was

**Table 1**  Relationship between restrictive RV physiology detected on CMR and RVOT fibrosis detected on LGE-CMR

<table>
<thead>
<tr>
<th></th>
<th>Patients with fibrosis (( n = 16 ))</th>
<th>Patients with no fibrosis (( n = 11 ))</th>
<th>( P &lt; 0.001, OR = 70.0 ) (CI: 5.6–882.7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restrictive RV physiology detected on CMR (( n = 15 ))</td>
<td>14</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Non-restrictive RV physiology detected on CMR (( n = 12 ))</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>
### Table 2  Patient characteristics in patients with and without fibrosis

<table>
<thead>
<tr>
<th></th>
<th>All patients (n = 27)</th>
<th>Patients with fibrosis in the RVOT (n = 16)</th>
<th>Patients without fibrosis in the RVOT (n = 11)</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>10.2 ± 2.6</td>
<td>10.5 ± 2.5</td>
<td>9.8 ± 2.8</td>
<td>0.73</td>
</tr>
<tr>
<td>Follow-up time (years)</td>
<td>9.2 ± 2.7</td>
<td>9.3 ± 2.7</td>
<td>9.0 ± 2.7</td>
<td>0.98</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.2 ± 0.3</td>
<td>1.2 ± 0.3</td>
<td>1.1 ± 0.3</td>
<td>0.69</td>
</tr>
<tr>
<td>Females (%)</td>
<td>48</td>
<td>50</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Age at repair (years)</td>
<td>0.8 ± 0.8</td>
<td>0.9 ± 0.9</td>
<td>0.7 ± 0.4</td>
<td>0.29</td>
</tr>
<tr>
<td>Repair &lt; 6 months (%)</td>
<td>37</td>
<td>31</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>TAP (%)</td>
<td>52</td>
<td>56</td>
<td>36</td>
<td>0.37</td>
</tr>
<tr>
<td>RVEDV, mL/m²</td>
<td>134 ± 50</td>
<td>158 ± 47</td>
<td>100 ± 31</td>
<td>0.002</td>
</tr>
<tr>
<td>RVESV, mL/m²</td>
<td>67 ± 33</td>
<td>83 ± 33</td>
<td>44 ± 17</td>
<td>0.002</td>
</tr>
<tr>
<td>RV EF, %</td>
<td>52 ± 7</td>
<td>49 ± 8</td>
<td>56 ± 5</td>
<td>0.18</td>
</tr>
<tr>
<td>RF, mL</td>
<td>29 ± 24</td>
<td>36 ± 23</td>
<td>17 ± 22</td>
<td>0.02</td>
</tr>
<tr>
<td>RF, mL/BSA</td>
<td>23 ± 16</td>
<td>30 ± 14</td>
<td>12 ± 17</td>
<td>0.02</td>
</tr>
<tr>
<td>RF, %</td>
<td>33 ± 19</td>
<td>40 ± 16</td>
<td>22 ± 19</td>
<td>0.02</td>
</tr>
<tr>
<td>RA, mL/m²</td>
<td>46 ± 12</td>
<td>50 ± 12</td>
<td>40 ± 12</td>
<td>0.046</td>
</tr>
<tr>
<td>Transatrial approach (%)</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>1.0</td>
</tr>
<tr>
<td>Cross-clamp time (min)</td>
<td>49 ± 17</td>
<td>49 ± 10</td>
<td>49 ± 25</td>
<td>0.32</td>
</tr>
<tr>
<td>Hospital stay (days)</td>
<td>13 ± 6</td>
<td>14 ± 7</td>
<td>12 ± 5</td>
<td>0.50</td>
</tr>
<tr>
<td>Need for PVR surgery on follow-up</td>
<td>41</td>
<td>56</td>
<td>18</td>
<td>0.07</td>
</tr>
</tbody>
</table>

