Sudden cardiac death (SCD) is a significant problem worldwide and the risk stratification of SCD remains an important challenge. Although current guidelines recommend the use of implantable cardioverter-defibrillator mostly based on left ventricular ejection fraction (LVEF), the limited sensitivity and specificity of LVEF to predict SCD has been highlighted. Cardiac magnetic resonance (CMR) is a noninvasive imaging modality with a unique ability to visualize and quantify myocardial scars, with proven histopathological correlation. In this review, we describe the current evidences of CMR for predicting SCD in various cardiac diseases and the future directions of clinical applications.

Key words Magnetic resonance imaging · Sudden cardiac death · Implantable cardioverter-defibrillator · Prognosis · Gadolinium.
nance (CMR). Good tissue contrast between blood and myocardium and volumetric analysis based on multiple slices allow more accurate morphologic diagnosis and evaluation of LV systolic volume and function. Moreover, tissue characterization is an unique property of CMR allowing visualization of the electrically unstable myocardium causing SCD. In the following sections, we discuss the growing evidences on the value of CMR in risk stratification for SCD based on various CMR techniques (Table 2).

### Table 1. Indications of ICD implantation related to structural heart disease [8]

<table>
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<th>Class</th>
<th>Secondary prevention</th>
<th>Primary prevention</th>
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| Class I   | ICM  
- Patients with nonsustained VT due to prior MI, LVEF ≤40%, and inducible VF or sustained VT on electrophysiological study (LOE B) | Patients with LVEF ≤35% due to prior MI who are at least 40 days post-MI and in NYHA functional Class II or III (LOE A)  
- Patients with LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF ≤30% and in NYHA functional Class I (LOE A) |
|           | NICM  
- Patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable (LOE B) | Patients with nonischemic DCM who have an LVEF ≤35% and in NYHA functional Class II or III (LOE B) |
| Class IIa | NICM  
- Patients with HCM who have 1 or more major risk factors* for SCD (LOE C)  
- Patients with unexplained syncope, significant LV dysfunction and nonischemic DCM (LOE C)  
- Patients with ARVC who have ≥1 risk factors† for SCD (LOE C)  
- Patients with cardiac sarcoidosis, giant cell myocarditis or Chagas disease (LOE C) | |
| Class IIb | NICM  
- Patients with nonischemic heart disease who have an LVEF ≤35% and in NYHA functional Class I (LOE C)  
- Patients with syncope and advanced structural heart disease in whom thorough invasive and noninvasive investigations have failed to define a cause (LOE C)  
- Patients with a familial cardiomyopathy associated with SCD (LOE C)  
- Patients with LV noncompaction (LOE C) | |

*major risk factor for SCD in HCM: prior cardiac arrest, spontaneous sustained or nonsustained VT, family history of SCD, syncope, LV thickness ≥30 mm and an abnormal blood pressure response to exercise. †risk factor for SCD in ARVC: presence of a previous cardiac arrest, syncope due to VT, evidence of extensive RV disease, LV involvement, or presentation with polymorphic VT and RV apical aneurysm, which is associated with a genetic locus on chromosome 1q42–43. ICM: ischemic cardiomyopathy, NICM: nonischemic cardiomyopathy, LOE: level of evidence, LVOT: left ventricular outflow tract, LVEF: left ventricular ejection fraction, SCD: sudden cardiac death, RV: right ventricle, LV: left ventricular, ARVC: arrhythmogenic right ventricular cardiomyopathy, ICD: implantable cardioverter-defibrillator, VT: ventricular tachycardia, MI: myocardial infarction, NYHA: New York Heart Association. Adapted from Epstein et al. J Am Coll Cardiol 2013;61:e6-e75 [8]

### Table 2. Advantages of CMR in risk stratification for SCD

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Applications</th>
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</thead>
</table>
| Cine-CMR  
- High temporal and spatial resolution  
- Excellent tissue contrast  
- Volumetric assessment independent from geometric assumption | Highly accurate and reproducible measure of myocardial wall thickness, chamber volumes and systolic function |
| LGE-CMR  
- Scar tissue characterization and quantification | More accurate morphologic and functional evaluation of various cardiomypathies causing SCD (i.e., HCM, LVNC, ARVC) |
| T1 mapping  
- Absolute quantification of myocardial fibrosis  
- Assessment of diffuse interstitial fibrosis | Differentiation of etiology of various cardiomyopathies causing SCD (i.e., sarcoidosis, amyloidosis, ARVC)  
- Assessment of myocardial scar presence and extent for the prediction of SCD in ICM, DCM, HCM, and cardiac sarcoidosis  
- Role of T1 mapping in SCD prediction is expected, however further studies are required for individual cardiomyopathies |

