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Short communication

Sudden cardiac death: The role of imaging

Pieter van der Bijl, Victoria Delgado, Jeroen J Bax *

Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands

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ABSTRACT

Sudden cardiac death (SCD) is defined as "a non-traumatic, unexpected, fatal event occurring within one hour of the onset of symptoms in an apparently healthy subject", and it causes a fifth of all deaths worldwide. It often occurs in individuals not previously known with cardiac disease, which makes prevention challenging. The mechanism underlying SCD is thought to be a trigger (e.g. ischemia) acting upon a substrate (e.g. scar), causing a lethal arrhythmia. Primary prevention refers to patients at high risk of SCD and secondary prevention to those who have had an aborted episode of SCD. Insertion of an implantable, cardioverter-defibrillator (ICD) is the most effective approach to primary prevention; currently ICD candidate selection is based on a left ventricular ejection fraction (LVEF) ≤35%. The LVEF is neither sensitive nor specific in identifying individuals who will benefit from ICD therapy, and therefore alternative strategies are required. The present review article summarizes the evidence on various non-imaging (e.g. microvolt T-wave alternans, signal-averaged ECG, QRS fragmentation and measures of autonomic function) and imaging (echocardiography, cardiac magnetic resonance and radionuclide) modalities showing incremental value over LVEF to identify the patients who will benefit from an ICD.
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1. Introduction

Sudden cardiac death (SCD) is defined as "a non-traumatic, unexpected, fatal event occurring within one hour of the onset of symptoms in an apparently healthy subject" in the guideline on management of patients with ventricular arrhythmias (VA) and the prevention of SCD of the European Society of Cardiology [1]. An unwitnessed death can still be considered a SCD if the individual in question was in good health 24 h before the event [1]. SCD accounts for >4 million global deaths per year, which translates into one fifth of all recorded deaths [1]. About half of SCD's occur in individuals who are not known to have underlying heart disease before the fatal event, thus presenting a significant challenge to effective prevention [1].

2. SCD: causes, mechanisms and prevention

The most common cause of SCD is coronary artery disease, which accounts for up to 50% of SCD in white males. Other causes of SCD are: cardiomyopathies, cardiac hypertrophy, valvulopathies, myocarditis and primary electrical disorders.

The underlying mechanism of SCD is currently understood as a trigger which acts on a substrate, thereby causing a lethal arrhythmia (ventricular tachycardia or ventricular fibrillation) and subsequent hemodynamic instability. A typical example of such an interaction is myocardial ischemia (trigger) interacting with post-infarct myocardial scar (substrate) in the so-called peri-infarct zone. This zone represents a transition between the infarct core and healthy myocardium, and contains scar tissue which is interspersed with normal cardiomyocytes. Slow conduction of electrical impulses occur in the peri-infarct zone, allowing the establishment of re-entry circuits and arrhythmias.

Prevention of SCD is twofold: primary, i.e. in patients deemed to be at high risk of SCD, or secondary, i.e. who have had an episode of SCD aborted spontaneously or by resuscitation efforts. The most effective strategy for both primary and secondary prevention of SCD is the implantable, cardioverter-defibrillator (ICD). Evidence for the use of ICD in primary prevention arose from the Multicenter Automatic Defibrillator Implantation Trial (MADIT II) and the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) [2,3]. The rate of SCD was decreased by >30% in patients with a myocardial infarction and a left ventricular ejection fraction (LVEF) of ≤30% in MADIT II [2]. Similarly, all-cause mortality was reduced by >20% in heart failure patients with LVEF ≤35% in SCD-HeFT [3].

3. Selection of ICD candidates: current practise

The decision to implant an ICD for primary prevention is currently based primarily on the LVEF. An LVEF ≤35% is a class IA indication for ICD according to current European Society of Cardiology guidelines [1]. An exception to this recommendation is the patients with LVEF ≤35% within the first 6 weeks after myocardial infarction. This is based on the results of the Defibrillator in Acute Myocardial Infarction Trial

* Corresponding author at: Department of Cardiology, Leiden University Medical Center, Albinusdreef 2, 2333 ZA Leiden, The Netherlands.
E-mail address: j.j.bax@lumc.nl (J.J. Bax).

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Table 1
Summary of studies reporting the characteristics of the patients with appropriate ICD therapy.

Study	Year published	No. of patients	LVEF (%)[a]	Duration of follow-up	Appropriate therapy (%)
Sabbag et al. [5]	2015	2349	<40% in 66% of patients	2.5 years	2.6 (shock)
Weeke et al. [6]	2013	1609	25 (20−30)	Mean 1.9 ± 1.3 years	13.4 (shock & ATP) 7.8 (shock)
MacFadden et al. [7]	2012	3822	29 ± 11 (women) 31 ± 14 (men)	1 year	15.3 (women) 21 (men) (shock & ATP)
Huikuri et al. [8]	2009	312	30 ± 6	2 years	8 (VT/VF on loop recorder)
Chow et al. [9]	2008	575	24 ± 4.8	Mean 2.1 ± 0.9 years	11.1 (shock & ATP)
Bardy et al. [3]	2005	829	24 (19–30)	Median 3.8 years	21 (shock)
Moss et al. [10]	2004	720	23 ± 5	3 years	35 (shock & ATP)

ATP: antitachycardia pacing; LVEF: left ventricular ejection fraction; VF: ventricular tachycardia, VT: ventricular fibrillation.
[a] LVEF is presented as mean ± standard deviation or median and interquartile range.

