

# Structured reporting of cardiovascular magnetic resonance based on expert consensuses and guidelines

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## Abstract

**Background:** There is a gap between China and developed countries in Europe and America as to the normalization of cardiac magnetic resonance (CMR) diagnostic reports. The aim of this study was to construct a structured CMR report template suitable for China's actual conditions.

**Methods:** Cardiac magnetic resonance standardized image interpretation and post-processing guidelines and CMR report guidelines are the consensus and recommendations of the Society for Cardiovascular Magnetic Resonance (SCMR) experts whose goal is to ensure the consistent quality and repeatability of CMR reports. This structured CMR report template was constructed based on the guidelines for standardized image interpretation, post processing and reporting of CMR examinations, combined with the experiences learned in Germany and the practical experiences in China. It consisted of three parts: Device and Methods, Findings (Structure and function, Tissue Characterization), Summary and Conclusion. Detailed directions were provided section by section.

**Results:** This structured CMR report template underlined the comprehensive analysis of the results of morphological, functional and tissue characteristics to provide conclusive opinions and answer the corresponding important questions raised by the clinicians.

**Conclusion:** The standardization of qualitative and quantitative assessments of CMR results is the core of structured reporting of CMR.

## KEYWORDS

cardiovascular disease (structured reporting), cardiovascular magnetic resonance, report quality

## 1 | INTRODUCTION

Cardiac function is altered in an age-related manner and cardiovascular diseases increase with age worldwide. Cardiovascular

disease is the leading cause of death of the elderly in China, causing more deaths than tumors or any other diseases.<sup>1</sup> Cardiovascular magnetic resonance imaging (CMR) is a non-invasive imaging technique that offers multi-parameter and multi-directional

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information of the cardiovascular system and has become the gold standard method for the assessment of cardiac structure and function.<sup>2</sup> CMR is now receiving more and more attention and is widely used in clinical work because of its irreplaceable value in the etiological diagnosis, assessment of disease severity, and prognosis of cardiovascular disease.

However, some problems prevent this technology from becoming widely available in China; for example, it requires highly skilled technicians, and costs more and takes longer to perform than other imaging methods. Irregularities in image post-processing and diagnostic report of CMR are common in China at present. Many CMR reports have mainly focused on morphologic abnormalities and qualitative diagnosis but have ignored cardiac dysfunction and quantitative diagnosis, leading to diagnoses that were incomplete or even unreliable, and that could not be recognized by clinical staff.

To improve the quality of CMR reports, the authors construct a structured report template for CMR based on the guidelines for standardized image interpretation,<sup>3</sup> post-processing, and reporting of CMR examinations.<sup>4</sup> This work is also combined with the authors' experiences of a clinical visit to Germany and practical work after returning to China.

## 2 | INTERPRETATION OF CMR STRUCTURED REPORT

This structured report takes into account both qualitative and quantitative diagnosis, and is divided into three parts: (a) Device and Methods, (b) Findings (Visual Assessment + Quantitative Analysis), and (c) Summary and Conclusion. When using this report template, radiologists should have knowledge of relevant magnetic resonance physics, equipment, safety, cardiovascular anatomy and pathophysiology, CMR scanning and image post-processing technology, and diagnosis and treatment of cardiovascular disease.

## 3 | DEVICE AND METHODS

The Device and Methods section should illuminate the scanning device, sequences, vasodilator, and contrast agent used in this examination (Figure 1):

- Device: Field strength/model, for example: "3.0 T Prisma (Siemens)";
- Cardiac structure and function: Short-axis and long-axis cine imaging; and
- Histological characteristics:
  - a. T1 Mapping + T2 Mapping/ T2 weighted imaging (T2WI);
  - b. First-pass perfusion scanning (stress/rest, vasodilator: trade name, usage; contrast agent: trade name, usage); and
  - c. Late gadolinium enhancement (scars and fibrosis).

## 4 | FINDINGS (VISUAL EVALUATION + QUANTITATIVE ANALYSIS)

### 4.1 | Structure and function

Professional CMR post-processing software (Syngo.via, Medis, CVI 42, etc) should be used for image post-processing, including the following main steps: (a) Measure left ventricle (LV) wall thickness and LV mass in end-diastole (Figure 2). (b) Accurately outline the endocardium and epicardium in end-diastole and end-systole to get the absolute and indexed values of LV and right ventricle (RV) function parameters, including ejection fraction (EF), end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), cardiac output (CO), and LV mass (Figure 3). Indexed values are obtained by dividing the absolute value by the patient's body surface area (BSA). The BSA is obtained from the patient's height and weight data through formula calculations:  $BSA (m^2) = 0.0061 \times \text{height (cm)} + 0.0128 \times \text{weight (kg)} - 0.1529$  (Figure 4). (c) Measure the area of the left atrium and right atrium in the end-systole (Figure 2). To determine whether it is abnormal, the observer should use BSA indexed values. The ranges of normal values reported in Schulzmenger et al<sup>3</sup> and Hudsmith et al<sup>5</sup> were used to interpret the results, which are affected by sex and age.