CMR: cardiac magnetic resonance, SCD: sudden cardiac death, LGE: late gadolinium enhancement, HCM: hypertrophic cardiomyopathy, LVNC: left ventricular noncompaction cardiomypathy, ARVC: arrhythmogenic right ventricular cardiomypathy, ICM: ischemic cardiomyopathy, DCM: dilated cardiomyopathy
MORPHOLOGY AND FUNCTION ASSESSMENT: CINE-CMR

Due to the widespread bedside availability, cost and non-invasive nature, echocardiography has been the method of choice for the analysis of LV morphology and function in most situations. However, there are several important limitations including inter- and intra-observer variability, and poor image quality in a specific population with coexisting lung or breast disease and obesity. In addition, both the M-mode and 2D TTE measurements of LV volume and systolic function rely on geometric assumptions regarding LV, leading to inaccuracies in measurement. Conversely, breath-hold gradient echo or steady state free precession cine-CMR imaging possesses unique strengths of high spatial and temporal resolution with excellent tissue contrast between blood pool and myocardium, without limitations of either imaging window or imaging plane. Thus, CMR provides reliable and highly reproducible volumetric data independent of the geometric assumptions and serves as a gold standard technique [14,15].

With these important advantages, CMR has emerged as a useful adjunctive imaging modality for the evaluation of various cardiomyopathies [16,17]. For example, CMR provides more sensitive diagnosis of hypertrophic cardiomyopathy (HCM) and enables the precise characterization of disease phenotypic variations (Fig. 1) [18,19]. Although diagnosis of HCM is usually based on the TTE finding of unexplained LV hypertrophy, over- or under-estimation commonly occurs with echocardiography owing to the oblique cross section and limitation of lateral resolution. However, CMR with excellent tissue contrast allows for more accurate measurement of the most thickened segment, which is an important predictor of SCD in HCM [20,21], even in patients with basal anterolateral wall hypertrophy (Fig. 1A) or in those with an extremely thickened LV wall (Fig. 1B) [19]. Compared to echocardiography, CMR can detect twice as many apical aneurysms which can be a substrate for the ventricular arrhythmia leading to SCD (Fig. 1C) [22,23]. Furthermore, CMR allows reproducible LV mass quantification which has been considered unreliable with TTE due to the asymmetric distribution of hypertrophy and heterogeneous morphology. Although not a requirement for the diagnosis of HCM, increased LV mass is considered more sensitive for predicting the outcome [24].

CMR appears superior to TTE when assessing left ventricular noncompaction cardiomyopathy (LVNC), characterized by a thin, compacted epicardial layer and an extensive non-compacted endocardial layer. Although LVNC is a relatively rare cardiomyopathy, it is associated with high risk of ventricular tachyarrhythmia and SCD [25,26]. Diagnosis of LVNC relies on noninvasive imaging studies, and echocardiography remains the most common diagnostic strategy, largely due to widespread availability, ease of interpretability and low cost. However, currently, CMR is increasingly used in diagnosis and surveillance of LVNC since demarcation between noncompact and compact myocardium is more apparent on cine-CMR (Fig. 2A) [25]. Because a gold standard of diagnosis is lacking, many researchers are attempting to find more sensitive and accurate diagnostic features. Petersen et al. [27] reported a noncompact myocardium to compact myocardium ratio in diastole over 2.3 showed good diagnostic accuracy for LVNC. Jacquier et al. [28] suggested the percentage of trabeculated myocardium among total LV mass above 20% can predict the diagnosis of LVNC with a 93.7% sensitivity and 93.7% specificity.

