ACUTE CORONARY SYNDROMES

Optimising the dichotomy limit for left ventricular ejection fraction in selecting patients for defibrillator therapy after myocardial infarction

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Background: The selection of patients for prophylactic implantable cardioverter-defibrilator (ICD) treatment after myocardial infarction (MI) remains controversial.
Aim: To determine the optimum left ventricular ejection fraction (LVEF) dichotomy limit for ICD treatment in patients with a history of MI.
Methods and results: Data from the placebo arms of four randomised trials were pooled to create a cohort of 2828 patients (2206 men, mean (SD) age 65 (11) years) with reduced left ventricular function after MI. The median LVEF was 33% (range 6–40%). LVEF significantly predicted mortality. Each 10% reduction in LVEF $<40\%$ conferred a 42% increase in all-cause mortality, a 39% increase in arrhythmic cardiac mortality and a 49% increase in non-arrhythmic cardiac mortality over the 2-year period of follow-up ($p<0.001$ for all modes of mortality). As the LVEF progressively decreased from $\leqslant 40\%$ to $\leqslant 10\%$, the data show a U-shaped relationship between the dichotomy limit for LVEF used and the number of patients who must be treated to prevent one arrhythmic death in 2 years. At an LVEF of 16–20%, more patients are likely to die from arrhythmic than non-arrhythmic cardiac deaths, whereas in those with LVEF $\leqslant 10\%$ all deaths were non-arrhythmic. However, the total number of deaths substantially decreased with lower LVEF.
Conclusion: A trade-off exists between the sensitivity and positive predictive accuracy across a range of LVEF, and no single dichotomy limit is completely satisfactory. In patients with LVEF $\leqslant 10\%$ ICD treatment was not beneficial as all patients in this subgroup died from non-arrhythmic causes. The use of a single dichotomy limit for LVEF alone is not sufficient in selecting patients for ICD treatment in the primary prevention of cardiac arrest.

The Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) showed that the prophylactic implantation of a defibrillator in patients with a prior myocardial infarction (MI) and a left ventricular ejection fraction (LVEF) of $\leqslant 30\%$ reduced mortality from 19.8% to 14.2% at 2 years,[1] an effect explicable entirely by a 67% reduction of sudden cardiac death.[2] In this patient group, 18 high-risk patients needed to be treated for 2 years to save one life. Despite the approval by the US Food and Drug Administration for the use of implantable cardioverter defibrillator (ICD) in patients meeting the MADIT II entry criteria, the cost effectiveness of the use of ICD treatment in this group is still debated widely.

In patients with a history of MI, there is a progressive increase in 1 year mortality as the LVEF falls $<40\%$.[3] Subgroup analysis of previous trials suggests that treatment with amiodarone provides most benefit in patients with moderate (LVEF 31–40%) rather than severe left ventricular impairment ($\leqslant 30\%$) who are more likely to die from pump failure.[4] The aim of this study was to assess the relationship between LVEF and the risk of arrhythmic death and thus to determine the optimum dichotomy limit for LVEF in selecting patients who had MI whose risk of arrhythmic death is sufficient to justify prophylactic ICD treatment.

METHODS

We pooled the individual placebo patient data from European Myocardial Infarct Amiodarone Trial (EMIAT), Survival With ORal d-sotalol, TRAndolapril Cardiac Evaluation (TRACE) and Danish Investigation of Arrhythmias and Mortality on Dofetilide-Myocardial Infarction (DIAMOND-MI) studies, which recruited patients with LVEF $\leqslant 40\%$ after MI. Details of the design of these studies have been published elsewhere.[4–7]

All four trials pooled for this study were prospective multicentre randomised placebo-controlled trials conducted in the thrombolytic era, which recruited patients with recently documented acute myocardial infarction (AMI), LVEF $\leqslant 40\%$ and clear clinical end points determined by an event committee including all-cause mortality, arrhythmic cardiac mortality and non-arrhythmic cardiac mortality. Similar rules were employed by each of the committees. Arrhythmic death was defined according to the Hinkle and Thaler classification as ''the abrupt spontaneous cessation of respiration and pulse and loss of consciousness in the absence of other progressive severe medical conditions likely to cause death''. Documented arrhythmic death or cardiac arrest was considered to be present if ventricular tachycardia or fibrillation was recorded within 10 min of clinical death. Other cases were classified as ''presumed arrhythmic''.