#### 1. Left ventricle

- Left ventricular volume: An enlarged LV volume is confirmed if the end-diastolic LV EDV is larger than the normal range of the corresponding sex and age.
- Ventricular wall motion: Global hyperkinesia and regional wall motion abnormalities should be noted and categorized as hyperkinetic, hypokinetic, akinetic, or dyskinetic. The location of regional wall motion abnormalities should be specified by use of the 17-segment model adopted by the American College of Cardiology/American Heart Association guidelines.<sup>6</sup> At the same time, reduced LV long-axis function, aneurysm or pseudoaneurysm, or dyssynchrony should be detected.
- Global LV systolic function: A reduced global LV systolic function is confirmed by comparing the result of LVEF with the normal range of the corresponding sex and age, and the grade is determined by the severity of the LVEF reduction: mildly reduced (EF = 45%-50%), moderately reduced (EF = 36%-45%), or severely reduced (EF < 35%).
- LV mass: LV mass is obtained by accurately drawing the endocardial and epicardial outline of myocardium in end-diastolic short-axis cine images. An enlarged LV mass is identified by comparing the value of indexed LV mass with the normal range of the corresponding sex and age.
- LV wall thickness: Interventricular wall thickness (IVS) and posterior wall dimensions (PWD) are measured in the end-diastolic three-chamber (3CH) long-axis cine image (Figure 2b). The normal range of PWD and IVS is 8-11 mm, and LV wall thickening is identified when PWD and/or IVS is greater than 11 mm. When the LV wall is diffusely thickened, it is necessary to measure the

Xiangya Hospital Central South University  
**MRI diagnostic report**

**Name:** \*\*\*      **Gender:** Male      **Date of Birth:** \*\*\*      **Age:** 28y

**Application department:** \*      **Patient ID:** \*      **Examination number:** \*

**Examination item:** Cardiac magnetic resonance imaging

**Clinical diagnosis:** Chest pain

**TECHNIQUE:**

Equipment: 3.0T Prisma (SIEMENS), Short and long axis cine images (function and structure), T1 mapping, T2 mapping, First-pass perfusion images (contrast agent: Gadobenate Dimeglumine 0.1 mmol/kg), Delayed-enhanced images (scars and fibrosis)

**FINDINGS:**

1. MORPHOLOGY AND FUNCTION:

i) LEFT VENTRICULAR WALL THICKNESS AND MASS: Normal

IVSd: 8 mm, LVPWd: 5 mm, LV mass: 110 g/m<sup>2</sup>, LV mass index: 78 g/m<sup>2</sup>

ii) WALL MOTION: No evidence of regional wall motion abnormalities.

iii) VOLUME AND FUNCTION

a) LEFT VENTRICLE: Normal LV volume; Good LV global systolic function.

ED volume: 153.54 ml (115 - 231 ml)      ED volume index: 107.95 ml/m<sup>2</sup> (68 - 112 ml/m<sup>2</sup>)

ES volume: 59.3 ml (27 - 87 ml)      ES volume index: 41.69 ml/m<sup>2</sup> (16 - 44 ml/m<sup>2</sup>)

Stroke volume: 94.24 ml (82 - 154 ml)      Stroke volume index: 66.26 ml/m<sup>2</sup> (44 - 76 ml/m<sup>2</sup>)

Cardiac output: 5.28 l/min (4 - 8 l/min)      Cardiac output index: 3.71 l/min/m<sup>2</sup> (2.6 - 4.2 l/min/m<sup>2</sup>)

Ejection fraction: 61.38 % (57 - 77 %)

b) RIGHT VENTRICLE: Normal RV volume; Good RV global systolic function.

