Prevention of Sudden Cardiac Death

Introduction and Case Discussion

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Sudden Cardiac Death and Outcomes

- Sudden cardiac death (SCD) accounts for ~15% of total mortality in US.
- The rhythms most often recorded (75-80%) at the time of SCD are ventricular tachycardia (VT) and fibrillation (VF).
- Pts survived from SCD rarely recovery without residual neurologic damage.
Definition

**Cardiac arrest** with cessation of cardiac function, whether or not resuscitation or spontaneous reversion occurs.

An unexpected death from a cardiac cause occurring within 1 hour of onset of symptoms.
Sudden Cardiac Death due to Ventricular Fibrillation

Defibrillation:

High success rate

Low success rate without Chest Compressions

68 Y/O male with HT and CAD

Chen, Wu et al. *Circulation* 2003
Aborted Sudden Cardiac Death
# Sudden Death

<table>
<thead>
<tr>
<th>Country</th>
<th>Rate per 100,000 / year</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.S.A.</td>
<td>100-200 / 100,000 / year</td>
</tr>
<tr>
<td>Japan</td>
<td>68-104 / 100,000 / year</td>
</tr>
<tr>
<td>Netherlands</td>
<td>100 / 100,000 / year</td>
</tr>
<tr>
<td>Greece</td>
<td>90 / 100,000 / year</td>
</tr>
<tr>
<td>Taiwan</td>
<td>58~73 / 100,000 / year</td>
</tr>
<tr>
<td>Iceland</td>
<td>56 / 100,000 / year</td>
</tr>
<tr>
<td>Finland</td>
<td>48 / 100,000 / year</td>
</tr>
<tr>
<td>Israel</td>
<td>46 / 100,000 / year</td>
</tr>
<tr>
<td>England</td>
<td>40 / 100,000 / year</td>
</tr>
</tbody>
</table>

*(Huang et al. *Acta Cardiol Sin* 2006)*
OHCA in Taipei
First Rhythm

69.1%

12.6%

18.3%

VT/VF
PEA
Asystole

Epidemiology

• 80-90% are due to ventricular arrhythmias.

• 85-90% are due to 1st event, 10-15% due to recurrent events.

• Survival rates for out of hospital arrest <5%.
## Ambulatory ECG monitoring findings in 157 cases of sudden cardiac death

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular tachyarrhythmia</td>
<td>83.4</td>
</tr>
<tr>
<td>VT/VF</td>
<td>62.4</td>
</tr>
<tr>
<td>Primary VF</td>
<td>8.3</td>
</tr>
<tr>
<td>Torsade polymorphic VT</td>
<td>12.7</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>16.5</td>
</tr>
</tbody>
</table>
Time References in SCD

**Prodromes**
- New or worsening cardiovascular symptoms
  - Chest pain
  - Palpitations
  - Dyspnea
  - Fatiguability
  Days-to-months

**Onset of terminal event**
- Abrupt change in clinical status
  - Arrhythmia
  - Hypotension
  - Chest pain
  - Dyspnea
  - Lightheadedness
  Up to 1 hour

**Cardiac arrest**
- Sudden collapse
  - Loss of effective circulation
  - Loss of consciousness
  Minutes-to-weeks

**Biological death**
- Failure of resuscitation OR
- Failure of electrical, mechanical, or CNS function after initial resuscitation

*Heart Disease*, 7th edition, C 33.

- 90% of SD: Cardiac causes
- 80% of SCD: CAD
- 10-15% of SCD: CM
Precipitating Factors

- Transient alterations of coronary blood flow
- Vasomotor dynamics
- Acute (transient) ischemia
- Reperfusion after ischemia
- Systemic factors
- Hemodynamic failure
- Hypoxemia, acidosis
- Electrolyte imbalance
- Neurophysiological interactions
- Transmitters, receptors
- Central influences
- Toxic effects
  - Proarrhythmic drugs
  - Cardiac toxicity

Structure

- Myocardial infarction
  - Acute
  - Healed
- Aneurysm
- Hypertrophy
  - Secondary
  - Primary
- Myopathic ventricle
  - Dilation, fibrosis
- Infiltration
- Inflammation
- Structural electrical abnormality

(Substrates)

Electrogenic theory

- PVCs
- VT/VF (80%)

(Triggers)

Function

- Asystole
- AVB
- PEA

AJC 1989

Heart Disease, 7th edition, C 33.
Major Causes of Sudden Cardiac Death

Ischemic heart disease
- Coronary artery disease with myocardial infarction or angina
- Coronary artery embolism
- Nonatherogenic coronary artery disease (arteritis, dissection, congenital coronary artery anomalies)
- Coronary artery spasm