In the selected studies, patients were followed up for at least 24 months. We analysed survival at 2 years in all trials. All the trials selected were antiarrhythmic drug trials involving class III antiarrhythmic agents except TRACE, which investigated the

Abbreviations: AMI, acute myocardial infarction; DIAMOND-MI, Danish Investigation of Arrhythmias and Mortality On Dofetilide-Myocardial Infarction; EMIAT, European Myocardial Infarct Amiodarone Trial; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; MADIT II, Multicenter Automatic Defibrillator Implantation Trial II; MI, myocardial infarction; TRACE, TRAndolapril Cardiac Evaluation

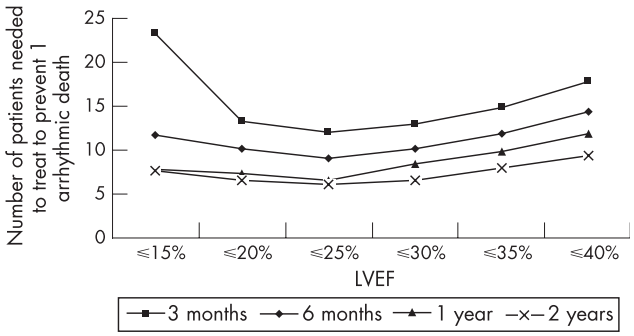

Figure 1 The use of various left ventricular ejection fraction LVEF dichotomy limits and different periods of follow-up. The number of patients needed to treat to prevent one arrhythmic death followed a U-shaped curve that is more accentuated at shorter periods of follow-up.

Table 1 Baseline characteristics of patients

Age (years)	65 (11)
Sex (M/F) (%)	2206/622 (78/22)
Previous MI (Y/N) (%)	885/1933 (31/69)
Antecedent hypertension (Y/N) (%)	760/2047 (27/73)
NYHA (0–1/II–IV) (%)	906/1866 (33/67)
Q wave (Y/N) (%)	2080/709 (75/25)
Heart rate	78 (14)
Systolic blood pressure	119 (18)
Smoking (Y/N) (%)	1826/538 (77/23)
Atrial fibrillation (Y/N) (%)	352/2140 (14/86)
Diabetes (Y/N) (%)	342/2020 (14/86)
Digoxin (Y/N)	490/2331 (17/83)
Thrombolytic (Y/N)	1428/1379 (51/49)
Calcium antagonists (Y/N)	576/2246 (20/80)
ACE inhibitors (Y/N)	845/1976 (30/70)
β Blockers (Y/N)	734/2088 (26/74)

F, female; M, male; MI, myocardial infarction; N, no; NYHA, New York Heart Association; Y, yes.

use of the ACE inhibitor trandolapril. As class III antiarrhythmic drugs and trandolapril have been shown to influence mortality from arrhythmia, we used only data pertaining to patients in the placebo arm of each trial.

Short-term and long-term survival were analysed separately to allow for differences between the studies in the interval between the index MI and trial entry. Short-term survival was analysed using logistic regression at 45 days from the time of the index infarct using only data from the TRACE and DIAMOND-MI studies that recruited patients within 2 weeks of infarct. Long-term survival in patients surviving at least 45 days after the index MI was analysed from the combined data of all four trials. The effect of LVEF on long-term survival was investigated using Cox's regression, adjusted for treatment and study effects and for demographic factors that had been preassessed and shown to be associated with survival. A two-sided significance test was used, with $p<0.05$ considered significant. We investigated whether hazards remained proportional over time by comparing the results before and after the first year. Non-linear effects of continuous variables were assessed by adding polynomial terms.

To assess the optimum dichotomy limit for LVEF, we examined the number of patients needed to treat to prevent one arrhythmic or sudden cardiac death. The number needed to treat is defined as the number of patients who would have to receive the treatment for one of them to benefit, calculated as one divided by the absolute risk reduction. As we are only dealing with the placebo patients and had no treatment group to compare, we calculated the number needed to treat on the basis of the finding from MADIT II that ICD implantation in this patient group reduces sudden cardiac death by 67% without altering other forms of mortality.[1] Patients who were censored before 45 days were excluded from this analysis. Patients who died from other causes (non-arrhythmic cardiac death and non-cardiac death) were included in the total population.