ED volume: 160.32 ml (137 - 269 ml)      ED volume index: 112.72 ml/m<sup>2</sup> (74 - 134 ml/m<sup>2</sup>)

ES volume: 67.86 ml (47 - 127 ml)      ES volume index: 47.71 ml/m<sup>2</sup> (26 - 62 ml/m<sup>2</sup>)

Stroke volume: 92.46 ml (78 - 154 ml)      Stroke volume index: 65.01 ml/m<sup>2</sup> (41 - 77 ml/m<sup>2</sup>)

Cardiac output: 5.18 l/min (4 - 8 l/min)      Cardiac output index: 3.64 l/min/m<sup>2</sup> (2.6 - 4.2 l/min/m<sup>2</sup>)

Ejection fraction: 57.67 % (47 - 67 %)

iv) LVOT AND AORTIC ROOT: Normal

v) ATRIUM: Normal

LA 21 cm<sup>2</sup> (< 24 cm<sup>2</sup>) RA 20 cm<sup>2</sup> (< 23 cm<sup>2</sup>)

2. TISSUE CHARACTERIZATION

i) EDEMA, INFLAMMATION AND DIFFUSE FIBROSIS: Normal native T1 and T2 values.

ii) PERFUSION: No evidence of relevant hypoperfusion.

iii) DELAYED ENHANCEMENT: No evidence of myocardial LGEs.

3. VALVES AND PERICARDIUM

No evidence of valve disease and pericardial effusion.

**IMPRESSION:**

Normal biventricular volumes and good global systolic function. Normal LV mass and wall thickness. No evidence of regional wall movement disorders. No evidence of relevant hypoperfusion. No evidence of scar or necrosis. No evidence of myocardial diffuse fibrosis and inflammation.

Findings consistent with exclusion of a relevant myocardial infarction, myocarditis or specific cardiomyopathy.

Reporter: Dr.\*

Reviewer: Dr.\*

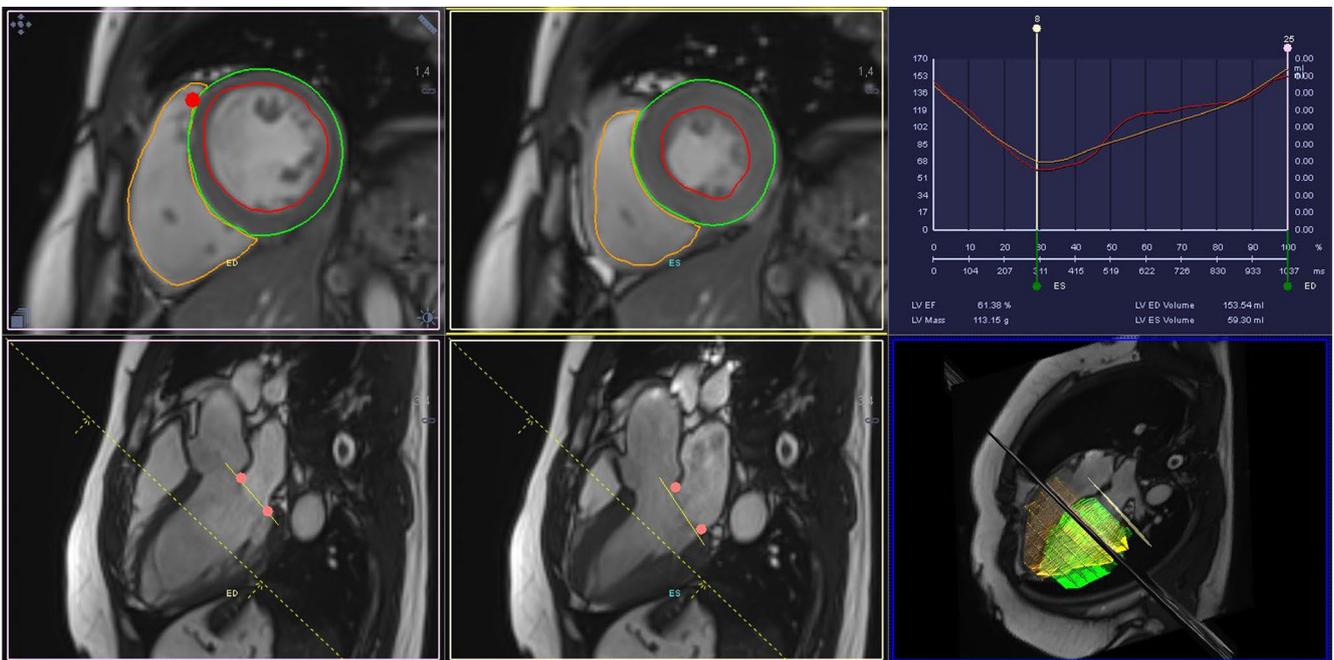
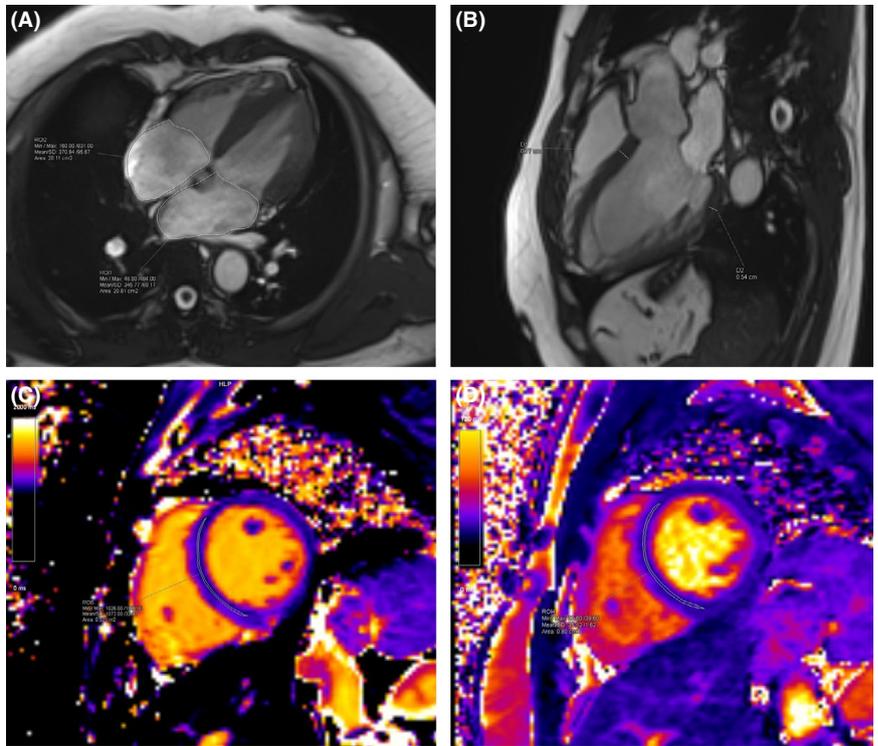
Date: 23/05/2019

