Cardiac Implantable Electronic Device

Heart Failure

นพ. ธรณิศ จันทรารัตน์
ราชamma hospital
Scope of presentation

- Impact of Problem
- Mechanism of SCD- Ventricular arrhythmia
- Prevention according to guidelines
Pathophysiology-Structure

Inherited cardiomyopathies:
- Long QT, short QT, WPW, Brugada, ARVC, HCM, DCM
- Common genetic variants

Prior infarct

Pre-existing myocardial damage

Figure 1

Scheme of drivers for arrhythmias in acute coronary syndromes. A pre-existing substrate for ventricular arrhythmias, either secondary to an old myocardial infarction, due to a cardiomyopathy, or secondary to a genetic predisposition to ventricular arrhythmias, interacts with acute ischaemia, autonomic tone, and acute left ventricular strain to create triggered activity and ventricular arrhythmias.

ARVC, arrhythmogenic right ventricular cardiomyopathy; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; VF, ventricular fibrillation; VT, ventricular tachycardia; WPW, Wolf-Parkinson-White syndrome (Adapted from Heart, Kirchhof P, Breithardt G, Eckardt L. Primary prevention of sudden cardiac death, 92, 1873–8, Copyright 2006, with permission from BMJ Publishing Group Ltd and from J Cardiovasc Pathol, 19, Basso C, Rizzo S, Thiene G. The metamorphosis of myocardial infarction following coronary recanalization, 22–8, Copyright 2010, with permission from Elsevier).
Dilated Cardiomyopathy

Genetic susceptibility

Genome

- Chemotherapy Anthracyclines & Monoclonal Abs
- Alcohol
- Pregnancy
- Endocrine Thyroid & Phaeo
- Inflammatory
- Infection Myocarditis & HIV
- Idiopathic

Figure 3. Detecting myocardial fibrosis using cardiovascular magnetic resonance. This figure demonstrates the acquired and genetic insults implicated in the pathogenesis of dilated cardiomyopathy (DCM) followed by the common genetic mutations associated with DCM (incidence of mutations in cases of idiopathic DCM followed by the protein encoded by the gene $2$) and data on disease outcomes. Abs indicates antibodies; LVRR, left ventricular reverse remodeling; phaeo, phaeochromocytoma; and SCD indicates sudden cardiac death.
Arrhythmogenic substrate prior to the index event

Most patients survive their first ACS. Owing to progression of arteriosclerosis, a second acute event will often occur despite maximal preventive therapy. Patients who suffer from an acute coronary event with pre-existing reduced LV function and myocardial scars are at risk for sustained VA in the acute and sub-acute phase of a MI. Echocardiographic signs of markedly reduced LV function or ECG signs of an old MI can identify such patients. Furthermore, patients with increased sympathetic activity, or taken to the extreme of cardiogenic shock, are at increased risk of sustained VA in the setting of a ‘recurrent’ acute coronary event. Recent evidence

Inherited cardiomyopathies: 
- Long QT, short QT, WPW, Brugada, ARVC, HCM, DCM

Common genetic variants

Prior infarct

Pre-existing myocardial damage

Acute ischaemia

Autonomic imbalance

Acute strain

Cellular and tissue proarrhythmia
- scar
- focal fibrosis
- hypertrophy
- altered ion homeostasis
- loss of intercellular connection

Electrical Instability

VT

VF
Pathophysiology

Pathophysiologica Cycle of Ventricular Arrhythmias and Progressive Pump Failure

- Myocardial Adverse Remodeling
- Hemodynamic decompensation with peripheral hypoperfusion
- "Peripheral" Metabolic adaptation
- Systemic Insulin Resistance

- Dyssynchronous myocardium (RV + LV)
- Mechanical / Bioenergetic Uncoupling
- Myocardial ("central") Metabolic Adaptation
  - shift back to lipids/ketones

Structural Electrical Neurohormonal

Heart Failure Disease Progression

ACC/AHA Stage

B C D

Santangeli et al. JACC VOL. 69, NO. 14, 2017
Management of VT in Advanced HF APRIL 11, 2017:1842