CMR also plays an important role in the evaluation of the right ventricle (RV). Since the role of TTE in the evaluation of RV morphology and function is limited due to its crescentic shape and limitation of acoustic window [17], CMR is the currently accepted reference standard for assessing RV size and function [29-31]. Due to superior depiction of the RV and the ability of non-invasive myocardial tissue characterization, CMR is currently one of the main imaging modalities for assessing patients with suspected or known arrhythmogenic right ventricular cardiomyopathy (ARVC), characterized by fibro-fatty replacement of the RV myocardium. Since ARVC is the leading cause of SCD in the younger population [32], early recognition and accurate risk stratification are important [33]. CMR allows early diagnosis of ARVC as well as more sensitive evaluation for global and regional systolic dysfunction of the RV (Fig. 2B) [34]. In addition, CMR provides important prognostic information in patients with suspected ARVC. In a previous longitudinal study that followed 369 consecutive patients with at least one criterion for ARVC and defined abnormal CMR findings as increased RV volumes, reduced RV ejection fraction, RV regional wall motion abnormalities, myocardial fatty infiltration and myocardial fibrosis, patients with multiple abnormalities were at a greater risk of SCD, while patients without any of those abnormal findings had a very low event rate [35].

SCAR TISSUE CHARACTERIZATION AND QUANTIFICATION: LATE GADOLINIUM ENHANCEMENT (LGE)-CMR

Tissue characterization is a unique advantage of CMR that other imaging modalities such as TTE or cardiac computed tomography do not possess. Late gadolinium enhancement (LGE)-CMR imaging noninvasively visualizes myocardial scar tissue that directly correlates with pathology [36]. Moreover, geographic distribution of LGE helps distinguish different types of cardiomyopathies (Figs. 3 and 4) and LGE-CMR pro-
vides quantification of myocardial scars using full-width-at-half-maximum and/or different standard deviation threshold of the mean signal intensity of remote myocardium [37,38]. In a retrospective study evaluating 137 survivors of sudden cardiac arrest, LGE was observed in 71% of survivors and LGE presence and extent were strong predictors of recurrent adverse events including all-cause death and appropriate defibrillator therapy [39].

Importantly, myocardial scar tissue, which serves as a substrate for malignant arrhythmias, might not be concordant with LVEF [40]. Therefore, continued efforts have been made to evaluate whether assessment of myocardial scarring using LGE-CMR would improve risk stratification for SCD over LVEF. Although the mechanisms contributing to arrhythmia generation remain poorly understood, the presence and extent of the myocardial scar from LGE-CMR appears to have prognostic value in both ischemic and nonischemic cardiomyopathies [41-46]. In a recent meta-analysis, Disertori et al. [47] showed that LGE is a powerful predictor of ventricular arrhythmia with a >5-fold increased risk [odds ratio (OR)=5.62], with no significant differences between ischemic (OR=5.05) and nonischemic patients (OR=6.27). Although LGE presence itself is considered a powerful predictor of SCD, several studies suggested that a certain amount of myocardial scarring is the most predictable factor for SCD [40,48,49]. Klem et al. [40] reported that a LGE extent >5% of LV myocardium was the most important predictor in patients with ischemic and nonischemic cardiomyopathies. Interestingly, Assomull et al. [48] sug-

![Fig. 1. CMR of HCM. TTE, cine image at ED and LGE image in patients with various types of HCM. (A) Fifty-eight-year-old male presented with Wolff-Parkinson-White syndrome. TTE showed mass-like hypertrophy of anterolateral wall. Cine-CMR clearly showed mass-like hypertrophy at basal anterolateral wall and LGE-CMR identified patch fibrotic changes in the hypertrophied myocardium. Genetic test showed MYBPC3 mutation, which is a representative genetic abnormality found in HCM. (B) Twenty-nine-year-old female with family history of HCM. TTE detected markedly increased LV septal wall thickness. However, accurate measure of septal wall thickness was limited and apical lateral wall hypertrophy was not apparent on TTE. However, asymmetric septal and apical hypertrophy was clearly delineated using cine-CMR. In addition, LGE-CMR showed extensive fibrosis of hypertrophied LV myocardium. (C) Fifty-year-old male with burnout stage of apical HCM. CMR showed focal apical aneurysm with subendocardial LGE. CMR: cardiac magnetic resonance, HCM: hypertrophic cardiomyopathy, LV: left ventricular, ED: end-diastole, TTE: transthoracic echocardiography, LGE: late gadolinium enhancement.]