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\* This report is only for clinical reference, and it is invalid without the (Electronic) signature of the doctor.

**FIGURE 1** An example of structured reporting of cardiovascular magnetic resonance.

**FIGURE 2** Measurements of (A) atrium areas, (B) LV wall thickness, (C) native T1 value, and (D) T2 value of middle septum.



**FIGURE 3** Measurements of biventricular volumes and function.

thickness of all 16 segments separately in the end-diastolic short-axis cine images. The analysis of the LV wall thickening needs to be combined with indexed LV mass. When LV is remodeled, LV wall thickness is usually normal but indexed LV mass is increased, while LV hypertrophy (LVH) is accompanied by both increased LV wall thickening and indexed LV mass, and a further distinction between centripetal LVH and eccentric LVH needs to be made.<sup>7</sup>

- Other morphological abnormalities: Myocardial crypt, diverticulum, congenital ventricular aneurysm, hypertrabeculation, and left ventricular non-compaction should be identified by combining the long-axis and short-axis cine images. At the same time, it is necessary to observe whether there is mural thrombus in the abnormal myocardium.

| Patient Name:                                                  |     |         |                       |       | Patient Name:                                                  |     |                       |                |                      |
|----------------------------------------------------------------|-----|---------|-----------------------|-------|----------------------------------------------------------------|-----|-----------------------|----------------|----------------------|
| Patient ID                                                     |     |         |                       |       | Patient ID                                                     |     |                       |                |                      |
| Examination Date 2019/5/22                                     |     |         |                       |       | Examination Date 2019/5/22                                     |     |                       |                |                      |
| Patient Height 158 cm Patient Weight 45 kg Heart Rate 56 b/min |     |         |                       |       | Patient Height 158 cm Patient Weight 45 kg Heart Rate 56 b/min |     |                       |                |                      |
| <b>Left Ventricle - Absolute</b>                               |     |         |                       |       | <b>Left Ventricle - Normalized</b>                             |     |                       |                |                      |
| Cardiac Function                                               |     |         | Normal Range(M (MRI)) | Unit  | Cardiac Function                                               |     | Normal Range(M (MRI)) | Unit           |                      |
| Ejection Fraction:                                             | EF  | 61.38   | 56.00 - 78.00         | %     | End Diastolic Volume                                           | EDV | 107.95                | 47.00 - 92.00  | ml/m <sup>2</sup>    |
| End Diastolic Volume                                           | EDV | 153.54  | 77.00 - 195.00        | ml    | End Systolic Volume                                            | ESV | 41.69                 | 12.75 - 30.00  | ml/m <sup>2</sup>    |
| End Systolic Volume                                            | ESV | 59.3    | 19.00 - 72.00         | ml    | Stroke Volume                                                  | SV  | 66.26                 | 32.00 - 62.00  | ml/m <sup>2</sup>    |
| Stroke Volume                                                  | SV  | 94.24   | 51.00 - 133.00        | ml    | Cardiac Index                                                  | CI  | 3.71                  | 1.74 - 4.20    | l/min/m <sup>2</sup> |
| Cardiac Output:                                                | CO  | 5.28    | 2.82 - 8.82           | l/min | Myocardial Mass (at ED)                                        |     | 77.66                 | 70.00 - 113.00 | g/m <sup>2</sup>     |
| Myocardial Mass (at ED)                                        |     | 110.46  | 118.00 - 238.00       | g     | Myocardial Mass (Avg)                                          |     | 79.55                 | 70.00 - 113.00 | g/m <sup>2</sup>     |
| Myocardial Mass (Avg)                                          |     | 113.15  | 118.00 - 238.00       | g     |                                                                |     |                       |                |                      |
| <b>Filling and Ejection Data</b>                               |     |         |                       |       | <b>Filling and Ejection Data</b>                               |     |                       |                |                      |
| Peak Ejection Rate:                                            |     | -413.76 | n.a.                  | ml/s  | Peak Ejection Rate:                                            |     | -290.9                | n.a.           | ml/s/m <sup>2</sup>  |
| Peak Ejection Time                                             |     | 0       | n.a.                  | ms    | Peak Filling Rate                                              |     | 307.08                | n.a.           | ml/s/m <sup>2</sup>  |
