Guidelines ICD implantation 2005 – an update

L. van Erven[1], P.F.H.M. van Dessel[2], T.A. Simmers[3], I.C. van Gelder[2], R.N.W. Hauer[4], E.F.D. Wever[5], B.Dijkman[6], L. Jordaens[7], A. Ramdat Misier[8], A. Meijer[9], H.A. Bosker[10], M.J.Schalij[1]

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[1] Department of Cardiology, Leids University Medical Center, Leiden
[2] Department of Cardiology, University Medical Center Groningen, Groningen
[3] Department of Cardiology, Academic Medical Center, Amsterdam
[4] Department of Cardiology, University Medical Center Utrecht, Utrecht
[5] Department of Cardiology, St Antonius Ziekenhuis, Nieuwegein
[6] Department of Cardiology, Academisch Ziekenhuis Maastricht, Maastricht
[7] Department of Cardiology, Erasmus University Medical Center, Rotterdam
[8] Department of Cardiology, Isala Klinieken, Zwolle
[9] Department of Cardiology, Catharina Ziekenhuis, Eindhoven
[10] Department of Cardiology, Rijnstate Ziekenhuis, Arnhem

Introduction

The first guidelines on implantable cardioverter/defibrillator (ICD) implantation were published in 1998 and 2001, by the ACC/AHA/NASPE and the ESC, respectively (1;2). With the completion of several important randomised clinical studies, reconsideration of the guidelines has been thought to be appropriate. Updates of both the American and the European guidelines on ICD implantation have been published in 2002 and 2003, respectively (3;4), based on these studies. Basically, the indications are expanding from secondary to primary prevention, depending on the underlying heart disease.

In this paper, we present an update on the Dutch guidelines as published in 2001 (5). The evidence has led to updates on two fronts: 1. primary prevention of sudden cardiac death (SCD) in patients with depressed left ventricular function after previous myocardial infarction, and 2. primary prevention of SCD in patients with dilated cardiomyopathy (DCM). The current paper is a composition of the Dutch guidelines as published in 2001 (5), the update published by the European Society (4;6) and recently published ICD studies. These guidelines also represent the consensus reached after discussion in the Netherlands Heart Rhythm Association.

Primary versus secondary prevention

Primary prevention is therapy that is given in order to prevent sudden death in patients who have not yet suffered a life-threatening sustained ventricular arrhythmia, but who are at high risk of such an arrhythmia. Secondary prevention is therapy for patients who have already suffered a cardiac arrest or syncopal/hypotensive ventricular tachycardia.

Level of evidence and recommendation

Disease-specific risk factors for SCD as well as indications for ICD implantation are classified according to the generally used ranking system (table 1). The class refers to the level of agreement that a risk factor is predictive of SCD or ICD implantation is useful and effective in this patient category. Level of evidence conveys the weight of evidence leading to this (dis-)agreement. Together, class and level of evidence result in strength of recommendation.

Pathophysiology of Sudden Cardiac Death

Sudden cardiac death is defined as “natural death due to cardiac causes, heralded by abrupt loss of consciousness within one hour of the onset of acute symptoms, pre-existing heart disease may have been known to be present, but the time and mode of death are unexpected” (1). Coronary artery disease is the most common underlying disease in SCD (75%) (7). In about 65% of cases, SCD is caused by monomorphic VT degenerating into VF or polymorphic VT. However, SCD also occurs in the setting of nonischemic dilated cardiomyopathy, hypertrophic cardiomyopathy, “channelopathies”, congenital heart disease and others.

Reduced left ventricular ejection fraction (≤30%) and occurrence of VTs have predictive value for SCD in ischemic cardiomyopathy (8-10). However, VTs in other conditions do not always predict SCD. Evaluation and treatment of ischemia or other treatable causes should be performed in this patient group prior to considering ICD implantation (11).

Therefore, a thorough pre-evaluation must be performed, to establish the existence or non-existence of coronary artery disease and other risk markers, to optimize left ventricular function, and to exclude reversible causes. A suggestion for the workup is presented in figure 1.

Ischemic cardiomyopathy

ICDs were originally developed to prevent SCD in patients who had experienced life-threatening ventricular arrhythmias such as VT or VF (12). Patients who have experienced out-of-hospital cardiac arrest have a low probability of survival (13). It was not, however, until landmark randomised controlled trials were reported that the device became widely accepted (14). These studies demonstrated that the ICD produced approximately 30% reduction in relative risk of SCD, in 3 years follow-up, in patients with prior sustained malignant ventricular arrhythmias. With the publications of these secondary prevention trials, ICDs became the therapy of choice for patients with prior cardiac arrest or hemodynamically poorly tolerated VT.

Secondary prevention in post MI patients is based on several controlled randomized trials comparing antiarrhythmic drugs with ICD therapy (1;14;15). Wever et al. were the first to show the benefit of ICD therapy in out-of-hospital cardiac arrest survivors with prior

myocardial infarction (14). In the AVID, CASH and CIDS trials, not all patients had suffered prior myocardial infarction and only about 80% of patients in these studies had coronary artery disease. Average ejection fraction in these studies was 34%, but differed considerably between studies. Only the AVID trial, the largest of the three, showed a significant reduction in mortality with ICD compared to drug therapy (mostly amiodarone). The same results were shown in a meta-analysis of the three studies, based on the individual patient data. The relative risk reduction was 31% in 3 years follow-up and 27% in 6 years follow-up, respectively. ICD implantation in post MI patients with VF or hemodynamically poorly tolerated VTs is accepted as a class I indication for ICD implantation with level of evidence A, referring to the AVID trial and Connolly's meta-analysis of the AVID, CIDS and CASH trials.

