Cardiac arrhythmias are a major cause of morbidity and mortality. Most feared are ventricular arrhythmias that include premature ventricular complexes, nonsustained ventricular tachycardia (NSVT), sustained VT, torsades de pointes, and ventricular fibrillation. Although ventricular arrhythmias are associated with increased risk, often they are not a direct threat but rather indicative of disease severity and may not warrant specific treatment. Antiarrhythmic treatment should be reserved for symptomatic patients and those considered at risk for the worst potential complication, an arrhythmia-induced sudden cardiac death (SCD). For patients considered at risk, randomized trials have shown that pharmacological antiarrhythmic therapy does not improve survival and is potentially harmful. Thus, the current standard is a prophylactic implantable cardioverter-defibrillator (ICD). Nevertheless, in other than primary arrhythmic conditions, the criteria for selecting patients for an ICD based largely on left ventricular ejection fraction (LVEF) are widely acknowledged to be deficient. Most patients (>80%) currently receiving an ICD do not use it over as long as 8 years. There are potentially serious implant and device complications. Better methods of identifying patients at risk for ventricular arrhythmic SCD are needed. Among techniques under investigation are noninvasive imaging procedures, including echocardiography, radionuclide imaging, and cardiac magnetic resonance (CMR).

Approach to Ventricular Arrhythmias: The Potential for Noninvasive Imaging

Identifying risk of lethal ventricular tachyarrhythmias is frustrated by the epidemiological phenomenon that although one can identify those at the highest risk, these represent a significant minority of people who will suffer a fatal event. The vast majority of SCD victims have minimal to no evidence specific to arrhythmic risk. Although an important approach to decreasing lethal arrhythmias is prevention of cardiac disease and aggressive treatments when present, the complex pathophysiology of arrhythmias makes it difficult to accurately identify most patients at risk. Ventricular arrhythmias are considered to be the result of a cascade of upstream events in an electrically unstable heart. As illustrated in Figure 1, the multiple potentially contributing variables are commonly classified as insults that create an abnormal myocardial substrate, acute triggers that can precipitate an arrhythmia, and modulating factors, such as altered neurohormonal balance or pathological activation of the autonomic nervous system, that can terminate or perpetuate an arrhythmia once it begins. Any aspect of cardiac disease can contribute to vulnerable substrate, including risk factors, recent ischemia with stunning, myocardial infarction (MI), genetic predispositions, and anatomic cardiac damage, all of which can disrupt normal electrophysiological processes creating arrhythmic vulnerability. Triggers include acute ischemia, electrolyte flux, environmental and hemodynamic stressors, sympathetic activation, any acute illness, and proarrhythmic drugs. Many of

Key Words: arrhythmias, cardiac defibrillators, implantable death, sudden, cardiac echocardiography magnetic resonance imaging MIBG radionuclide imaging
these factors are evaluable by imaging techniques that can both help identify patients at risk and potentially guide therapy.

**Imaging and Predicting Arrhythmic Risk Using Echocardiography**

Echocardiography is the most readily available imaging tool to assess structural heart disease. The success of any management strategy depends on accurate diagnosis of abnormalities in conditions, such as MI, nonischemic cardiomyopathies, including hypertrophic cardiomyopathy (HCM), cardiac amyloidosis, cardiac sarcoidosis, arrhythmogenic right ventricular cardiomyopathy (ARVC), and congenital heart diseases. Echocardiography plays an essential role not only in identifying disease but also in predicting arrhythmic risk. However, the heterogeneity of causes and mechanisms of SCD pose tremendous challenges.

**Assessment of LV Size and LV Ejection Fraction**

Simple echocardiographic measurement of the LV internal end-diastolic dimension was evaluated in the Oregon Sudden Unexpected Death Study, with severe LV dilatation shown to independently predict SCD. Combined with low EF, severe LV dilatation added value. However, although multiple studies have shown that LVEF is a robust predictor of SCD, in a population-based analysis, only 30% of those who experienced SCD had a severely decreased LVEF (<30%) on echocardiography, and the majority of patients who died suddenly after MI had LVEF>35%. In either ischemic or nonischemic dilated cardiomyopathy, LVEF can screen for individuals at high risk of SCD. However, arrhythmogenic substrate is different for patients with prior MI and those without. LVEF is not the whole story.