TTE  Cine-CMR  LGE-CMR  LGE-CMR

A

B

C
gested a similar cutoff value of 4.8% of LGE extent in nonischemic cardiomyopathy. Although speculative, these data could indicate that CMR can guide the management including ICD implantation. Klem et al. [40] showed that patients with LVEF >30% and significant scarring (LGE extent >5%) demonstrated a higher risk than patients with no or minimal scarring, but had similar risk compared to those with LVEF <30%. Conversely, patients with LVEF <30% and with minimal or no scarring showed favorable risk comparable to patients with LVEF >30%. Strategies using LGE-CMR for the prediction of SCD requires prospective evaluation in larger cohorts before being incorporated into the guidelines.

Regarding specific cardiomyopathies, the presence and extent of LGE were independently associated with SCD risk in patients with ischemic cardiomyopathy (ICM) [40,50,51]. Notably, in this population, infarct tissue heterogeneity was hypothesized to create electrical dispersion and areas of slow conduction acting as substrates for electrical re-entry and malignant arrhythmia [52]. Infarct tissue heterogeneity can be assessed with LGE-CMR by quantifying the myocardium with an intermediate signal intensity (commonly named the peri-infarct zone or gray zone), most likely reflecting an admixture of infarct tissue and viable myocardium [53,54]. Several studies reported that the extent of myocardial infarction as well as the extent of peri-infarct zone was independent predictors of in-ducible ventricular tachyarrhythmia [55,56] and appropriate ICD therapy [53]. However, the analysis of LGE-CMR parameters is lacking uniformity, thus in the aforementioned studies, different criteria were applied [53-56]. When these different methods were compared in 55 ICM patients with LVEF <35%, total infarct size was comparable, whereas core- and peri-infarct zones varied significantly between the tested models [57]. Furthermore, in that study, additional estimation of core- and/or peri-infarct zones did not appear to increase the diagnostic accuracy over total infarct size alone. Currently, disagreement remains regarding which type of scar was most effective, thus, further studies are required whether a specific scar pattern is most predictive of SCD.

Although dilated cardiomyopathy (DCM) is characterized by ventricular dilatation and impairment of cardiac function in the absence of significant coronary artery disease, observing LGE on CMR imaging is common. Approximately 30% of patients with DCM have a characteristic mid-wall enhancement pattern of replacement fibrosis on LGE-CMR imaging, which is distinct from infarctions with spared subendocardium (Fig. 3B) [36,58]. Previous small studies reported the presence of and extent of LGE had prognostic value in DCM [40,48,59]. Gulati et al. [60] recently reported that LGE-CMR imaging provided independent prognostic information beyond LVEF in a large-scale registry of DCM. In their study, although the

Fig. 2. CMR of LVNC and ARVC. (A) Sixty-two-year-old male showed marked trabeculation of apex on TTE. Cine-CMR showed the ratio of noncompact myocardium to compact myocardium >2.3 at end-diastole, resulting in a LVNC diagnosis. LGE was not apparent. (B) Forty-seven-year-old female who presented with ventricular tachycardia restored to sinus rhythm after DC cardioversion. Cine images of end-diastole (ED) and end-systole (ES) show dilated right ventricle (RV) and dyskinetic movement of RV aneurysm (asterisk) suggestive of ARVC. Diffuse LGE was also detected in the RV wall. CMR: cardiac magnetic resonance, LVNC: left ventricular noncompaction cardiomyopathy, ARVC: arrhythmogenic right ventricular cardiomyopathy, TTE: transthoracic echocardiography, LGE: late gadolinium enhancement.
primary endpoint was all-cause mortality, investigators also followed the occurrence of arrhythmic composite of SCD or aborted SCD and found that the presence and extent of mid-wall fibrosis on LGE-CMR were also significant independent predictors of SCD or aborted SCD. Although the potential clinical utility of assessment of mid-wall fibrosis on LGE-CMR in the risk stratification of patients with DCM requires further investigation, it is currently expected to improve risk stratification beyond LVEF.