**Figure 4** Correlation between the degree of fibrosis and (A) right ventricular ejection fraction (RVEF), (B) right ventricular end-diastolic volume (RVEDV) indexed to BSA, (C) right ventricular end-systolic volume (RVESV) indexed to BSA, and (D) pulmonary regurgitant volume.
originally described as patients with end-diastolic forward flow through the pulmonary valve during atrial contraction due to a stiff and non-compliant hypertrophied RV wall. Hence, RV restrictive physiology in the early and mid-term postoperative period protects the RV from dilatation and minimizes the effects of PR and this may be the reason why postoperative PR is well tolerated initially after TOF repair. In our patient population, at 9.2 ± 2.9 years after repair, we found end-diastolic forward flow through the pulmonary valve in patients with fibrosis of the RVOT coupled to higher PR and larger RV volumes. Furthermore, we found a positive correlation with the degree of fibrosis and RV volumes in line with previous studies. This indicates that the protective nature of restrictive RV physiology early after TOF repair previously described may attenuate with time, and fibrosis development may represent the substrate for the second form of restrictive RV physiology suggested by Lee et al. Therefore, the aetiology of the restrictive physiology in our patient population may be different from the ‘classical’ description. Lee et al. divided the patient groups according to restrictive physiology and RV volumes, and found that forward end-diastolic flow was present in both large and small RVs but was associated with better physical exercise tolerance only in conjunction with small RVs. This could explain why some previous studies have found restrictive physiology to be a positive prognostic factor and other studies have shown that restrictive physiology is coupled to a reduced RVEF and low quality of life.

We found slightly higher end-diastolic forward flow in restrictive patients (9% of SV) compared with earlier studies (6%). and this is probably explained by different definitions of restrictive physiology between the studies. We used 2.5% end-diastolic forward flow as a cut-off for restrictive physiology based on the forward flow seen in healthy children due to longitudinal AV-plane movement during atrial contraction.

The association of restrictive physiology and RV fibrosis in our patient population does not necessarily mean that RV fibrosis is the cause of restrictive physiology. However, our findings show that restrictive physiology found at follow-up in patients with large RV volumes and RF is strongly associated with myocardial fibrosis in the RVOT. The agreement between CMR and Doppler echocardiography was fair (kappa value 0.29), which is similar to the findings of Lee et al. (kappa value 0.35 calculated from the published results). A possible explanation for this difference is that CMR averages several cardiac and respiratory cycles and can therefore be viewed as more inclusive for restrictive physiology. CMR has been proposed to be more accurate compared with Doppler in the detection of restrictive physiology in dilated pulmonary arteries lacking laminar flow patterns.

### Cause of fibrosis

The cause of RVOT fibrosis is not clear, but several mechanisms have been proposed, such as the long-term effects of preoperative hypoxaemia. Dilatation of the RV caused by PR has been postulated to be one cause of progressive fibrosis and we have showed a positive correlation between the degree of RVOT fibrosis and PR as well as RV dilatation (Figure 4). However, the cause and effect relationship could be reverse with RV fibrosis causing RV dilatation and thereby worsening of the PR. Wald et al. showed that the fibrosis is associated with dys-/akinesia, aneurysmatic dilatation of the RVOT and conduction delay. The PR volume is mainly determined by the size of the pulmonary valvular orifice, which may become enlarged in RVOT fibrosis as the pulmonary annulus is connected to the RVOT myocardium.

One possible mechanism for RVOT fibrosis may be that the muscular resection of the infundibulum at repair imposes damages to the microvasculature causing fibrosis development and thereby contributes to restrictive RV physiology. All patients in our study were repaired via a transtorial or transpulmonary approach with only minimal ventricular incisions made in patients with TAP with the aim of preserving the RV infundibulum. Interestingly, in the study by Wald et al. fibrosis was found extending to the anterior RV free wall and neighbouring segments where surgery had not taken place and this was also found in our study. Therefore, it is possible that fibrosis caused by RV volume overload may co-exist with the surgically related substrates for fibrosis in RVOT creating RV dysfunction. There was no difference in fibrosis development or restrictive RV physiology between the early and late repaired group in the present study and this may be attributed to an over-all earlier repair age compared with previous studies.

There were no differences in cross-clamp times at repair between the fibrotic and the non-fibrotic groups. This indicates that prolonged myocardial hypoperfusion during surgery is not a cause for fibrosis. Consequently, neither long-term