**Stress Echocardiography**

Stress echocardiography is an invaluable tool for assessing functional capacity, exercise-induced arrhythmia and syncope, and ischemia. A recent article describing RV stress testing among endurance athletes with normal resting cardiac function found RV contractile dysfunction during exercise to be associated with ventricular arrhythmia.

**Assessment of Ventricular Mechanics by Strain Echocardiography**

**Hypertrophic Cardiomyopathy**

Two-dimensional (2D) strain echocardiography can detect subclinical changes of myocardial deformation related to fibrosis. Based on CMR imaging, the extent of fibrosis in HCM correlates with SCD risk and is considered the gold standard for noninvasive assessment of fibrosis. Investigations using strain echocardiography are evolving. One study found that HCM patients with impaired LV global longitudinal strain (GLS) <15% at rest to be at higher risk for SCD, sustained VT, resuscitated arrest, and progressive heart failure (HF) symptoms. In addition, the study found LVOT obstruction (≥250 mmHg) during exercise echocardiography to independently predict poor outcome.

**Nonischemic Cardiomyopathy**

In other forms of nonischemic cardiomyopathy (NICM), heterogeneous scarring predisposing to ventricular arrhythmias can manifest as temporal dispersion of contraction that is assessable by GLS analysis. In 94 NICM patients, Haugaa et al reported that GLS was independent of and superior to LVEF in predicting arrhythmic events, suggesting that strain echocardiography has potential to assist assessment of NICM patients not fulfilling current ICD indicators. Further investigations are needed in this regard including assessment of the enhanced potential of 3D strain analysis that, in a recent study of patients with asymptomatic aortic stenosis, was found superior to 2D methods in predicting adverse cardiac events.

**Tetralogy of Fallot**

Diller et al followed (≈2.9 years) 413 post-tetralogy of Fallot repair patients (mean age 36, QRS 148 ms, LVEF 55%) and found impaired LV GLS to be associated with SCD or life threatening ventricular arrhythmias independent of QRS duration. When combined with right heart size and echo functional parameters, LV GLS provided incremental SCD prognostic information.

---

**Figure 1.** Factors implicated in the genesis of lethal arrhythmias commonly divided into those contributing to a vulnerable substrate and those that can trigger arrhythmogenesis in vulnerable substrate. Some factors fall into both categories. Modulating factors, such as central and regional autonomic control, can terminate or sustain an arrhythmia. NEFA indicates nonesterified fatty acids; PUFA, polyunsaturated fatty acids; SCD, sudden cardiac death; VF, ventricular fibrillation; and VT, ventricular tachycardia.
However, RV GLS alone did not significantly correlate with SCD or ventricular arrhythmia. The role of RV strain analysis in tetralogy of Fallot and its ability to predict arrhythmia are unknown.

**Long-QT Syndrome**
LV function has been considered normal in long-QT patients, but subtle mechanical changes may create electric disturbances. Haugaa et al used strain echocardiography to evaluate 101 patients with genotyped long-QT syndrome (LQTS) mutations. The mean myocardial contraction duration, reflecting times from electrocardiographic Q waves to peak LV strain in all segments, was longer in LQTS mutation carriers than in healthy controls and interestingly longer in symptomatic individuals than in asymptomatic carriers. As in Figure 2, although in symptomatic LQTS patients, contraction duration in the longitudinal direction (mostly representing subendocardial fibers) was longer than that of circumferential direction (reflecting midmyocardial fibers), the phenomenon of myocardial transmural dispersion was not observed in asymptomatic LQTS carriers or in healthy controls. Although mechanical abnormalities in LQTS have not been shown to independently predict arrhythmic outcome, it is exciting to explore the potential mechanistic basis for LQTS.