HCM is the most actively studied cardiomyopathy using LGE-CMR due to its diverse disease spectrum. While the life expectancy of substantial patients with HCM is comparable with the unaffected population, a specific patient group is vulnerable to SCD with an approximate 5-fold increase in the SCD risk [61]. A major challenge in the management of HCM patients is to select those who are at a higher risk for clinical ventricular arrhythmia or SCD and may benefit from prophylactic ICD implantation. The currently used risk stratification algorithm to identify candidates for ICD therapy largely relies on the following six major risk markers: 1) prior personal history of ventricular fibrillation, SCD, or sustained ventricular tachycardia; 2) family history of SCD or appropriate ICD shock; 3) unexplained syncope; 4) nonsustained ventricular tachyarrhythmia on 24-h ambulatory ECG monitoring; 5) maximal LV wall thickness $\geq$ 30 mm; and 6) abnormal LV pressure response during exercise [20,21]. However, the strategy is often compounded by the unpredictability of the arrhythmogenic substrate and limited by the impracticability of prospective randomized trials. In a multicenter registry of HCM patients who had received ICD, only 13% of patients with prophylactic ICD implantation for high-risk features had an appropriate discharge for ventricular tachycardia or fibrillation [62]. Simultaneously, a significant proportion of ICD discharges occurred in patients with a single risk factor and the risk of appropriate ICD discharge was not significantly different for 1, 2 or 3 of the traditional risk factors [62]. In addition, even the absence of all risk factors does not convey immunity to SCD in HCM patients [63]. These considerations have led to the identification of a novel primary risk predictor for HCM.

There has been significant interest in using LGE-CMR to evaluate myocardial scars, which might provide the arrhythmogenic substrate in HCM. Multiple studies have shown a high prevalence of LGE in HCM, occurring in more than 50% of patients, predominantly in a patchy, multifocal distribution in the hypertrophied region (Fig. 1) [16]. Although LGE was shown predictive for SCD in HCM [44,64-67], LGE presence

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Fig. 3. CMR of ischemic and nonischemic cardiomyopathy. Cine- and LGE-CMR images of two patients with severe LV systolic dysfunction. (A) Sixty-year-old female complained of dyspnea on exertion with a history of percutaneous coronary intervention to left anterior descending artery (LAD). Cine-CMR showed LV systolic dysfunction (LVEF=34%) with regional wall motion abnormality in LAD territory. LGE-CMR showed subendocardial and transmural scars in the LAD territory. (B) Thirty-year-old male complained of dyspnea on exertion. Cine-CMR showed global LV hypokinesia (LVEF=11%). LGE-CMR showed a mid-wall fibrosis pattern distinct from myocardial infarction with spared subendocardium indicating nonischemic dilated cardiomyopathy. The patient was eventually diagnosed with LMNA-related familial dilated cardiomyopathy which is considered a high-risk factor for fatal arrhythmic complications and is prone to lead to SCD. CMR: cardiac magnetic resonance, LGE: late gadolinium enhancement, LVNC: left ventricular noncompaction cardiomyopathy, LVEF: left ventricular ejection fraction, SCD: sudden cardiac death, LV: left ventricular, ED: end-diastole, ES: end-systole.
alone is insufficient to stratify the risk of individual patients for SCD since it is common in HCM. Conversely, the absence of LGE is associated with a lower risk of SCD and represents a source of reassurance [49]. In addition, the risk of SCD is proportional to the LGE extent. In a recent meta-analysis by Weng et al., [64] LGE extent as well as presence was significantly associated with SCD risk. In a large multicenter HCM cohort study, Chan et al. [49] showed that LGE extent was associated with an increased risk of SCD event even after adjustment for other relevant disease variables; each 10% increase in LGE was associated with a 40% increase in relative SCD event risk (adjusted hazard ratio of 1.46 per 10% increase in LGE). Interestingly, in their study, the investigators performed a subgroup analysis in HCM patients otherwise considered at low risk and who did not have established markers, and found that extensive LGE defined as extent ≥15% of LV mass was associated with a 2-fold increase in SCD risk with an estimated likelihood for SCD events of 6% at 5 years [49]. Therefore, using LGE-CMR could resolve complex ICD decisions when the level of SCD risk remains uncertain after standard risk stratification. However, an important limitation of the current techniques of LGE ascertainment still exists with wide variability, including different cutoffs based on standard deviation and the full-width half-maximum technique [38,68]. Further standardization in the definition of abnormal LGE in HCM would be required before LGE-CMR is incorporated into the guidelines. In addition, the assessment of diffuse fibrosis using the T1 mapping, which will be discussed in the following section, requires further evaluation for the prediction of SCD [69].