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Arrhythmogenesis

- changes in electrical, structural, neurohormonal

- **Remodelling** (Maladaptive) processes and arrhythmogenic events
  - renin angiotensin aldosterone system (RAAS),
  - beta-adrenergic pathway,
  - Ca-Calmodulin-dependent kinase II (CaMKII)-
  - calcineurin-mediated signalling
Ionic/Electrical change

- ↓ potassium (K) channels

- ↑ sarcolemmal Na/Ca exchanger

- ↑ Ca-stored intracellularly in the sarcoplasmic reticulum (SR) and Ca turnover

- ↑ late Na current and ↓ transient outward current (Ito)
Ca-Mechanism
Late $I_{Na}$ Mechanism

$\uparrow Na$

$\uparrow Ca$

Na/Ca exchanger

DAD

PVC

PAC

Ca

Na/Ca exchanger

Late $I_{Na}$
Triggers

• The ‘external’ triggers of arrhythmias

  • mechanical stretch (volume)

  • neurohumoral activation

  • stressors (e.g. systemic inflammation)
Phase of LV injury

Initiating event:
Acute MI, valve abnormality, toxin, virus etc.

Cardiac output

Time

Compensated
Temporarily compensated
Decompensated
Decompensated

Arrhythmias

Figure 1 Arrhythmogenesis during the transition to chronic heart failure.

EHRA/HFA joint consensus document on arrhythmias in heart failure
Risk Stratification Technique

Competing risks of non-sudden death

SCD risk

Clinical factors
- Co-morbidity
- Heart failure stage

ECG variables
- Electrical instability
- MTWA

Echo
- Cardiac function

LGE-CMR and T1-mapping
- Structural substrate
- Myocardial fibrosis

Cardiac MIBG
- Autonomic dysfunction

Genetic testing
- Genetic pre-disposition

For those with a life expectancy <1 year.

The risk of death from nonsudden causes is especially relevant in older patients and in those with more comorbidities. In planned subgroup analysis of the DANISH trial, patients >68 years of age had a trend toward increased mortality with ICD implantation (HR, 1.19; 95% CI, 0.81–1.73; \( P = 0.38 \)), in contrast to patients <59 years of age who had a lower mortality with an ICD (HR, 0.51; 95% CI, 0.29–0.92; \( P = 0.02 \)).

In addition, a meta-analysis of trials of primary prevention ICDs in ischemic and nonischemic HF demonstrated the absence of survival benefit in patients with an estimated glomerular filtration rate of <60 mL·min\(^{-1}\)·1.73 m\(^{-2}\). This highlights the role that age and measures of kidney function may have in identifying patients who are unlikely to gain benefit from ICD implantation.

Risk scores such as the Seattle Heart Failure Model have been developed to predict prognosis in patients with HF, incorporating variables such as NYHA class and prescription of medical therapies with age and kidney function. The Seattle model has been shown to be more accurate in the stratification of the risk of nonsudden death compared with SCD in populations with ischemic and nonischemic HF.

For example, patients with a score of 3 and 4 compared with those with a score of 0 have a relative risk of HF death of 38.4 and 87.6 and a relative risk of SCD of only 6.5 and 6.5, respectively. This highlights that although the risk of SCD rises with worsening HF, the rise in the risk of HF death is even greater, reducing the chances of gaining quality-adjusted life-years from ICD therapy.

Although similar models have been developed for the prediction of SCD in HF populations and the wider general population, they are limited by an inability to reliably discriminate between the risk of SCD and nonsudden death and therefore have limited clinical utility.

There is growing interest in the use of circulating biomarkers of myocardial stress and fibrosis such as natriuretic peptides, troponin, galectin-3, and soluble ST2 to predict prognosis. However, these biomarkers generally reflect the severity of cardiac dysfunction rather the specific risk of SCD. They may be used to identify patients who are unlikely to benefit from ICD therapy because of a high risk of death resulting from the progression of HF. In prespecified subgroup analysis of DANISH, patients with a N-terminal pro-B-type natriuretic peptide >1177 pg/mL randomized to ICD therapy had an all-cause mortality similar to that of those in the control arm (HR, 0.99; 95% CI, 0.73–1.36; \( P = 0.96 \)), whereas mortality was lower in those assigned to an ICD when N-terminal pro-B-type natriuretic peptide was <1177 pg/mL (HR, 0.59; 95% CI, 0.38–0.91; \( P = 0.02 \)).