**Myocardial Infarction**
Timely and improved therapeutic strategies for acute MI have led to more patients with preserved LVEF post infarction. As LVEF may not adequately reflect arrhythmic risk, refined methods and newer tools are needed to evaluate electromechanical changes that predispose to arrhythmias. Haugaa et al conducted an important prospective multicenter study using 2D echocardiography to study 569 patients 40 days post-MI. GLS was a more sensitive predictor of arrhythmic events not only in patients with LVEF<35%, but also in patients with relatively preserved LVEF. Mechanical dispersion (MD) is a new parameter assessing myocardial contraction heterogeneity and has been shown to predict arrhythmic events independently of LVEF. The combination of GLS and MD had the best predictive value.

To elucidate potential echocardiographic indices that predict SCD soon after MI, 988 patients from Denmark were evaluated by 2D echocardiography within 48 hours of the event. During 30-month follow-up, GLS and MD were independent predictors of SCD.

**Arrhythmogenic Right Ventricular Cardiomyopathy**
Diagnosis and management of ARVC continue to be challenging. In addition to traditional predictors for SCD, such as RV morphological abnormalities, RV systolic dysfunction assessed by conventional methods, electrocardiographic changes, and frequency of NSVT on Holter, advanced echocardiographic techniques are under investigation. Sarvari et al used strain to evaluate 42 symptomatic ARVC patients and 27 mutation-positive asymptomatic individuals. RVMD was significantly more pronounced in ARVC patients with malignant arrhythmias compared with asymptomatic patients and even more pronounced than in healthy individuals. By multivariate analysis, RVMD independently predicted VT/ventricular fibrillation.

**Radionuclide Imaging**
Radionuclide imaging provides less substrate detail than echocardiography or CMR. Its strength is visualization and quantification of underlying pathophysiological processes predisposing to arrhythmias. Although indirect radionuclide assessment of arrhythmic risk has been described through determination of the extent and severity of perfusion imaging defects, cardiac autonomic innervation imaging is a promising method for directly assessing arrhythmic risk. Innervation abnormalities can contribute to initiating and sustaining arrhythmias by creating substrate abnormalities resulting in heterogeneity of sympathetic innervation, increasing systemic autonomic tone that can trigger an arrhythmia in denervated hypersensitive myocardium, and via modulation of central and regional autonomic tone that can accentuate electric heterogeneities of primary arrhythmic conditions.

Human work has been limited to assessing sympathetic innervation and uses analogues of norepinephrine.

---

**Figure 2.** Myocardial strain curves from the apical long-axis view in a healthy individual (left) and a long-QT syndrome (LQTS) patient (right). Contraction duration is homogeneous in all 6 segments in the healthy individual (370 ms). The LQTS patient shows prolonged contraction duration (460 ms) in the 3 septal segments (red, dark blue, and pink strain curves), indicating dispersed myocardial contraction. AVC indicates aortic valve closure. Reprinted from Haugaa et al with permission of the publisher. Copyright © 2010, Wolters Kluwer Health, Inc.
Clinically available radiotracers image presynaptic anatomy and function. Norepinephrine is synthesized in presynaptic terminals and stored in vesicles that on stimulation release the norepinephrine into the synaptic space for binding to postsynaptic membrane receptors, thereby increasing heart rate, augmenting contractility, and enhancing conduction effects.33,32 Active reuptake of norepinephrine into presynaptic terminals allows storage or disposal. Meta-iodobenzylguanidine, that is, mIBG, an analogue of the false neurotransmitter guanethidine, on intravenous administration, is taken into presynaptic terminals but is not metabolized and therefore accumulates.33 Labeling with 123-iodine (123I), allows visualization using a standard nuclear camera and depicts the functional state of cardiac adrenergic innervation. Various positron emission tomographic norepinephrine analogues have also been studied, having superior physical properties for imaging and closer similarities to norepinephrine providing biological advantages.30 11C-meta-hydroxyephedrine (HED) is most widely studied and better differentiates innervated from denervated myocardium.34 123I-mIBG image results are customarily reported as global cardiac uptake expressed as a quantitative heart-to-mediastinum ratio (HMR), tracer washout between early and delayed planar images, and regional tracer defects on tomographic images, often in reference to perfusion. HMR is most widely reported, suggested as akin to a biomarker, with a value ≥1.6 indicating a lower 1- to 2- year mortality risk.