(DINAMIT) trial showing that prophylactic ICD therapy within 40 days post-infarction failed to reduce all-cause mortality [4].

However, LVEF ≤35% may not be sensitive enough to select ICD candidates for primary prevention. In the Oregon Sudden Unexpected Death Study (2093 patients with SCD and 448 with echocardiographic data) only 20.5% of patients had LVEF ≤35%. Furthermore, a number of studies have demonstrated that appropriate therapy occurs in less than a third of ICD recipients with LVEF ≤35% (while still exposing them to potential complications of the device) (Table 1) [3,5–10]. Accordingly, relying on LVEF alone for guiding selection of ICD candidates for primary prevention, may not be the ideal strategy.

4. Patient selection based on non-imaging based techniques

Investigation of non-imaging approaches for SCD risk-stratification has been directed mainly at electrophysiological parameters, e.g. microvolt T-wave alternans (MTWA), signal-averaged ECG (SAECG), QRS fragmentation and measures of autonomic function (e.g. heart rate variability (HRV)).

MWTA is the consequence of abnormal handling of intracellular calcium and has been associated with increased risk of SCD in a study of 768 patients with ischemic cardiomyopathy (hazard ratio 2.29; p = 0.049) [11]. Late potentials, recorded in the terminal QRS complex, are the basis for an abnormal SAECG. VA and death occurred in 28% and 17% (p = 0.0001) of patients with an abnormal and normal SAECG, respectively, after 5 years of follow-up in 1925 patients with coronary artery disease [12]. Conversely, no clear link was established with VA or SCD in 313 patients referred for an electrophysiology study [13]. QRS fragmentation can be measured non-invasively with magnetocardiography, which records cardiac electromagnetic activity with detectors placed close to the thoracic wall. QRS fragmentation was significantly increased in patients with VA, compared to those

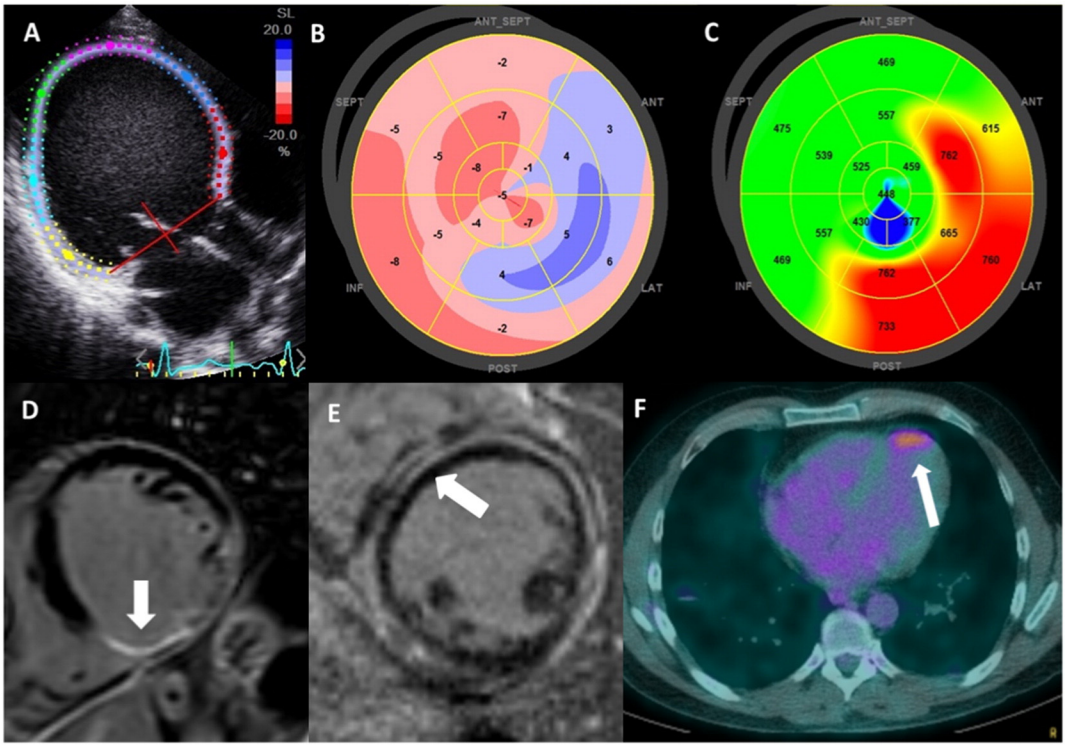

Fig. 1. Imaging modalities to assess the risk of ventricular arrhythmias and sudden cardiac death. With speckle-tracking echocardiography (A), left ventricular (LV) global longitudinal strain (B) and mechanical dispersion (C) can be assessed. Ischemic heart failure patient with dilated left ventricle (A) and significantly impaired LV global longitudinal strain, with LV segments coded in blue (meaning lengthening) and shades of red (meaning better shortening) (B), and large mechanical dispersion with the latest activated segments in the posterolateral regions (C). Thinned, inferoseptal and inferior, basal segments post-infarct, delineated by late gadolinium enhancement (LGE) cardiac magnetic resonance imaging (CMR) (D, arrow). Midwall-fibrosis of the interventricular septum in a patient with dilated cardiomyopathy, indicated by LGE-CMR imaging (E, arrow). Increased apical uptake of ^{18}F–fluorodeoxyglucose on a fused, positron emission tomography and computed tomography scan, indicating the location of cardiac inflammatory activity in a patient with sarcoidosis (F, arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

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