| Peak Filling Rate                                              |     | 436.77  | n.a.                  | ml/s  |                                                                |     |                       |                |                      |
| Peak Filling Time:                                             |     | 501.3   | n.a.                  | ms    |                                                                |     |                       |                |                      |
|                                                                |     |         |                       |       | * Patient Surface Area: 1.42 m <sup>2</sup>                    |     |                       |                |                      |
| Patient Name:                                                  |     |         |                       |       | Patient Name:                                                  |     |                       |                |                      |
| Patient ID                                                     |     |         |                       |       | Patient ID                                                     |     |                       |                |                      |
| Examination Date 2019/5/22                                     |     |         |                       |       | Examination Date 2019/5/22                                     |     |                       |                |                      |
| Patient Height 158 cm Patient Weight 45 kg Heart Rate 56 b/min |     |         |                       |       | Patient Height 158 cm Patient Weight 45 kg Heart Rate 56 b/min |     |                       |                |                      |
| <b>Right Ventricle - Absolute</b>                              |     |         |                       |       | <b>Right Ventricle - Normalized</b>                            |     |                       |                |                      |
| Cardiac Function                                               |     |         | Normal Range(M (MRI)) | Unit  | Cardiac Function                                               |     | Normal Range(M (MRI)) | Unit           |                      |
| Ejection Fraction:                                             | EF  | 57.67   | 47.00 - 74.00         | %     | End Diastolic Volume                                           | EDV | 112.72                | 55.00 - 105.00 | ml/m <sup>2</sup>    |
| End Diastolic Volume                                           | EDV | 160.32  | 88.00 - 227.00        | ml    | End Systolic Volume                                            | ESV | 47.71                 | 15.43 - 42.91  | ml/m <sup>2</sup>    |
| End Systolic Volume                                            | ESV | 67.86   | 23.00 - 103.00        | ml    | Stroke Volume                                                  | SV  | 65.01                 | 32.00 - 64.00  | ml/m <sup>2</sup>    |
| Stroke Volume                                                  | SV  | 92.46   | 52.00 - 138.00        | ml    | Cardiac Index                                                  | CI  | 3.64                  | 1.74 - 4.20    | l/min/m <sup>2</sup> |
| Cardiac Output:                                                | CO  | 5.18    | 2.82 - 8.82           | l/min | Myocardial Mass (at ED)                                        |     | 0                     | 16.00 - 36.00  | g/m <sup>2</sup>     |
| Myocardial Mass (at ED)                                        |     | 0       | 30.00 - 70.00         | g     | Myocardial Mass (Avg)                                          |     | 0                     | 16.00 - 36.00  | g/m <sup>2</sup>     |
| Myocardial Mass (Avg)                                          |     | 0       | 30.00 - 70.00         | g     |                                                                |     |                       |                |                      |
| <b>Filling and Ejection Data</b>                               |     |         |                       |       | <b>Filling and Ejection Data</b>                               |     |                       |                |                      |
| Peak Ejection Rate:                                            |     | -293.58 | n.a.                  | ml/s  | Peak Ejection Rate:                                            |     | -206.41               | n.a.           | ml/s/m <sup>2</sup>  |
| Peak Ejection Time                                             |     | 103.72  | n.a.                  | ms    | Peak Filling Rate                                              |     | 158.79                | n.a.           | ml/s/m <sup>2</sup>  |
| Peak Filling Rate                                              |     | 225.85  | n.a.                  | ml/s  |                                                                |     |                       |                |                      |
| Peak Filling Time:                                             |     | 976.67  | n.a.                  | ms    |                                                                |     |                       |                |                      |
|                                                                |     |         |                       |       | * Patient Surface Area: 1.42 m <sup>2</sup>                    |     |                       |                |                      |