There are several studies reporting LGE as a potential prognostic marker in other myocardial diseases, including myocarditis, myocardial infiltrative diseases and ARVC. Although

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**Fig. 4. CMR of various cardiomyopathies.** (A) Seventeen-year-old male presented with nonsustained ventricular tachycardia. Although LV systolic function was preserved, LGE-CMR showed extensive myocardial necrosis. Black-blood T2-weighted image (BB T2WI, asterisk) showed extensive myocardial edema which suggests acute myocarditis. (B) Fifty-five-year-old male diagnosed with sarcoidosis. Patchy subepicardial and transmural LGE was distributed in the regions discordant with coronary territory indicating cardiac involvement of sarcoidosis. (C) Fifty-one year-old male diagnosed with amyloidosis. LGE-CMR showed diffuse uptake of gadolinium in the wall of both atria and ventricles. Subendocardial ring-like enhancement shown in short axis view is a characteristic finding of cardiac amyloidosis. CMR: cardiac magnetic resonance, LGE: late gadolinium enhancement, LV: left ventricular.
CMR plays an increasingly important role in the diagnosis of these myocardial diseases (Fig. 4), evidence regarding SCD risk prediction is limited. For example, CMR has become the diagnostic tool of choice in tertiary care centers for patients with suspected myocarditis (Fig. 4A) [70]. Although a comprehensive CMR examination allows visualization of several important features of myocarditis, including contractile dysfunction with and without pericardial effusion, myocardial edema and necrosis, we could find only a single study evaluating the prognostic value of LGE-CMR in patients with myocarditis [71]. Grün et al. [71] followed 222 patients with viral myocarditis and showed the presence of LGE is the strongest independent predictor of all-cause mortality and cardiac mortality. However, in that study, SCD analysis was not performed because no patient without LGE experienced SCD regardless of LVEF. Larger prospective studies are required to validate that the absence of LGE might identify the patients with low risk of SCD who can be followed-up without ICD implantation.

CMR has been shown to have excellent diagnostic accuracy for the detection of cardiac sarcoidosis and is becoming the gold standard for the diagnosis (Fig. 4B) [72,73]. In addition to the diagnosis of cardiac sarcoidosis based on the presence of LGE, CMR can also predict SCD or ICD-aborted cardiac death in patients who underwent CMR on the suspicion of cardiac involvement of sarcoidosis [74,75]. Although those studies were single center studies involving a small number of patients, a recent meta-analysis showed the presence of LGE on CMR imaging is associated with increased risk of both all-cause mortality and arrhythmogenic events [76].

When evaluating ARVC, which is associated with a high prevalence of appropriate ICD therapy and SCD [77], the fibrous replacement of RV myocytes with or without fatty replacement is the core pathological finding. In addition to the morphological and functional evaluation of RV using cine-CMR, myocardial fibro-fatty changes in both RV and LV can be identified on LGE-CMR (Fig. 2B) [78]. LGE-CMR findings were well correlated with histopathological findings and associated with RV dysfunction, inducible ventricular arrhythmia on electrophysiological testing [79] and adverse cardiac outcome [35]. Therefore, LGE-CMR may provide guidance for electrophysiological studies and endomyocardial biopsy in patients with suspected ARVD. However, detection of LGE in the RV is often hampered by the thin RV wall, and distinguishing fat from fibrosis on LGE-CMR is challenging and highly dependent on the reader’s experience [78]. Importantly, a substantial portion of patients with ARVD receive an ICD irrespective of the LGE-CMR results in clinical practice [8,32].