RESULTS

Data pertaining to 2828 placebo patients were pooled (2206 men, aged: 65 (11) years) from all four trials. Table 1 summarises the baseline characteristics of the patients. The median LVEF was 33% (range 6–40%). There were 652 deaths (all-cause mortality) at 2 years, of which 303 were arrhythmic cardiac deaths and 242 were non-arrhythmic cardiac deaths. After adjusting for factors that were associated with survival (ie, age, sex, previous MI, hypertension history, New York Heart Association class, presence of abnormal Q wave in the surface ECG, heart rate, smoking and systolic blood pressure), LVEF significantly independently predicted long-term (45 day–2 year) all-cause mortality, arrhythmic cardiac mortality and non-arrhythmic cardiac mortality ($p<0.001$ for all three modes of mortality; table 2).

Each 10% reduction in LVEF <40% conferred a 42% increase in all-cause mortality, a 39% increase in arrhythmic cardiac mortality and a 49% increase in non-arrhythmic cardiac mortality over the 2-year period. The total number of patients and, consequently, the number of deaths with LVEF <20% was much lower than the number with LVEF 31–40%. Patients with LVEF <20% were slightly more likely to die from non-arrhythmic than from arrhythmic causes, whereas the reverse was true if the LVEF was 31–40% (table 3). LVEF was not predictive of short-term mortality up to 45 days after AMI (all-cause mortality, $p = 0.1$; arrhythmic mortality, $p = 0.1$; cardiac mortality, $p = 0.96$).

As the LVEF dichotomy limit was reduced from ⩽40% to ⩽15%, the number of patients needed to treat to prevent one arrhythmic death in 2 years declined from 9.3 (for LVEF ⩽40%) to 6.1 (for LVEF ⩽25%) but then increased to 7.9 (for LVEF ⩽15%). This trend became more pronounced, showing a progressively deeper U shape as the period of follow-up was reduced to 3 months (fig 1 and table 4).

When we examined the number of patients needed to treat to prevent one arrhythmic death in 2 years at different ranges of LVEF (ie, 5%, 10%, 15%, 20%, 25% and 30%) and at various dichotomy limits (⩽10, ⩽15, ⩽20, ⩽25, ⩽30 ⩽35, 0–10, 0–15, 11–15, 0–20, 11–20 and so on), it was lowest at 4.3 when the LVEF was 16–20% (fig 2). Among patients with LVEF ⩽10%, all deaths occurred exclusively from non-arrhythmic cardiac causes.

DISCUSSION

The bulk of available evidence regarding indications for ICD treatment as primary prevention is derived from three trials

Table 2 Effect of incremental 10% increase in left ventricular ejection fraction on survival

	Mortality <45 days OR (95% CI), p value	Mortality 45 days–2 year HR (95% CI), p value
All-cause mortality	0.77 (0.56 to 1.06), 0.1	0.58 (0.49 to 0.68), <0.001
Arrhythmic mortality	0.72 (0.47 to 1.09), 0.1	0.61 (0.48 to 0.78), <0.001
Non-arrhythmic cardiac mortality	1.01 (0.61 to 1.68), 0.96	0.51 (0.39 to 0.66), <0.001

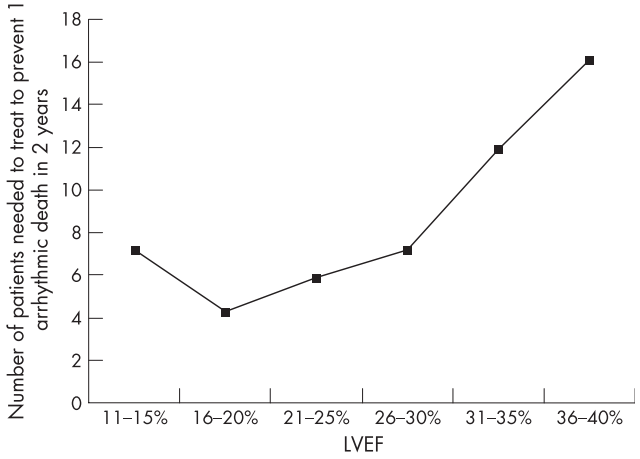

Figure 2 Left ventricular ejection fraction (LVEF) is represented in 5% intervals rather than dichotomy limits. The number of patients needed to treat was lowest at 4.3 when the LVEF was 16–20%.