FIGURE 4 Results of biventricular volumes and function.

## 2. Right ventricle

- RV volume: An enlarged RV volume is confirmed if the end-diastolic RV EDV is larger than the normal range of the corresponding sex and age.
- RV wall motion: A straight ventricular septum, "D" shaped LV, and paradoxical motion of the ventricular septum indicate right heart strain. Signs of arrhythmogenic right ventricular cardiomyopathy include increased RV volume, reduced global RV function, and abnormalities of right ventricular wall motion (hypokinesia, akinesia, segmental dyskinesia, aneurysm, or dyssynchrony).<sup>8</sup>
- Global RV function: Whether the global RV systolic function is reduced is confirmed by comparing the result of RVEF with normal range of the corresponding sex and age.
- RV thickness: Normal RV thickness is <4 mm.

- Other morphological abnormalities: Infiltration of fat in the myocardium, thinning or thickening of local wall, hypertrophy and disorder of trabeculation, right ventricular outflow tract expansion, and so forth can also be seen in arrhythmogenic right ventricular cardiomyopathy.<sup>8</sup>

## 3. Atria

The left atrium (LA) and right atrium (RA) areas should be drawn in the four-chamber long-axis cine image at the end-systole (Figure 2a), which is used to evaluate whether LA and RA are enlarged (normal area: LA < 24 cm<sup>2</sup>, RA < 23 cm<sup>2</sup>).

## 4. Left ventricular outflow tract and aortic root

- Left ventricular outflow tract (LVOT): The presence of LVOT obstruction is the main indicator distinguishing between obstructive and non-obstructive hypertrophic cardiomyopathy. LVOT obstruction and systolic anterior motion should be observed

in the 3CH long-axis cine image. During scanning, if LVOT obstruction is found in the 3CH cine image, more LVOT and aortic valve cine images should be scanned. Flow is scanned at the aortic valve level and quantitative flow measurements are acquired by post-processing software. The antegrade volume, retrograde volume, peak velocity, mean transvalvular gradients, and regurgitant fraction should be reported.

- Aortic root (AoR): Enlarged AoR, aortic valve stenosis, and bicuspid valve deformity should be identified. If aortic valve regurgitation or aortic valve stenosis is found in the 3CH cine image during scanning, it is also necessary to scan the flow sequence at the aortic valve level and take the quantitative flow measurements. The measurement of the AoR should combine 3CH and LVOT cine images and the internal diameters of the AoR, sinus Valsalva, and sino-tubular junction should be reported; for example, AoR (3CH x LVOT): AoR: 2.2 x 2.3 cm; sinus Valsalva: 3.7 x 3.5 cm; and sino-tubular junction: 3.4 x 3.5 cm.

#### 5. Pericardium

- Pericardial thickening ( $\geq 3$  mm);
- Pericardial effusion;

Classification of pericardial effusion size: trace  $< 5$  mm, small 5 mm-14 mm, moderate 15 mm-24 mm, and large  $\geq 25$  mm;

- Pericardial enhancement, pericardial calcification; and

Pericardial thickening, pericardial effusion, and pericardial enhancement are all signs of pericarditis. The pericardial enhancement shown by the late gadolinium enhancement (LGE) sequence is often related to the degree of inflammatory activity. At the same time, the presence of restricted diastolic ventricular filling, abnormal septal motion (shudder), and atrial and vena cava enlargement in the cine image should be observed as these factors suggest constrictive pericarditis.

#### 6. Extracardiac discovery

Extracardiac findings should not be missed. Common extracardiac findings found on CMR include the following: pleural effusion, pulmonary nodules/mass, mediastinal mass/lymph node enlargement, liver disease, spleen disease, renal cyst, pulmonary embolism, aortic dissection, and aortic aneurysm.