Similarly, Ahmad and colleagues demonstrated a stronger association between N-terminal pro-B-type natriuretic peptide, galectin-3, and soluble ST2 and HF death compared with SCD in patients with ischemic and nonischemic HF. In summary, biomarkers, in combination with clinical variables and prognostic scores, offer the most potential for the identification of those at high risk for SCD.
MARKERS OF ELECTRIC INSTABILITY

The results of these, often small, studies have been inconsistent, and their combined utility is limited by the wide variability in end points, including SCD, ventricular arrhythmia, or arrhythmia that is not sustained. Although interstudy reproducibility was poor for many studies, the majority of the remaining parameters were between 1.5 and 3.0, suggesting lower predictive value (\( \text{OR} = 2.92 \); 95% CI, 2.17–3.93). In the era of global and mechanical dyssynchrony, to predict sustained ventricular arrhythmia and SCD, diastolic function, imaging, and electrical markers are used as an alternative when they were not available. Although the small number of studies limits the ability of global and mechanical dispersion, a meta-analysis of patients with nonischemic DCM has corroborated the findings of Goldberger and colleagues, who have proposed the use of microvolt T-wave alternans (MTWA), and a meta-analysis of patients with nonischemic DCM over 22 months by Haugaa and colleagues have shown that longitudinal strain and mechanical dispersion, a measure of MTWA, and a meta-analysis of patients with nonischemic DCM in an attempt to predict sustained ventricular arrhythmia and SCD was investigated in 94 patients on 

Table 2.

<table>
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<tr>
<th>Parameter</th>
<th>Positive</th>
<th>Value, ( \text{P} )</th>
<th>Predictive Strength, ( \text{OR} ) (95% CI)</th>
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<tbody>
<tr>
<td>Fragmented QRS</td>
<td>9</td>
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<td>1.51 (1.13–2.01)</td>
<td>18.5</td>
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<td>Positive signal-averaged electrocardiogram</td>
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EMPIRICAL EVIDENCE

The value of electrical markers to predict arrhythmic events has been consistently low in most studies. The ability of global and mechanical dispersion, a measure of MTWA, and a meta-analysis of patients with nonischemic DCM over 22 months by Haugaa and colleagues have shown that 

STATE OF THE ART

The use of different end points.

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Halliday et al. 

July 11, 2017


220 curve analyses for the prediction of the primary outcome compared with LVEF (area under the curve: strain, 0.82; mechanical dispersion, 0.80; LVEF, 0.72). Another study investigated 124 patients with nonischemic DCM before primary-prevention ICD implantation. Longitudinal strain was independently associated with the primary end-point of appropriate ICD therapy, albeit to a modest degree (per 1% increase: HR, 1.12; 95% CI, 1.01–1.20; \( P =0.032 \)). Importantly, however, it appears unlikely that functional techniques such as strain measurement will provide adequate discrimination between the risk of SCD and death resulting from HF.

THE ROLE OF MYOCARDIAL FIBROSIS IN SCD RISK STRATIFICATION

One of the characteristic pathological features of DCM is the formation of myocardial fibrosis, a consequence of an increase in collagen formation in the extracellular matrix and myocyte cell death.

Histological studies have demonstrated 2 forms of fibrosis: replacement and interstitial fibrosis. Replacement fibrosis describes discrete areas of myocardial scarring that develop as a result of myocyte cell death, whereas interstitial fibrosis is the result of expansion of the interstitium with accumulation of collagen in the absence of cell death (Figure 2).

Fibrosis is the result of activation of the renin-angiotensin-aldosterone system and the \( \beta \)-adrenergic axis, which occurs as part of the HF syndrome. Other environmental insults, implicated in the origin of DCM such as chemotherapy and viral myocarditis, play a role through the activation of inflammatory networks and the production of reactive oxygen species. The result is the activation of myofibroblasts, the production of collagen, and myocyte cell death.