that used a dichotomy limit for LVEF only as a selection criterion. On the basis of these trials, ICD implantation is commonly undertaken on the sole basis of LVEF <30% in patients after MI. Although there is evidence that low-risk and high-risk subgroups exist within this group, there is no agreed way to stratify for risk. Doing so could allow substantial cost saving at negligible sacrifice in population life expectancy.[8] There is also uncertainty about the need for ICD treatment in those with an LVEF of 30–40%. Patients in this group do not generally receive ICD treatment, although, as we have shown, their risk of sudden death is substantial.

This study showed that as the LVEF reduced from 31–40% to <20%, the incidence of all-cause and arrhythmic cardiac mortality increased threefold and that of non-arrhythmic cardiac mortality increased fivefold such that the risk of non-arrhythmic cardiac death superseded that of arrhythmic death. In line with this, the number of patients who must be treated to prevent one arrhythmic death in 2 years decreased as the LVEF dichotomy limit decreased until a ''trough'' was reached when a dichotomy limit of ⩽25% was used.

The number of patients who must be treated to prevent one arrhythmic death reduced to its lowest value of 4.3 in patients with LVEF in the range 16–20%. As the LVEF decreased further, the number of patients needed to treat began to rise. At LVEF ⩽10%, all deaths were from non-arrhythmic cardiac causes (table 3). This suggests that the use of a single cut-off value of LVEF is inappropriate, and that the specific risk of arrhythmic and non-arrhythmic mortality should be estimated for each individual. Refinement of risk measurement is necessary. This could involve the use of improved strategies for combining the available indices or the development of indices more precise than those considered in this study.

Combining LVEF and other criteria

Of the ICD primary prevention trials in patients with ischaemic heart disease, only the Multicenter Unsustained Tachycardia Trial (LVEF ⩽35%, non-sustained ventricular tachycardia and inducible and non-suppressible ventricular tachycardia at electrophysiological study) and coronary artery bypass graft-patch (LVEF <30%, positive signal-averaged ECG and need for coronary bypass surgery) involved the use of LVEF in combination with other risk factors as entry criteria. In the Multicenter Unsustained Tachycardia Trial, fewer patients needed to be treated with ICDs to save one life than in MADIT II,[9] but the registry patients from this study who were non-inducible at electrophysiological testing and had an LVEF <30% had a total mortality and arrhythmic mortality nearly identical to that of patients who were inducible but had an LVEF of 30–40%,[8] and data from MADIT II suggest that electrophysiological testing adds little prognostic information.[10] Thus, patients who had MI with an LVEF <30% remain at risk of sudden death even if electrophysiological testing proves negative. Other methods of risk stratification such as microvolt-level T-wave alternans seem more promising.[11]

Time dependence of arrhythmic risk

The number of patients needed to treat to prevent one arrhythmic death reduced significantly as the follow-up period increased from 3 months to 2 years. Thus, 3 months of follow-up is inadequate to provide a true picture of mortality after MI, particularly for arrhythmic mortality. This is consistent with the MADIT II study which showed that although approximately 88% of the patients were recruited >6 months from the last MI, prophylactic ICD treatment confers a survival benefit.[1]

We did not find any effect of LVEF on short-term mortality up to 45 days after AMI. This observation is intriguing in view of the recently published Defibrillator in Acute Myocardial Infarction Trial that showed that patients who had MI with a reduced LVEF ⩽35% and impaired cardiac autonomic modulation did not benefit from ICD treatment if implanted early after MI (within 6–40 days after MI).[12] The mechanism for this is unclear but it seems that remodelling of the left ventricle that occurs in the early weeks after MI makes risk assessment based on a single echocardiographic study less reliable than in the chronic phase.[13] It may also be that non-arrhythmic causes of death are so frequent in the early months after acute MI that the benefit of ICD treatment is masked, or that the proximity to revascularisation procedures reduces the benefit of ICD treatment.[14]