## 4.2 | Tissue characterization

### 1. Myocardial edema, inflammation, and diffuse fibrosis

To evaluate the presence of myocardial edema or inflammation, it is recommended preferentially to scan with the combination of T1 mapping and T2 mapping sequences.<sup>9</sup> If mapping sequences are not available, T2WI can be used to observe myocardial edema. Extracellular matrix volume fraction (ECV) is still in the stage of research and cannot be used for clinical diagnosis.

- T2 Mapping + T1 Mapping

Diverse equipment, magnetic strengths, and mapping sequences can cause differences in T1 and T2 values. Therefore, hospitals

should develop the corresponding native T1 and T2 values and standard deviations (SD) of normal people by using their own equipment. About the region of interest (ROI) of T1 and T2 measurement, it is recommended to adopt the standardized method of middle septum<sup>10</sup> (Figure 2c,d), and the interpretation of results as follows:

- Normal native T1 and T2
  - a. No evidence of myocardial diffuse fibrosis and inflammation;
- Elevated native T1 ( $> 2$  SD), normal native T2
  - a. Consistent with diffuse myocardial fibrosis, no significant inflammation;
- Elevated native T1 ( $> 2$  SD) and T2 ( $> 2$  SD)
  - a. Consistent with diffuse myocardial fibrosis and inflammation;
- Very high native T1 ( $> 5$  SD) and T2 ( $> 2$  SD)
  - a. Evidence of high active myocardial inflammation or myocardial necrosis (acute phase); and
- Reduced native T1 ( $< 2$  SD) and T2 ( $< 2$  SD)
  - a. Evidence of myocardial iron deposition or fat accumulation

### T2WI.

T2WI images are more susceptible to the effect of heart rate and breathing, which may cause artifacts. At the same time, slow blood flowing in the heart cavity could also produce band-like high signals near the endocardium, which may be mistaken for myocardial edema. Short-axis slice is recommended to measure T2WI signal intensity (SI). The global ROI should include all myocardium between the endocardium and epicardium.<sup>11</sup> Meanwhile, the SI of skeletal muscle in the same slice should be measured. The SI ratio between myocardium and skeletal muscle is more than 2.0, indicating myocardial edema.<sup>11</sup> On T2WI images of patients with acute myocardial infarction, low signal in the edema, which reflects intracardiac hemorrhage (IMH), should be reported.

### 2. Myocardial perfusion

Vasodilators (adenosine/regadenoson) are recommended for stress perfusion imaging. A low signal should be checked first to determine if it is a dark-rim artifact caused by the Gibbs effect. Examples of interpretation of myocardial hypoperfusion:

- No hypoperfusion
  - a. No evidence of relevant hypoperfusion or ischemia;
- Evidence of hypoperfusion in medial-apical inferior lateral and inferior wall within the range of LGE
  - a. No evidence of relevant ischemia;
- Evidence of hypoperfusion in basal-medial anterior and anteroseptal wall
  - a. Evidence of relevant ischemia in the left anterior descending branch area;
- Evidence of hypoperfusion in basal lateral wall with extension beyond the range of LGE
  - a. Evidence of inducible ischemia around the ischemic scar in the left circumflex branch area, suggesting the formation of collateral circulation; and

- Non-regional hypoperfusion
  - a. Evidence of microvascular disorder.
- 3. Myocardial hyperemia

A short axis level is recommended to measure the SI of the myocardium in native and post-enhanced T1WI. The global ROI should include all myocardium between the endocardium and epicardium.<sup>11</sup> The SI of skeletal muscle at the same slice should also be measured as the reference. An early gadolinium enhancement ratio > 4.0 or absolute early myocardial enhancement ratio > 45% is regarded as myocardial hyperemia.<sup>11</sup> Due to the difficulty of early gadolinium enhancement in guaranteeing the consistency of image quality, the newest expert consensus suggests that T1WI is no longer recommended as routine sequence, and T1 mapping is recommended as a replacement.<sup>9</sup>