Heart Failure

In systolic HF, HMR is a robust marker with lower values predicting increased adverse events, including symptomatic progression, higher likelihood of potentially lethal cardiac arrhythmias, cardiac death, and all-cause mortality.34–36 Focusing on arrhythmias, in a pilot study of advanced HF patients with an ICD, Arora et al37 found that those with a prior device discharge had a significantly lower HMR than those without (Figure 3). Nagahara et al38 found that SCD or an arrhythmic event associated with increased arrhythmic risk. In Arora et al,37 patients who had ICD discharges had more extensive 123I-mIBG and autonomic/perfusion mismatching defects (Figure 3). In patients undergoing ICD placement, Boogers et al43 found that 52% of those with an 123I-mIBG SPECT score >26 had subsequent ICD therapy versus 5% of patients with scores ≤26 (P<0.01). In patients referred for primary prevention ICD, Marshall44 et al reported that patients with arrhythmic events...
had higher late $^{123}$I-mIBG SPECT scores, with a threshold $\geq 31$ having 78% sensitivity and 77% specificity.

**Ischemic Heart Disease**

Innervation abnormalities are common in ischemic heart disease. Sympathetic neurons are more sensitive to ischemic oxygen deprivation than cardiomyocytes, and with nerve injury often persists beyond myocyte recovery. In postinfarct patients with LVEF $\leq 40\%$ referred for electrophysiological testing because of syncope or NSVT, Bax et al found that induced sustained VT was associated with a higher summed $^{123}$I-mIBG SPECT score. Zhou et al, based on CMR work showing that a larger border zone (mixture of viable and scar tissue) around myocardial scar increases arrhythmic susceptibility, reported that post-MI patients with LVEF $\leq 40\%$, who had lower $^{123}$I-mIBG border zone to mediastinum uptake ratios, were more likely to be electrophysiologically inducible (Figure 4).

Recurrent or chronic ischemia produces reversible neural stunning, termed dysinnervation, that seems to predispose to lethal arrhythmias. Canty et al developed a porcine model of ischemia and showed that dysinnervation is associated with an increase in arrhythmic susceptibility. CMR imaging allows for the assessment of myocardial substrate and provides a non-invasive method to predict arrhythmic risk. The use of CMR to assess myocardial substrate, such as late gadolinium enhancement (LGE), provides a means to visualize myocardial infarction and its spatial extent. The correlation between histological and CMR images has been shown to be excellent, with both imaging modalities providing valuable information for risk stratification.

**Figure 4.** Scar extent, border zone extent, and metaiodobenzylguanidined border zone/mediastinal ratio (BZ/M) in patients with prior myocardial infarction.

**Figure 5.** The use of cardiac magnetic resonance (CMR) to assess myocardial substrate: late gadolinium enhancement (LGE) in an animal study showing excellent correlation with histology. Myocardial infarction was induced in a dog by ligation of a coronary artery. Infarct location, spatial extent, and 3-dimensional shape determined by histology and LGE were essentially identical. Slices are arranged from base to apex starting in upper left and advancing left to right with the left panels of each pair containing triphenyltetrazolium (TTC)-stained slices and the right panels containing ex vivo CMR images. The 2 large images on the right are magnified views, with the TTC-stained image on the top and CMR on the bottom. Reprinted from Kim et al with permission of the publisher. Copyright © 1999, Wolters Kluwer Health, Inc.
model of myocardial hibernation and found that reductions in $^{123}$I-mIBG uptake that was greater in the subendocardium predisposed to arrhythmic SCD. Using $^{11}$C-HED, the Prediction of Arrhythmic Events with Positron Emission Tomography trial of 200 patients with ischemic cardiomyopathy found that the 4-year occurrence of SCD increased in relation to the severity/extent of $^{11}$C-HED tomographic abnormalities independently of B-type natriuretic peptide, symptoms, or LVEF.\textsuperscript{50}