Nonischemic heart disease
- Hypertrophic cardiomyopathy
- Dilated cardiomyopathy
- Valvular heart disease
- Congenital heart disease
- Arrhythmogenic right ventricular dysplasia
- Myocardiitis
- Acute pericardial tamponade
- Acute myocardial rupture
- Aortic dissection

No structural heart disease
- Primary electrical disease (idiopathic ventricular fibrillation)
- Brugada syndrome (right bundle branch block and ST segment elevation in leads V1 to V3)
- Long QT syndrome
- Preexcitation syndrome
- Complete heart block
- Familial sudden cardiac death
- Chest wall trauma (commotio cordis)

Noncardiac disease
- Pulmonary embolism
- Intracranial hemorrhage
- Drowning
- Pickwickian syndrome
- Drug-induced
- Central airway obstruction
- Sudden infant death syndrome
Severity of Heart Failure
Mode of Death

SCD is a major mode of death in heart failure patients.

Mechanisms of Cardiac Fibrillation:

"Multiple-Wavelet" Hypothesis
Moe 1962

"Focal-Source" Hypothesis   "Mother-Rotor" Hypothesis
Prinzmetal 1950   Jalife et al. 1998

Competing rather than complimentary
Example of short-lived reentry during VF

Evaluation of Survivors of SCD

- Hypothermic therapy
- Transient and reversible causes
- Evaluation for structural heart disease
- SCD in those without structural heart disease
- Family history in hereditary arrhythmic disease
• Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32-34 °C for 12-24 h when the initial rhythm was VF (Class I, LOE B).
A  Baseline 37°C  
B  Hypothermia 30°C  
C  Rewarming 37°C  

Site  x  y  

Local Optical Recordings  

Stop S1 pacing  

SDA before VF !!  

Spontaneous rhythm  

Stop S1 pacing  

Spontaneous rhythm  

T-Cx43 distribution (lateralization) during MH and SH

Figure 3

A

Baseline (37°C)  MH (33°C)  SH (30°C)  Rewarm (37°C)

B

T-Cx43 area (% tissue area)

C

% Lateral / Total T-Cx43

Transient or reversible causes

- Acute cardiac ischemia and myocardial infarction
- Anti-arrhythmic drugs
- Medication (eg, QT prolonging drugs), toxin, or illicit drug ingestion
- Electrolyte abnormalities, most notably hypokalemia, hyperkalemia, and hypomagnesemia.
- pH changes, especially acidosis
Risk Stratification of SCD


2. LV dysfunction: CAD and NICM.

3. ECG abnormality: AVB or IVCD in survivors of cardiac arrest; prolonged QTi, increased QTi dispersion, resting HR>90; NSVT (+) by Holter.

4. EP studies: Induction of monomorphic VT.

Hurst's The Heart, 11th edition, C41.
Investigative Tools for sudden cardiac death risk

- ECG - Myocardial infarction
  - Brugada Syndrome
  - Long QT
  - Wolf-Parkinson White Syndrome

- ECHO - LV function
  - Cardiomyopathy (DCM/HCM)
  - Valves (AS)
  - ARVD
Bradycardia

Sinus bradycardia

Sick sinus syndrome

Sinus pauses

Complete HB
<table>
<thead>
<tr>
<th>Clinical Feature</th>
<th>LQT1</th>
<th>LQT2</th>
<th>LQT3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmia onset</td>
<td>Physical exercise</td>
<td>Auditory stimuli</td>
<td>Rest, sleep</td>
</tr>
<tr>
<td>T wave abnormality</td>
<td>Prolonged T wave duration</td>
<td>Small or notched T wave</td>
<td>Delayed onset of T wave</td>
</tr>
</tbody>
</table>

**Table 2. Genotype-Specific Clinical Features**

**Electrocardiograms**

- **LQT1**
  - II: Varying T wave morphology
  - aVF: Varying T wave morphology

- **LQT2**
  - II: Prolonged T wave duration
  - aVF: Varying T wave morphology

- **LQT3**
  - II: Normal T wave morphology
  - aVF: Normal T wave morphology
The WPW pattern

The width of the delta wave depends on the ratio between the AV conduction time over the accessory AV connection (X) and over the AV node and bundle of His (Y).