Cost effectiveness

The evidence from this study highlights the heterogeneity of the risk of sudden death in those with LVEF ⩽40%. Patients with an LVEF of 16–20% seem likely to benefit most from ICD treatment, although these account for only a small minority of the sudden deaths in the entire cohort (33 of 294 sudden deaths; table 4). Our study showed that patients with

Table 3 Number of patients for different intervals of left ventricular ejection fraction and rates of different forms of mortality

	Mortality rate per person-year between 45 days and 2 years (%)		
LVEF (%, n)	All-cause mortality (%)	Arrhythmic mortality (%)	Non-arrhythmic cardiac mortality (%)
31–40 (1432)	6.8	3.2	2.2
21–30 (881)	17.5	7.7	6.3
<20 (193)	23.1	9.4	10.6

LVEF, left ventricular ejection fraction.

Table 4 Number of patients for three different modes of death at various left ventricular ejection fraction dichotomy limits at a follow-up period of 2 years

Dichotomy limits for LVEF (%)	Number of patients	All-cause mortality	Arrhythmic mortality	Non-arrhythmic cardiac mortality
⩽40	2828	652	303	242
⩽35	1973	536	250	195
⩽30	1250	415	189	156
⩽25	570	207	94	79
⩽20	213	74	33	33
⩽15	70	25	9	12

LVEF, left ventricular ejection fraction.

LVEF ⩽10% will not benefit from ICD treatment since all patients in this subgroup died from non-arrhythmic causes (fig 2).

The cost effectiveness of ICD use in addition to optimal medical treatment is dependent on absolute risk of arrhythmic death and on the ratio of sudden to non-sudden cardiac death.[15] In patients with high rates of sudden cardiac death but low rates of non-sudden death, the ICD provides a large benefit at a reasonable cost. An increase in the rate of deaths not preventable by the ICD within the lifetime of the device would erode greatly its cost-effectiveness.[15] Further studies are needed to determine the cost-effectiveness of ICD use among patients who had MI with low LVEF.

LIMITATIONS

The modes of death were determined by event committees, and the exact mode of death cannot be known for certain in all cases. This method of classifying the mode of death has proved to be consistent and sufficiently accurate in the ICD trials. We are dependent on a single measurement of LVEF in most patients, and measurement is operator dependent and often variable over time, particularly in the period soon after MI.

The proportion of patients receiving thrombolytic treatment was low in our study compared with currently expected standards and the data pertain to a period before the widespread use of percutaneous revascularisation, glycoprotein IIb/IIIa receptor inhibitors or clopidogrel in acute MI. The use of ACE inhibitors and β-blockers in the study population was also low compared with the use observed in more recent trials including MADIT II. The widespread use of ACE inhibitors and β-blockers could reduce the rate of both arrhythmic and non-arrhythmic death and alter the balance between arrhythmic and non-arrhythmic deaths in a manner that is difficult to predict. The use of statins could also influence both arrhythmic and non-arrhythmic mortality,[16] but their use was not recorded in all the trials analysed. The use of all appropriate pharmacological treatments and cardiac resynchronsation could reduce the rate of non-arrhythmic mortality in those with low LVEF sufficiently to allow them to benefit from the reduction in the risk of arrhythmic mortality provided by ICD treatment. The population of patients in MADIT II was more stable than that in our study in terms of the risk of sudden cardiac death, as almost 90% patients enrolled in MADIT II experienced MI >6 months earlier compared with the cut-off of 45 days in our combined cohort of patients, although approximately 70% patients in Survival With Oral D-Sotalol were enrolled at an average of 76 weeks after MI.

CONCLUSIONS

The use of a single dichotomy limit of LVEF to determine the need for ICD treatment leads to a trade-off between the sensitivity and positive predictive accuracy across a range of LVEF. No single dichotomy limit is completely satisfactory as all patients with an LVEF ⩽10% in our study died from non-arrhythmic causes. The cost effectiveness of ICD treatment is maximal in those with an LVEF of 16–20%.