### Primary Arrhythmic Conditions

Adrenergic image abnormalities are found in primary ventricular arrhythmias. Wichter et al\textsuperscript{51} found $^{123}$I-mIBG defects in Brugada syndrome, often localized to the inferior and inferoseptal walls. Paul et al\textsuperscript{52} reported focal SPECT $^{123}$I-mIBG defects in the left ventricle—mostly inferior, septal, and anterior—in 59\% of 42 patients with ARVC, with life-threatening ventricular tachyarrhythmias more frequent and leading to worsened survival independent of the extent of RV dysfunction.

### Cardiac MRI

#### Tissue Characterization

Pathological and electrophysiological substrate predisposing to life-threatening ventricular arrhythmias has been well characterized. Inhomogeneous scarring with varying degrees of subendocardial myocardial fiber preservation within dense zones of fibrosis results in slowed conduction, nonuniform anisotropy, and the presence of conduction channels leading to re-entrant VT.\textsuperscript{53,54} Having superb imaging quality with high temporal and spatial resolution in even the most challenging patients, CMR provides excellent visualization and characterization of substrate. The most useful technique is late gadolinium enhancement (LGE) imaging, which highlights areas of fibrosis and scarring.

**Figure 6.** The use of cardiac magnetic resonance (CMR) to identify infarct core and border zone regions. Diastolic (A) and systolic (B) cine images show akinesis of the anteroseptal and anterior walls indicative of infarction (endocardial border is blue and epicardial border is red). C and D, Computer-assisted semi-automatic quantification of \% late gadolinium enhancement (LGE) in the infarct territory (white arrows). The infarct core region was defined as an LGE signal intensity >3 SD above normal segments (red region) and the peri-infarct gray zone defined as an LGE signal intensity 2 to 3 SD above normal segments (yellow region). Reprinted from Yan et al\textsuperscript{61} with permission of the publisher. Copyright © 2006, Wolters Kluwer Health, Inc.
Risk of SCD and Cardiovascular Events in ICM

Scar and Scar Burden for Prediction of Ventricular Arrhythmias and SCD
The value of LGE was first demonstrated by Bello et al.57 with infarct size by LGE shown better than LVEF in predicting electrophysiological inducible VT. Kwong et al.58 showed that LGE was the strongest predictor of MACE in patients with CAD, with scars as small as a mean of 1.4% of LV mass having >7-fold increased risk. More recently, in cardiomyopathy patients, Dawson et al.59 found LGE to be an independent predictor of MACE, with scars as small as a mean of 1.4% of LV mass having >7-fold increased risk. More recently, in cardiomyopathy patients, Dawson et al.59 found LGE to be an independent predictor of MACE, and by multivariate analysis, LGE was the only predictor of NSVT, whereas LVEF was not.

An important question is whether CMR can guide decision-making, such as ICD selection. In a study population with mixed cardiomyopathic causes, in patients with LVEF>30%, scar that was >5% of the LV mass on CMR identified a high-risk cohort similar in risk to patients with LVEF<30%.60 Conversely, patients with LVEF<30% and minimal or no scar had low risk similar to those with LVEF>30%. Scar burden resulted in a 32% net reclassification. LGE was superior to EPS for predicting end points on multivariable analysis. Such stratification is relevant when weighing the risk/benefit ratio regarding ICD placement.