A. Small delta waves as X is only slightly shorter than Y.
B. Higher and broader delta waves due to a greater difference between X and Y.
C. Because of a very short X the QRS complex becomes one large delta wave resulting in total block of AV conduction through the AV node-bundle of His.

Fig. 6.4
Echocardiography
Other Specialist Tests

- Angiography  - Coronary artery disease
- MRI  - Arrhythmogenic RV dysplasia
  - Hypertrophic Cardiomyopathy
- EP study  - Risk stratification,
  - ICD programming
  - Exclude other arrhythmias
- Genetic testing  - Long QT/HCM/Brugada
Coronary Angiogram
Coronary Angiogram

• In SCD survivors without an ACS, angiography is still considered to exclude stable, chronic CHD

• Diagnostic CAG may not be necessary in selected pts without signs or symptoms of CHD if another clear cause for SCD is identified (eg, long QT syndrome, WPW, Brugada, HCM, LVNC, or ARVD).

• It is unclear whether or not immediate CAG to exclude an ACS should be performed in all SCD survivors without a clear alternative explanation.
MDCT: Short Axis

Patient

Normal
Hereditary SCD Syndrome

**Structurally normal --**
- LQTS
- SQTS (QTc < 300 ms)
- BS: Brugada syndrome
- CPVT: Catecholaminergic polymorphic VT

**Structurally abnormal --**
- HCM: Sarcomere disease
- ARVD: Desmosome disease
- LVNC: LV non-compaction
Brugada Syndrome

Normal ECG

<table>
<thead>
<tr>
<th></th>
<th>Type 1</th>
<th>Type 2</th>
<th>Type 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>J wave amplitude</td>
<td>≥2 mm</td>
<td>≥2 mm</td>
<td>≥2 mm</td>
</tr>
<tr>
<td>T wave</td>
<td>negative</td>
<td>positive or biphasic</td>
<td>positive</td>
</tr>
<tr>
<td>ST-T configuration</td>
<td>covered type</td>
<td>saddleback</td>
<td>saddleback</td>
</tr>
<tr>
<td>ST segment (terminal portion)</td>
<td>gradually descending</td>
<td>elevated ≥1 mm</td>
<td>elevated &lt;1 mm</td>
</tr>
</tbody>
</table>

1 mm = 0.1 mV. The terminal portion of the ST segment refers to the latter half of the ST segment.
EP Study
### Electrophysiological Testing in Cardiomyopathy

<table>
<thead>
<tr>
<th>Cardiomyopathy</th>
<th>Role of EPS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ischemic CM</strong></td>
<td>Confirmed by MADIT II</td>
</tr>
<tr>
<td><strong>Hypertrophic CM</strong></td>
<td>Controversial</td>
</tr>
<tr>
<td><strong>Dilated CM</strong></td>
<td>Minor, low predictive value</td>
</tr>
<tr>
<td><strong>ARVD</strong></td>
<td>Unclear</td>
</tr>
</tbody>
</table>

- The ability to induce ventricular arrhythmias is not predictive of SCD risk in patients with non-ischemic cardiomyopathy.
- EP testing does not have a role in risk stratification in these patients.
## Electrophysiological Testing in Genetic arrhythmia Syndromes

<table>
<thead>
<tr>
<th>Genetic arrhythmia syndrome</th>
<th>Role of EPS</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Long QT syndrome</em></td>
<td><em>not proved useful</em></td>
</tr>
<tr>
<td><em>Brugada syndrome</em></td>
<td><em>debated</em></td>
</tr>
</tbody>
</table>

ACC/AHA/ESC Practice Guidelines September 5, 2006:e247–e346
Antiarrhythmic Drugs for the Prevention of SCD

I. $\beta$ Adrenergic Blocker (JAMA 1982)

II. CAST I (Cardiac Arrhythmia Suppression Trial, NEJM 1989), CAST II (NEJM 1992)

↓ $\times$

“PVC suppression hypothesis”

III. Amiodarone Therapy vs Placebo after MI:

“No survival advantage”

EMIAT (European MI Amiodarone Trial):
LVEF < 0.40 (Lancet 1997)

CAMIAT (Canadian MI Amiodarone Trial):
$\geq$ 10 PVCs / hr or $\geq$ 1 run of VT (Lancet 1997)
ICD

Implantable Cardioverter Defibrillator (ICD)

Heart

Leads

Courtesy of Guidant Corp.
ICD Candidates?