TISSUE CHARACTERIZATION OTHER THAN LGE: T1-MAPPING

Although LGE-CMR is a robust technique to evaluate myocardial scars, several important limitations exist, including partial volume effect, post-processing time and different quantification techniques [80]. Specifically, the discrimination between scarred or fibrotic myocardium and normal myocardium relies on contrast concentration combined with inversion-recovery sequence parameters, which are chosen to null the normal myocardial signal. Therefore, although providing a sensitive and reproducible qualitative assessment of myocardial replacement fibrosis, LGE-CMR is limited in the absolute quantification of myocardial fibrosis and the assessment of diffuse interstitial fibrosis [81]. T1 mapping is an emerging tissue characterization method which enables direct myocardial signal quantification on a standardized scale. By directly measuring the underlying T1 relaxation time of each myocardial voxel, a better characterization of myocardial tissue composition on a global or regional level can be obtained. Pre-contrast T1 values of myocardial fibrosis are significantly longer than with normal myocardium, and post-contrast T1 values of myocardial fibrosis are significantly shorter than normal myocardium due to the retention of gadolinium contrast in fibrotic tissue [81]. In addition, extracellular volume fraction can be calculated from T1-maps acquired at pre- and post-contrast calibrated by blood hematocrit [81]. Since T1 mapping is currently an emerging technique, limited data is available showing the prognostic value of T1 mapping, especially for the prediction of SCD. To the best of our knowledge, only 1 study assessed whether myocardial tissue characterization using T1 mapping would predict arrhythmogenic events [69]. Chen et al. [69] showed that reclassification with native T1 value of ≥1015 ms due to the retention of gadolinium contrast in fibrotic tissue [81]. T1 mapping is an emerging tissue characterization method which enables direct myocardial signal quantification on a standardized scale. By directly measuring the underlying T1 relaxation time of each myocardial voxel, a better characterization of myocardial tissue composition on a global or regional level can be obtained. Pre-contrast T1 values of myocardial fibrosis are significantly longer than with normal myocardium, and post-contrast T1 values of myocardial fibrosis are significantly shorter than normal myocardium due to the retention of gadolinium contrast in fibrotic tissue [81]. In addition, extracellular volume fraction can be calculated from T1-maps acquired at pre- and post-contrast calibrated by blood hematocrit [81]. Since T1 mapping is currently an emerging technique, limited data is available showing the prognostic value of T1 mapping, especially for the prediction of SCD. To the best of our knowledge, only 1 study assessed whether myocardial tissue characterization using T1 mapping would predict arrhythmogenic events [69]. Chen et al. [69] showed that reclassification with native T1 value of ≥1015 ms using 1.5-T CMR is useful to distinguish high-risk patients for the appropriate ICD therapy or ventricular arrhythmia events in patients who received ICD implantation for primary prevention. Given the growing evidence supporting the prognostic value of T1 mapping [82,83], the use of T1 mapping might help stratify the SCD risk better by detecting subclinical myocardial changes before the emergence of systolic dysfunction and replacement fibrosis.

LIMITATIONS

Although the value of CMR for the prediction of SCD has been evaluated in multiple studies, the applicability of the study results was limited by a small number of events even in common diseases such as ICM, DCM and HCM. Therefore, in many of the studies, all-cause mortality was set as the primary endpoint instead of SCD. In addition, trials specifically de-
signed to evaluate the value of the CMR-guided strategy prospectively are lacking. For example, although numerous studies were conducted to determine the CMR predictor of SCD in HCM, an outstanding predictive model for SCD or randomized clinical trial with that risk stratification model has not been included [21]. Decision making for ICD implantation using CMR was attempted in ICM [84], but was discontinued due to poor enrollment [85]. Therefore, prospective studies evaluating the value of CMR-guided strategies with well-accepted endpoints in larger populations are required. In addition, standardization of quantification methods for scar extent is needed for more delicate risk assessment.

Furthermore, several results indicate the prevalence of SCD in the Asian population is lower than non-Asian population [86]. In a large epidemiological study from China, 41.8 of 100000 people died suddenly, while annual SCD incidence in the U.S. is approximately 100 of 100000 people [87]. The cause of the discrepancy in SCD prevalence between Asian and non-Asian populations is unknown, but is possibly due to the genetic background influences. Given this gap between the Asian population and others, studies evaluating SCD risk stratification using CMR need to be performed specifically in the Asian population.

Currently, implementing changes in the guidelines is premature. However, large scale studies such as global CMR registry and CMR-GUIDE have begun [88] and studies using CMR are increasing. Considering the growing evidences delineating the value of CMR for the prediction of SCD, CMR should be integrated into the SCD risk stratification strategy in the future.

Conflicts of Interest

The authors declare that they have no conflict of interest.

REFERENCES


www.e-cvia.org
60. McCrehon JA, Moon JC, Prasad SK, McKenna WJ, Lorenz CH, Coats AJ,