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IMAGES IN CARDIOLOGY

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A rare single coronary artery with an anomalous origin of the left coronary artery from the posterior atrioventricular right coronary artery

A 51-year-old man was referred to coronary angiography because of a non ST-elevation acute coronary syndrome. The coronary angiography showed a rare single coronary artery arising from the right anterior sinus of Valsalva. This artery was responsible for the entire vascularisation of the heart. The left coronary artery arises from the posterior atrioventricular right coronary artery (RCA). Stenoses of the large posterior atrioventricular coronary artery of the RCA and the first segment of the left anterior descending artery were detected and determined the clinical status (panels A, B and C below; also, supplementary movies I and II are available online at http://heart.bmj.com/supplemental). Drug-eluting stent implantations were performed both with good clinical and angiographic results (panels D, E and F below; supplementary movie III available online).

Subsequently, an enhanced 16-multislice spiral computed tomography (MSCT) confirmed the absence of a coronary artery from the left anterior sinus (panel G), detailed the anatomic findings (panels H, I and J) and confirmed the success of stenting both lesions (panels K and L) (panels G–L are in the supplementary figure available online at http://heart.bmj.com/supplemental).

To the best of our knowledge, this is the first description of a whole left coronary artery arising from the posterior atrioventricular coronary artery of the RCA. We also assessed the interest of MSCT in such a situation, because conventional exercise tests often produce conflicting results in patients with coronary anomalies alone or in combination with an obstructive coronary disease. Comprehensive MSCT examinations may thus be useful in understanding the anatomy of abnormal coronary artery and in controlling the result of uncommon angioplasties as a non-invasive reference test for the follow-up to detect in-stent restenosis.

Supplementary figure and movies are available online at http://heart.bmj.com/supplemental

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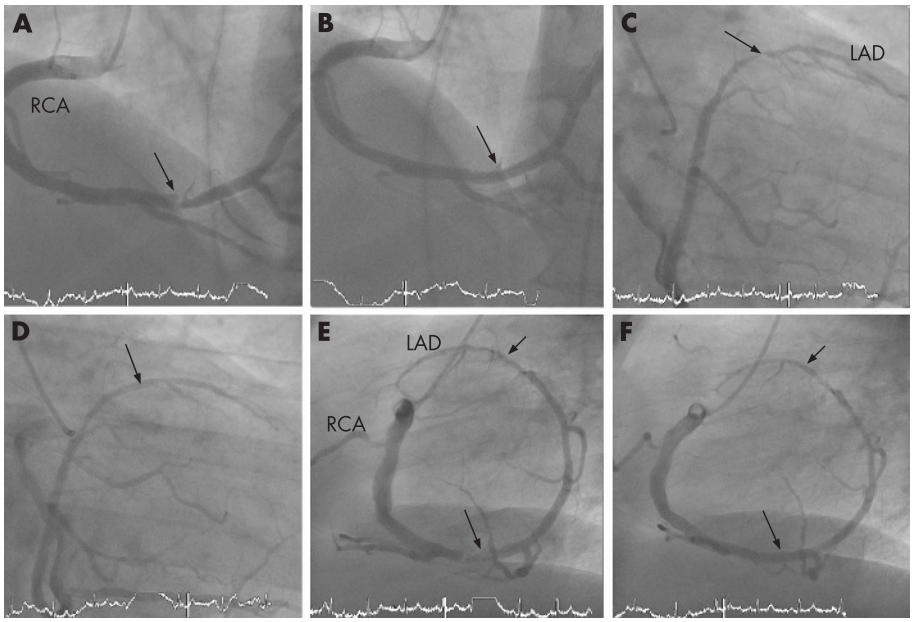

Coronary angiography and multislice spiral computed tomography. The coronary angiography showed (A) a stenosis of the posterior atrioventricular artery (arrow), (B) a stenosis of the left anterior descending artery (arrow) and (C) an overview of both stenoses (arrows). The coronary angiography showed the result after stent implantation on (D) the posterior atrioventricular artery (arrow), (E) the left anterior descending artery (arrow) and (F) the final result after the implantation of both stents (arrows). RCA, right coronary artery; LAD, left anterior descending artery. (Further details and the supplementary figure are available at http://heart.bmj.com/supplemental)