Ischemic or Non-ischemic HD?
Primary or Secondary Prevention?
<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>No.</th>
<th>Population</th>
<th>Mean Follow-up, mo</th>
<th>Main Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVID, 8 1997</td>
<td>Antiarrhythmic medications (97% amiodarone, 3% sotalol) vs ICD</td>
<td>1016</td>
<td>Survived VT/VF/cardiac arrest; VT with syncope; VT with LVEF ≤40%</td>
<td>18</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.66; 95% CI, 0.51-0.85; P&lt;.02)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NNT = 9 at 3 y</td>
</tr>
<tr>
<td>CASH, 9 2000</td>
<td>Antiarrhythmic medications propafenone (withdrawn early), metoprolol, or amiodarone vs ICD</td>
<td>288</td>
<td>Survived VT/VF/cardiac arrest</td>
<td>57</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.82; 95% CI, 0.60-1.11; P = .08)*</td>
</tr>
<tr>
<td>CIDS, 10 2000</td>
<td>Amiodarone vs ICD</td>
<td>659</td>
<td>Survived VT/VF/cardiac arrest; VT with syncope; VT with LVEF ≤35% and cycle length ≤400 ms</td>
<td>35</td>
<td>Reduction in death from any cause with ICD therapy (P = .14)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Reduction in risk of death from arrhythmia with ICD therapy (P = .09) HR, 0.85; 95% CI, 0.67-1.10*</td>
</tr>
<tr>
<td>DEBUT, 11 2003</td>
<td>β-Blocker vs ICD</td>
<td>86</td>
<td>Survived VT/VF/cardiac arrest; no structural abnormalities</td>
<td>36</td>
<td>Total of 7 deaths, all of which occurred in the β-blocker group (3 deaths during 2-y follow-up, P = .07; 4 deaths during 3-y follow-up, P = .02)†</td>
</tr>
<tr>
<td>MAVERIC, 12 2004</td>
<td>Electrophysiologically guided therapy (antiarrhythmic, revascularization, or ICD) vs amiodarone</td>
<td>214</td>
<td>Survived VT/VF/cardiac arrest</td>
<td>60‡</td>
<td>Lower mortality with ICD therapy vs non-ICD therapy (HR, 0.54; 95% CI, 0.39-0.97; P = .04)§</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No advantage to electrophysiologic testing</td>
</tr>
</tbody>
</table>

**Antiarrhythmics vs Implantable defibrillators**

**Cardiac Arrest Study Hamburg**

**Secondary Prevention**

**Canadian Implantable Defibrillator Study**

**Defibrillator vs β Blockers for Unexplained Death in Thailand**

**Empirical Amio vs EP-Guided Intervention and Cardioverter Implant**
Table 2. Primary Prevention of Sudden Cardiac Death in Ischemic Cardiomyopathy

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>No.</th>
<th>Population</th>
<th>Mean Follow-up, mo</th>
<th>Main Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT, 1996</td>
<td>Antiarrhythmic therapy (74% amiodarone) vs ICD</td>
<td>196</td>
<td>Prior MI; LVEF ≤35%; asymptomatic NSVT; NYHA class I-III; inducible VT refractory to intravenous procainamide on electrophysiologic study</td>
<td>27</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.46; 95% CI, 0.26-0.92; P = .009) NNT = 4 at 5 y</td>
</tr>
<tr>
<td>Multicenter Automatic Defibrillator Trial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No reduction in total mortality with ICD therapy (HR, 1.07; 95% CI, 0.81-1.42; P = .64)†</td>
</tr>
<tr>
<td>CABG-Patch, 1997</td>
<td>CABG surgery plus ICD vs CABG surgery plus conventional therapy</td>
<td>900</td>
<td>Patients scheduled for CABG; LVEF ≤35%; positive SAECG result</td>
<td>32</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.69; 95% CI, 0.32-0.63; P &lt; .001) NNT = 3 at 5 y</td>
</tr>
<tr>
<td>MUSTT, 1999</td>
<td>Electrophysiologically guided therapy (antiarrhythmic or ICD) vs conventional therapy</td>
<td>704</td>
<td>Prior MI; LVEF ≤40%; CAD; NSVT; inducible VT on electrophysiologic study</td>
<td>39*</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.69; 95% CI, 0.32-0.63; P &lt; .001) NNT = 3 at 5 y</td>
</tr>
<tr>
<td>Multicenter Unsustained Tachycardia Trial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No reduction in death from any cause with ICD therapy (P = .66)</td>
</tr>
<tr>
<td>MADIT II, 2002</td>
<td>Conventional therapy vs ICD</td>
<td>1232</td>
<td>Prior MI; LVEF ≤30%</td>
<td>20</td>
<td>Reduction in total mortality with ICD therapy (HR, 0.69; 95% CI, 0.51-0.93; P = .02) NNT = 18 over mean 20 mo</td>
</tr>
<tr>
<td>Defibrillator in Acute MI Trial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Risk of arrhythmic death lower with ICD therapy (P = .009) HR, 1.08; 95% CI, 0.76-1.55†</td>
</tr>
<tr>
<td>DINAMIT, 2004</td>
<td>Conventional therapy vs ICD</td>
<td>674</td>
<td>Recent MI (within 4-40 d), LVEF ≤35%; impaired cardiac autonomic modulation (heart rate variability)</td>
<td>39</td>
<td>Overall: reduction in mortality with ICD therapy (P = .007) Ischemic heart disease: reduction in mortality with ICD therapy (P = .05) HR, 0.77; 97.5% CI, 0.62-0.96 NNT = 14 at 5 y</td>
</tr>
<tr>
<td>SCD-HeFT, 2005</td>
<td>Conventional therapy vs amiodarone vs ICD</td>
<td>2521</td>
<td>NYHA class II/III CHF (ischemic and nonischemic); LVEF ≤35%</td>
<td>45.5*</td>
<td></td>
</tr>
</tbody>
</table>
### Cardiomyopathy Trial