4. Myocardial scars
  - The scar shows high signal in the LGE sequence. If an SI is detected of 2 SD higher than the normal myocardium, it should be reported as a scar after excluding false positive enhancement. The distribution of LGE should be described as subepicardial, intramural, subendocardial, or transmural. According to the distribution characteristics of LGE, it can be speculated that the reasons for LGE may be ischemic (endocardial to transmural distribution, which should be consistent with coronary artery perfusion territories) or non-ischemic (intramural or epicardial distribution, which is not consistent with distribution of coronary artery perfusion territories). Patchy or stria LGE should be described when evaluating intramyocardial enhancement. Diffuse distribution of LGE and insufficient contrast between blood pool and myocardium can be found in cardiac amyloidosis.
  - The location, segment, and transmural extent of ischemic LGE should be reported. The transmural extent of LGE is usually defined as 0, ≤25%, 26% to ≤50%, 51% to ≤75%, and 76% to 100%. Myocardial viability can be inferred from the transmurality:
    - a. transmural degree ≤25%
      - with residual vitality;
    - b. transmural degree 26% to 50%
      - with most likely residual vitality;
    - c. degree of transmural wall 51% to 75%
      - with low probability of residual vitality; and
    - d. transmural degree >75%
      - without residual vitality.
  - In the LGE area, it is also necessary to observe whether or not thrombus and/or ventricular aneurysm is formed. The location and size of the thrombus should be described. The neck and body of the ventricular aneurysm should be measured, meanwhile true and pseudo ventricular aneurysms should be distinguished. Microvascular obstruction associated with LGE should be identified in patients with acute myocardial infarction, and the location of microvascular obstruction should be described.

## 5 | SUMMARY AND CONCLUSION

The summary and conclusion of the CMR report should be obtained by comprehensive analysis of the results of morphological, functional, and tissue characteristics. It is recommended to provide conclusive opinions and answer the corresponding important questions raised by the clinicians so that they can develop correct treatment plans based on the report. Besides positive conclusions, important negative results should also be listed, such as exclusion of a specific cardiomyopathy or myocardial infarction, no evidence of active inflammation, or diffuse fibrosis of the myocardium. Recommendations for follow-up should be given, for instance, "We recommend a non-contrast CMR examination in 6 months" or "We propose a re-introduction to CMR in 3 months." If necessary, suggestion of treatment and screening can also be added to the Conclusion, for example, "Intensive heart failure therapy and optimization of blood pressure control is recommended" or "CMR screening of immediate family members is recommended."

## 6 | CONSIDERATIONS ON THE APPLICATION OF CMR STRUCTURED REPORTING

Special circumstances that affect the scanning process should be stated in the report, such as "With pronounced claustrophobia, an enhanced scanning could not be performed," "In the history of Myasthenia Gravis, the stress remedy is contraindicated," or "Due to severe COPD, a stress perfusion with adenosine could not be performed for safety reasons." The quality of an MR image can affect the diagnosis directly. For instance, the presence of atrial fibrillation, frequent ventricular premature beats, and other arrhythmias will mean that the volumetric/functional evaluation is somewhat limited: LV-EDV and ESV are rather overestimated; the patient's inability to hold their breathe may cause respiratory motion artifacts that significantly interfere with the quality of cine and LGE images; a thin ventricular wall will lead to inaccurate measurement of LV volume, thickness, and mass; improper setting of inversion time in LGE sequence scan will lead to underestimation of LGE. All the above factors may result in false positive or false negative results, which must be analyzed in combination with the specific circumstances during scanning to avoid missed diagnosis or misdiagnosis.

## 7 | PROSPECT OF CMR STRUCTURED REPORTING

Structured reporting is conducive to standardizing the content and improving the integrity, accuracy, and pertinence of the report, which will facilitate effective communication between radiologists and clinicians, helping clinicians to extract important information, develop correct treatment programs,<sup>12</sup> and make comparative analysis at follow-up. The utilization of a structured report improves the clinical

value of image diagnostic reports.<sup>13</sup> With the rapid development and application of artificial intelligence (AI) in diagnostic imaging, the radiomics characteristics of the myocardium could be extracted so that model of radiomics would be established on the basis of the structural report. Meanwhile, efficiency and accuracy of the analysis of cardiac function, tissue characteristics, and other aspects will be improved by using machine-learning technology. Structured reporting is a platform that can integrate the results obtained by AI technology and radiomics into the clinical process, which would provide better guidance in risk stratification, making effective treatment plans and prognosis evaluation, helping to continuously raise the clinical value and status of CMR in precision medicine.

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#### CONFLICTS OF INTEREST

Nothing to disclose.

#### AUTHOR CONTRIBUTIONS

Yihua Huang: Design, data collection and analysis. Hui Zhou: Design, literature review, coordination. Yabo Feng: Literature review and English language edit. Moling Zhou and Haixiong Tang: Data collection. Gaofeng Zhou and Jinkang Liu: Data analysis. Weihua Liao: Data analysis, review of medical notes. All authors were involved in the drafting, review, and approval of the report and the decision to submit for publication.

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