### Table 3 Patient characteristics in patients with and without restrictive physiology detected on CMR

<table>
<thead>
<tr>
<th></th>
<th>All patients (n = 31)</th>
<th>Patients with restrictive physiology (n = 16)</th>
<th>Patients without restrictive physiology (n = 15)</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVEDV/BSA, mL/m²</td>
<td>136 ± 47</td>
<td>159 ± 49</td>
<td>111 ± 29</td>
<td>0.003</td>
</tr>
<tr>
<td>RVESV/BSA, mL/m²</td>
<td>68 ± 31</td>
<td>83 ± 34</td>
<td>52 ± 18</td>
<td>0.008</td>
</tr>
<tr>
<td>RVSV/BSA, mL/m²</td>
<td>68 ± 18</td>
<td>76 ± 19</td>
<td>59 ± 13</td>
<td>0.005</td>
</tr>
<tr>
<td>RVEF</td>
<td>52 ± 7</td>
<td>49 ± 8</td>
<td>54 ± 6</td>
<td>0.07</td>
</tr>
<tr>
<td>RF, mL</td>
<td>29 ± 22</td>
<td>39 ± 21</td>
<td>18 ± 20</td>
<td>0.005</td>
</tr>
<tr>
<td>RF/BSA, mL/m²</td>
<td>25 ± 16</td>
<td>33 ± 11</td>
<td>16 ± 16</td>
<td>0.003</td>
</tr>
<tr>
<td>RF, %</td>
<td>35 ± 18</td>
<td>45 ± 9</td>
<td>23 ± 19</td>
<td>0.003</td>
</tr>
</tbody>
</table>
effects of hypoxaemia, TAP or ventriculotomies are likely to be the cause of the RVOT fibrosis in children after TOF repair.

Interestingly, we did not find any LGE in four patients with TAP. In our material, the patch material used for TAP consisted of the pericardium and the size was quite small (5–10 mm including the supravalvular and subvalvular parts), which may explain why CMR ‘missed’ to detect LGE in this region.

Limitations

The lack of fibrosis in the RVOT on LGE-CMR does not exclude the existence of a more diffusely spread fibrosis throughout the entire RV wall that was not detected. LGE-CMR lacks spatial resolution to detect very small areas of fibrosis and the use of an inversion-recovery pulse does not permit the detection of evenly distributed diffuse intercellular fibrosis. Further studies using high-resolution T1-mapping to quantify the extracellular volume fraction may provide information on the relationship between increased amount of diffuse fibrosis and restrictive physiology. In this study, fibrosis detection was performed by two experienced observers in consensus with visual scoring. Better image quality with higher resolution and better signal-to-noise ratio would be needed for a semi-automatic quantification as used in left ventricular infarct quantification. The number of patients in the study is fairly limited, which may explain why we did not find a statistically significant relationship between restrictive physiology on Doppler echocardiography and fibrosis on LGE-CMR and the comparisons of cross-clamp times.

Conclusion

To our knowledge this study is the first showing an association between restrictive RV physiology and RVOT fibrosis visualized on LGE-CMR in children repaired for TOF. The cause for fibrosis still remains unclear and needs to be further addressed in future studies.

Acknowledgements

Annica Maxedius is greatly appreciated for help with patient administration. This study was supported by a grant from the Swedish Heart Lung foundation.

Conflict of interest: none declared.

Appendix: MR sequence parameters

Cine

Philips: A steady-state free-precession sequence with retrospective ECG triggering was used with acquired temporal resolution of typically 47 ms reconstructed to 25 ms, echo time 1.4 ms, flip angle 60°, and a slice thickness of 8 mm. Siemens: A gradient-echo sequence with prospective ECG triggering was used with typically 15 phases per cardiac cycle, with acquired temporal resolution of 100 ms reconstructed using echo sharing reconstructed to every 50 ms, echo time 4.8 ms, flip angle 30°, and a slice thickness of 10 mm. Breath-hold times were typically 15 s.

Flow

Philips: A fast field echo velocity-encoded sequence with retrospective ECG triggering was used with repetition time 10 ms, echo time 5 ms, flip angle 15°, slice thickness 6 mm, 35 phases, number of acquisitions 1, no parallel imaging, and a velocity-encoding gradient (VENC) of 200 cm/s. Siemens: Imaging parameters were the same as above except that the images were obtained with prospective ECG triggering. Velocity information was acquired over two heartbeats to quantify the flow during the end of diastole. The flow sequences were non-segmented without echo sharing with an acquired temporal resolution of 20 ms for Philips and 35 ms for Siemens.

Late gadolinium enhancement

Philips: An inversion-recovery balanced turbo field echo sequence with a slice thickness of 8 mm field of view 340 mm, matrix 126 × 256, repetition time 3.14 ms, and echo time 1.58 ms was used. Siemens: An inversion-recovery turbo fast low-angle shot sequence with a slice thickness of 10 mm, field of view 380 mm, matrix 126 × 256, flip angle 25°, repetition time 100 ms, and echo time 4.8 ms was used.

The inversion time was manually adjusted to null the signal from the non-fibrotic myocardium and images were acquired at end-diastole.

References