**Amio vs ICD in Pts with Nonischemic CM and Asymptomatic Nonsustained VT**

**SCD in HF Trial**

**Prophylactic ICD Implantation in Pts with Nonischemic DCM**

<table>
<thead>
<tr>
<th>Study</th>
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</thead>
<tbody>
<tr>
<td>CAT, 33 2002</td>
<td>Conventional therapy vs ICD</td>
<td>104</td>
<td>NYHA class II/III, NIDCM; LVEF ≤30%; asymptomatic NSVT</td>
<td>66</td>
<td>No reduction in total mortality with ICD therapy (P = .55)*</td>
</tr>
<tr>
<td>AMIOVIRT, 34 2003</td>
<td>Amiodarone vs ICD</td>
<td>103</td>
<td>NYHA class I-III, NIDCM; LVEF ≤35%; asymptomatic NSVT</td>
<td>36</td>
<td>No reduction in total mortality with ICD therapy (P = .80)*</td>
</tr>
<tr>
<td>DEFINITE, 35 2004</td>
<td>Conventional therapy vs ICD</td>
<td>458</td>
<td>NIDCM; LVEF &lt;36%; NSVT or PVCs</td>
<td>29</td>
<td>Reduction in total mortality with ICD therapy (P = .08)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Reduction in death from arrhythmia with ICD therapy (P = .006)</td>
</tr>
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<td>SCD-HeFT, 25 2005</td>
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<td>2521</td>
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<td>45.5‡</td>
<td>Overall: reduction in mortality with ICD therapy (P = .007)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nonischemic heart disease: reduction in mortality with ICD therapy (P = .06)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>HR, 0.77; 97.5% CI, 0.62-0.96</td>
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<td></td>
<td></td>
<td></td>
<td>NNT = 14 at 5 y</td>
</tr>
</tbody>
</table>

**JAMA 2006;295:809-818.**
2006 ACC/AHA/ESC Practical Guidelines
ICD Candidates?

Ischemic or Non-ischemic HD?
Primary or Secondary Prevention?

Yes, ICD therapy does reduce mortality!
ICD Guideline

- Class I
  - VT/VF survivors with irreversible etiology
  - Sustained VT with structural heart disease
  - Syncope + VT/VF at EPS
  - NYHA II-III, LV EF<=35% (Is and non-Is)
  - NYHA I, post-MI, LV EF<=30%, prior MI
  - NSVT, post-MI, LV EF<=40%, VT/VF at EPS
心臟整流去顫器 (ICD) - B103-1

◎ 適應症：
（一）嚴重心室頻脈、心室顫動導致猝死可能或昏迷。
（二）反覆發作之持續性心室頻脈。
（三）高危險性心臟血管疾病，如：曾經心肌梗塞併左心室射出分率 ≤ 40%，肥厚性心肌症，擴張性心肌症，且合併心室快速不整脈者。
（四）高危險性心臟遺傳性疾病，如 long QT syndrome, short QT syndrome, Brugada syndrome, idiopathic ventricular fibrillation, arrhythmogenic right ventricular dysplasia, catecholaminergic polymorphic ventricular tachycardia 等，且合併心室快速不整脈或合併猝死症之家族史者。