Fibrosis is thought to provide a substrate for ventricular arrhythmia. An electric mapping study in patients with DCM demonstrated that only those with replacement fibrosis, identified by late gadolinium-enhanced imaging, were at increased risk for appropriate ICD therapy.

Figure 2. Detecting myocardial fibrosis using cardiovascular magnetic resonance.

A, Late gadolinium enhancement cardiovascular magnetic resonance image of a midventricular short-axis slice in a healthy control subject.

B, Native T1 map of a midventricular short-axis slice in a healthy control with a mean myocardial T1 of 1240 milliseconds.

C, Late gadolinium enhancement image of a midventricular short-axis slice in a patient with dilated cardiomyopathy (DCM) demonstrating linear midwall enhancement.

D, Native T1 map of a midventricular short-axis slice in a patient with DCM with a mean myocardial T1 of 1375 milliseconds. Scans were performed on a 3-T Siemens Skyra (Erlangen, Germany).

E, Microscopic examination of a sample taken from the septum of an explanted heart after transplantation demonstrating the presence of replacement fibrosis (blue arrow) and pericellular interstitial fibrosis (yellow arrow).

by JENNIFER ADGEY on August 4, 2017

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Genome specific

**Nuclear Envelope**
- LMNA (6%, Lamin A/C)
- Malignant phenotype; high rate of ventricular arrhythmia even in early disease

**Sarcomeric**
- TTN (25%, Titin)
- MYH6 (4%, α-myosin heavy chain)
- MYH7 (4%, β-myosin heavy chain)
- TNNT2 (3%, Troponin T)
- MYPC (3-4%, Myosin phosphatase)
- TNNT1 (<1%, Troponin C)
- TNNT2 (<1%, Troponin I)

**Sarcoplasmic reticulum**
- PLN (<1%, Phospholamban)

**Cystoskeleton**
- DMD (N/A, Dystrophin)
- DES (<1%, Desmin)
- Filamin (N/A, FLNC)

**Spliceosomal**
- RBM20 (2%, RNA-binding protein)

**Ion Channels**
- SCN5A (2-3%, Sodium channel)

**Mitochondrial**
- TAZ (N/A, Tafazzin)

High Rate of VA

Nuclear Envelope (LMNA)
Sarcomeric (MYH 6,7)
Sarcoplasmic Reticulum (PLN)
Invesigations in VA patients

- ECG, EST, Imaging CT/CMR
- EPS

Electrophysiological study in patients with CAD is recommended for diagnostic evaluation of patients with remote myocardial infarction with symptoms suggestive of ventricular tachyarrhythmias, including palpitations, presyncope and syncope.

Electrophysiological study in patients with syncope is recommended when bradyarrhythmias or tachyarrhythmias are suspected, based on symptoms (e.g. palpitations) or the results of non-invasive assessment, especially in patients with structural heart disease.

Electrophysiological study may be considered for the differential diagnosis of ARVC and benign RVOT tachycardia or sarcoidosis.

**Level of evidence.**

- **I**
- **IIb**
- **Class.**

- **I**
- **IIa**
- **I**
Ischemic and Nonischemic Cardiomyopathy

- Premature ventricular complexes
- Non-sustained ventricular tachycardia
- Sustained monomorphic ventricular tachycardia
- Sustained polymorphic ventricular tachycardia/ventricular fibrillation
Monomorphic VT

Evaluate cardiac structure and function

SHD

Treat SHD as appropriate ICD if indicated

Non-Ischaemic SHD

Optimize ICD programming antiarrhythmic drugs preferred first line

Catheter ablation when drug-refractory

No SHD

Amiodarone Beta Blockers

Xylocaine

SHD from idiopathic VT

Ischaemic SHD

Catheter ablation may be considered first line

Beta Blockers, or catheter ablation: may all be considered first line

(ICI required for rare malignant idiopathic VT)

Transient myocardial ischaemia is an uncommon sole cause of episode. Testing for ischaemia with only modest positive predictive value. Cardiac MRI and positron emission tomography-computed angiography should be considered. Artery disease is suspected as the presence or absence of underlying heart disease, which includes echocardiography, exercise testing, and anatomical mapping of the RV has been used to identify otherwise unapparent RV scar. An e.g. at a session with bet better prognosis has been associated with recurrent VT and may provide clues to the cause of the episode. Patients presenting with syncope or sustained palpitations often occurring many years earlier. However, treatment is largely based on the patient’s symptoms and haemodynamic response. For patients with a new fixed region of myocardial scar that is a sequela of prior MI, the acute treatment of sustained VT is only “15%.