Peri-Infarct Border Zone in Relation to Ventricular Arrhythmias and SCD
In ICM, electric mapping has revealed that reentrant VT usually originates from the subendocardial surface of infarcted myocardium adjacent to dense scar. CMR characterization of scar zones may provide additional insights into arrhythmias. It has been postulated that intermediate intensity regions (gray zones) on LGE represent more arrhythmogenic heterogeneous tissue containing viable and nonviable myocardium, with evidence that increased gray zone mass predicts ICD shock, VT inducibility on EPS, and mortality (Figure 6).48,61

Nevertheless, the predictive role of the gray zone has been inconsistent, and it is unclear whether there is a scar threshold effect. A variety of arrhythmogenic risk thresholds have been reported using LGE, ranging from 1.4% to 8.1% of LV mass.59,61,62 Possible explanations for the inconsistency are small sample sizes, differing patient characteristics, and lack of standardization for grading gray zones. In addition, although some gray zones may represent true mixed scar and viable muscle islands, others might be imaging artifacts from volume averaging. Finally, scar size, the number of scars, and scars from different causes may have different arrhythmogenic potential.

Risk of SCD and Cardiac Events in NICM
Assomull et al.63 were the first to assess the prognostic value of CMR midwall fibrosis in NICM patients. Fibrosis was associated with higher all-cause death, SCD, and VT rates, and by multivariate analysis, fibrosis was the sole predictor of MACE. Wu et al.64 reported similar results in NICM patients referred for primary ICD implantation, with 44% of patients with LGE having a MACE versus 8% of those without.

Adjusting for LV volume and functional class, LGE patients had an 8-fold higher risk of MACE. Gulati et al.65 reported a 26.8% death rate among patients with fibrosis versus 10.6% among those without, with an arrhythmic composite of 29.6% versus 7.0% patients, respectively. Fibrosis and its extent were

Table. Uses, Strengths, and Limitations of Noninvasive Imaging for Predicting Arrhythmic Risks

<table>
<thead>
<tr>
<th>Noninvasive Imaging</th>
<th>Uses</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radionuclide imaging</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac magnetic resonance</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
independently associated with all-cause mortality and other events, significantly improving risk reclassification beyond LVEF.

**CMR and Prognosis for HCM**

Several investigators have reported the prognostic implications of fibrosis detected by LGE in patients with HCM. Rubinshtein et al found that patients with a positive gene mutation were more likely to have fibrosis correlating with a higher frequency of premature ventricular complexes and NSVT. In addition, LGE predicted increased risk of SCD and ICD therapy (0.94% per year versus 0%), with the predictive power of LGE remaining significant after controlling for other SCD risk factors.66

In an international study of ≈600 patients, Chan et al67 showed that the extent of LGE was independently associated with increased SCD risk, with a 1.25 odds ratio for every 5% LGE increase, despite 8 of the 11 SCD patients not having any clinical risk factors. The absence of LGE was associated with a low event rate. However, LGE in HCM has a poor positive predictive value.

**CMR in Other Uncommon Cardiac Conditions**

CMR is increasingly used as a diagnostic tool in a variety of myocardial diseases, including myocarditis, sarcoidosis, amyloidosis, and ARVC.68,69 CMR imaging also provides information beyond established prognostic indicators, thereby providing additional risk stratification and predicting disease progression and response to treatment. In 222 biopsy-proven viral myocarditis patients, LGE yielded a hazard ratio of 8.4 for all-cause mortality and 12.8 for cardiac mortality, independent of follow-up clinical symptoms, and was superior to LVEF, LVEDV, or NYHA class.70 No patient without LGE experienced SCD even if the LV was enlarged, and systolic function was impaired. Schumm et al71 reported similar findings.

**CMR and Sudden Cardiac Arrest Survivors**

Identifying the cause of sudden cardiac arrest in survivors is challenging. Even after extensive investigation with electrocardiography, echocardiography, and coronary angiography, the cause is often unclear. Neilan et al62 investigated 137 sudden cardiac arrest patients, finding that LGE identified scar in 71% of patients and provided a potential arrhythmic substrate in 76%, including an infarct-pattern LGE in 44%, noninfarct LGE in 15%, myocarditis in 10%, HCM in 2%, sarcoidosis in 2%, and ARVC in 2%. By multivariate analysis, the presence and the extent of LGE was the strongest predictor of recurrent events, whereas LVEF was not predictive.