◎ 不宜列入項目：
（一）末期心臟衰竭，無法藥物控制又非心臟移植對象者。
（二）猝死可能經急救後，無意識恢復之患者。
（三）末期疾病患者且存活不足六個月者。
（四）惡性且任何治療無法控制 (intractable) 之心室頻脈或心室顫動。

（中華民國 Guidelines）
ICD in Taiwan (1999-2008)

Total ICD: 1,112

Year

Number
17 30 26 29 91 109 118 200 230 262

資料: 吳德朗教授 葉森洲教授 整理提供
Etiology of ICD implantation in Taiwan

- IHD: 47%
- DCM: 30%
- ARVD: 3.0%
- IVF: 7.1%
- LQTS: 4.5%
- Brugada: 4%
- CPVT: 0.5%
- SQT: 0.2%
- Others: 0.1%
- HOCM: 3.6%

資料: 吳德朗教授 葉森洲教授 整體提供
Cardioversion: Success, Failure, or Inappropriate Shock?

#5 VT Episode

**Shock I**   Successful Shock with early re-

Success: 36%

9/25

**Shock III**  Failed

16/25
Adjuvant Anti-arrhythmic therapy

• Reduce the frequency of ventricular arrhythmias in patients with frequent ICD shocks.

• To suppress other arrhythmias that cause symptoms or interfere with ICD function (eg, causing "inappropriate" shocks).

• To reduce the ventricular rate of VT so that it is better tolerated hemodynamically and more amenable to termination by anti-tachycardia pacing or low energy cardioversion

• Amiodarone in combination with a beta blocker reduced the incidence of shocks compared to beta blocker alone

• Sotalol reduced the incidence of ICD shocks compared to placebo
Case 1
General Data of Patient

• Name: 金x強
• Sex: Male
• Age: 63 years old
• Underlining Disease:
  1. History of gastric ulcers.
  No definite risk factor of cardiovascular disease.
General Data of Patient

• Chief Complain:
  Sudden collapse with cyanosis appearance on 8/16 afternoon (PM 04:30)
Consulting Neurologist: Hypoxic-ischemic encephalopathy was favored. Therapeutic hypothermia therapy was indicated.
08/16 evening Admitted to CCU:

Therapeutic hypothermia therapy was processed by protocol.

- Induction phase (Goal to achieve 34°C within 80-120 minutes), achieved goal on 08/17 early morning.
Cause of SCD

- HCM related SCD
- ICD is implanted with adjuvant beta-blocker therapy
- No neurological impairment at CV OPD
Case 2
A 76 Y/O female.

C.C: Loss of consciousness suddenly for 5 min 2 days ago.

Past Hx: 1. HCVD with CHF, treated with ARB, calcium channel blocker and diuretics. 2. Complete AV block, s/p PPM implantation (DDD mode) at 2000. 3. DM with nephropathy. 4. Colon Ca, s/p OP 10 years ago.

P.I: Admitted to local hospital for 2 weeks due to R’t staghorn stone associated with infection. PCNL was done smoothly. Cardiac Echo: LVEF 23%.
Breath sounds: diffuse.

R: III

Cough: soft, dry.

Chest: clear.

[Diagnosis, causes?]

Resp failure
VTE ep. Di shock.
Lx: renal abscess Ep, Tx

Arts: 20

Hx: B//D, U/C, U/C
Blood transfusion 3 E

From: Conscious change due to VT: No pulse

7:40

Frequent VT

D: Sharp: 300 I, K

Xylocain 1M S 800

(Handwritten notes on graph: 'Diffuse paresthesia')
9:35 PM
300 ml
7:30 PM
300 ml

Frequent VT & Conscious Loss
9:32 PM
No pulse. Dr. show 30 J for times, no even xylocaine.
8:10 PM
Cases:
- 30 ml xylocaine
- 6 ml lidocaine 1% + 5 ml saline 500 cc
- 50 cc

8:50 PM
2004, 12, 30, at CCU
K 3.5, Mg 2.3
2000, 4, 24, at Taichung VGH (after PPM implantation, DDD mode)
Atrial Pacing CL: 600 ms
Conclusions:


Amiodarone is not everything!
Take Home Message

• Identify acute reversible causes
• Evaluation for structural heart disease
• In selected patients with a suspected or confirmed heritable syndrome, evaluation of family members
• ICD implantation with adjuvant anti-arrhythmic medication