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Polymorphic VT

**Trigger PMVT or VF.** Use of the Valsalva manoeuvre or high precordial leads may improve the sensitivity of the 12-lead ECG for detecting such triggers.

In addition, the QRS and QT changes occurring after extrasystoles as well as during standing may help to identify J-wave abnormalities or abnormalities of the QT interval. Ambulatory monitoring may help identifying QTc prolongation during sleep.

The role of genetic testing has been recently reviewed and plays an important part in the evaluation of patients in whom an inherited arrhythmia syndrome is suspected.

Adjuvant therapy to reduce ICD shocks

<p>| Conditions that can cause PVT/VF in the absence of SHD and potential therapies |</p>
<table>
<thead>
<tr>
<th>Clues</th>
<th>Tests to Consider</th>
<th>Diagnoses</th>
<th>Therapies</th>
</tr>
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<tbody>
<tr>
<td>Long QT/T-wave alternans ECG/Monitor</td>
<td>Congenital LQTS Beta-blockers/stellatectomy</td>
<td></td>
<td></td>
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<tr>
<td>TdP pattern Epinephrine challenge</td>
<td>Avoid QT prolonging drugs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of seizures Genetic testing</td>
<td>Mexilitine/flecainide (LTQ3)</td>
<td></td>
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<tr>
<td>Speciﬁc trigger (loud noise) Pacemaker/ICD</td>
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<td>Long QT/T-wave alternans ECG/Monitor</td>
<td>Acquired LQTS Mg²⁺/K⁺</td>
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<tr>
<td>TdP pattern</td>
<td>Stop offending drug</td>
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<tr>
<td>Renal failure</td>
<td>Temporary pacing</td>
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<td>New medication or drug abuse</td>
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<tr>
<td>AV block ECG/Monitor</td>
<td>Bradycardia Pacemaker</td>
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<tr>
<td>Incomplete RBBB with STE in leads V1–V2 ECG</td>
<td>BrS Isoproterenol/quinidine</td>
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<td>Fever Drug challenge</td>
<td>Anipyretic</td>
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<td>Genetic testing Ablation</td>
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<td>ICD</td>
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<td>Monomorhic PVC trigger ECG/Monitor</td>
<td>Focal PVC origin Ablation/ICD</td>
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<td>J-point elevation ECG</td>
<td>Early repolarization ICD</td>
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<td>Ventricular pre-excitation ECG</td>
<td>WPW Ablation</td>
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<tr>
<td>Short QT interval ECG</td>
<td>Short QTS ICD</td>
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<td>Bidirectional VT pattern exercise-induced</td>
<td>Digoxin level CPVT Stop digoxin</td>
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<tr>
<td>STE and chest pain Proactive testing</td>
<td>Coronary spasm Vasodilators/coronary stent ICD</td>
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<tr>
<td>Short-coupled PVC trigger ECG/Monitor</td>
<td>Idiopathic ICD</td>
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<tr>
<td>BrS = Brugada syndrome; CCBs = calcium channel blockers; CPVT = catecholaminergic polymorphic ventricular tachycardia; ICD = implantable cardioverter-deﬁbrillator; LQTS = long QT syndrome; PVC = premature ventricular complex; RBBB = right bundle branch block; short QTS = short QT syndrome; STE = ST elevation; TdP = torsade de pointes; VT = ventricular tachycardia; WPW = Wolff-Parkinson-White (syndrome).</td>
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### AAD in CHF

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<tr>
<th>Class and Drug</th>
<th>ABP</th>
<th>PCWP</th>
<th>CO</th>
<th>SVR</th>
<th>Predominant Effect</th>
<th>Vasodilation</th>
<th>Myocardial Depression</th>
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Structural
Electrical
Neurohormonal