**Cardiac Multidetector Computed Tomography**

Similar to CMR, cardiac multidetector computed tomography can provide high resolution information about myocardial substrate predisposing to high-risk arrhythmias. Multidetector computed tomography may be particularly useful in patients with implanted metal hardware for whom CMR is contraindicated. The scope of this article precludes discussion of this topic that is reviewed in detail by Dickfeld.72

**Uses, Strengths, and Limitations of Noninvasive Imaging in Ventricular Arrhythmias**

Table shows potential clinical uses, strengths, and weakness of echocardiography, radionuclide imaging, and CMR in predicting ventricular arrhythmic risk. Each modality has demonstrated ability for arrhythmic risk stratification that has been shown to be as good as or better than customarily used.
parameters, such as LVEF. It is unclear regarding which imaging modality would be best for a particular cardiac disease as there are no comparative studies. As each technique depicts different factors related to arrhythmic risk, they are likely to be complementary.

Of course, risk stratification alone is insufficient for clinical management, and we need to demonstrate that imaging leads to treatments that decrease mortality and improve well-being. Although the current principal management of ventricular arrhythmias is prophylactic ICD, because of the acknowledged deficiency of the current patient selection approach based predominantly on LVEF, many investigators are focusing on the potential for imaging to better select patients for the device. As data for each imaging technique are from observational studies, they are insufficient for recommending ICD implantation guidance. Although one can cite flaws in key ICD randomized studies and discuss conceptual difficulties using LVEF as the crucial decision variable, it is widely understood that prospective, randomized trials are required before these novel imaging techniques can be incorporated into established guidelines. In lieu of such studies, it may be reasonable to use imaging to assist decision making for patients in whom ICD benefit is uncertain, but there has been no study in this regard.

Another potential use of imaging is guidance of ventricular arrhythmia ablation. A recent report by Klein et al suggests that concurrent 3D innervation mapping based on 123I-mIBG may enhance procedure success beyond electroanatomical mapping. Figure 7 illustrates a case in which an inferior septal ablation site was within an area of denervation (close to a denervation/transition zone) but preserved bipolar voltage.

For CMR, in a swine MI model, Ashikaga et al reported that a VT isthmus identified by VT activation patterns corresponds to LGE scar detected by high-resolution 3D CMR. In NICM patients referred for ablation, Bogun et al found that critical VT sites identified by electrophysiological mapping occurred within LGE scar areas, with scar location able to guide catheter ablation. Dickfeld et al reported that CMR can provide supplementary guidance to facilitate substrate-guided VT ablations. Intraprocedure real-time CMR, together with the ability to visualize complex 3D arrhythmogenic anatomy and target additional regions of incomplete lesion formation, may allow for better outcome.

Technical advances will enhance all modalities. For echocardiography, incorporation of 3D strain analysis seems to improve risk stratification beyond 2D. Radionuclide imaging can benefit from the higher tissue resolution afforded by position emission tomographic tracers. Advances in CMR using T1-mapping with or without gadolinium promise to allow better delineation for distinguishing focal abnormal areas from diffuse processes.

Disclosures
Dr Travin has received research funding from GE Healthcare and has been on advisory boards for GE Healthcare. The other authors report no conflicts.

References
Prediction of Arrhythmic Risk With Imaging


29. Inoue H, Zipes DP. Value of myocardial 123I-metaiodobenzylguanidine paramet-


Novel Imaging Approaches for Predicting Arrhythmic Risk
Mark I. Travin, DaLi Feng and Cynthia C. Taub

Circ Cardiovasc Imaging. 2015;8:
doi: 10.1161/CIRCIMAGING.115.003019
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2015 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/8/11/e003019

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Imaging can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Imaging is online at:
http://circimaging.ahajournals.org//subscriptions/