The extremely low survival rate after out-of-hospital cardiac arrest was the motivating factor to conduct primary prevention trials (16). Primary prevention was initially based on the MADIT and MUSTT trials. These studies showed that patients with coronary artery disease, a reduced left ventricular ejection fraction (35% or less and 40% or less, respectively) and inducible sustained VT benefited from prophylactic implantation of a defibrillator (8;17). In 2002 the MADIT II trial was published (18). This was the rationale for revision of the guidelines on ICD indication in post-myocardial infarction patients. In the MADIT II study, the effectiveness of ICD implantation was evaluated in patients with low ejection fraction ($\leq$ 30%) more than one month after myocardial infarction that never had shown any ventricular arrhythmia. A total number of 1232 patients were included and ICD implantation was compared to conventional medical therapy in a 3:2 fashion. The study was terminated preliminary, after a follow-up of 20 months (range 6-53 months), because of a significant reduction of 31% in all-cause mortality in the ICD patient group. Thus, MADIT II provides evidence that patients with poor left ventricular function at least one month post-myocardial infarction have a better survival receiving a prophylactic ICD. Primary prevention in post-myocardial infarction patients with depressed left ventricular (LV) function has been accepted as a class IIa and not a class I indication for ICD implantation (4) due to limitations in the study design: firstly, as 24-hour ECG recording was not performed in MADIT II, the contribution of patients meeting the MADIT criteria is unclear (nonsustained VT and inducible / nonsuppressible VT during EP study). Secondly, the early termination might have led to overestimation of the long-term beneficial effects of ICD treatment. Thirdly, it should be noted that coronary angiography or myocardial scintigraphy in order to exclude current (silent) ischemia as treatable cause were not a prerequisite for inclusion in MADIT II. So, it

cannot be excluded that at least some of the patients, with for example multivessel coronary artery disease, who were included in the trial may have been candidates for a revascularization procedure alone. Fourthly, retrospective analysis performed by the MADIT II investigators showed a lesser benefit from ICD therapy early (< 18 months) after myocardial infarction (19), indicating that the issue of timing of ICD implantation after myocardial infarction is still not fully resolved. Finally, heart failure related hospitalization was more common in the ICD-treated group for which to date no explanation has been published. The clinical significance of these issues remains to be resolved. Based on these considerations, ICD therapy in MADIT II-like patients has been recommended as class IIa with level of evidence B in the 2003 ESC guidelines (4).

Recently, the SCD-Heft confirmed the efficacy of primary prevention by ICD therapy in ischemic cardiomyopathy (20). In this study, 2521 patients with NYHA class II or III CHF and LVEF of 35% or less were randomized to conventional therapy for CHF plus placebo (847 patients), conventional therapy plus amiodarone (845 patients), or conventional therapy plus a conservatively programmed, shock only, single-lead ICD (829 patients). Placebo and amiodarone were administered in a double-blind fashion. The primary end point was death from any cause. The median LVEF in patients was 25%; 70% were in NYHA class II, and 30% were in class III CHF. The cause of CHF was ischemic in 52% and nonischemic in 48%. The median follow-up was 45.5 months. There were 244 deaths (29%) in the placebo group, 240 (28%) in the amiodarone group, and 182 (22%) in the ICD group. As compared with placebo, amiodarone was associated with a similar risk of death and ICD therapy was associated with a significantly relative risk reduction of 23% and an absolute decrease in mortality of 7.2 percentage points after five years in the overall population. Results did not vary according to either ischemic or nonischemic causes of CHF.

Since two large randomized controlled trials (SCD-Heft and MADIT II) proved ICD therapy to be efficacious in primary prevention for SCD in ischemic heart disease, ICD therapy should be considered at least a class IIa indication with level of evidence A, pending further discussion of the SCD-Heft results.

Although the COMPANION study also addresses the issue of primary prevention in patients with reduced LVEF of whom approximately 50% had ischemic cardiomyopathy, inclusion criteria for this study differed significantly from the MADIT II and SCD-Heft (21). The proportion of patients suffering from advanced heart failure (NYHA ≥ III) mounted up to 85% in COMPANION vs. 25-30% in MADIT II or SCD-Heft.

As of yet, there is no evidence for ICD implantation early after myocardial infarction. Analysis of the MADIT II data showed a lesser benefit of ICD therapy early (< 18 months) after myocardial infarction (hazard ratio 0.98, p=0.95). The DINAMIT study was designed to evaluate the use of ICD therapy within 40 days of myocardial infarction, compared to optimal medical therapy alone, in patients with ejection fraction ≤ 35%, depressed heart rate variability or elevated heart rate (mean 24 h >79/min) (22). Patients were enrolled early (6 to 40 days) after acute myocardial infarction. During mean follow-up of 30 ± 13 months, there was no difference in all-cause mortality between the conventionally treated patients and the group randomised to ICD implantation. Although ICD therapy was associated with a reduction in the rate of death due to arrhythmia, this was offset by an increase in the rate of death from nonarrhythmic causes. Therefore, the primary endpoint of all-cause mortality showed no difference between the groups (23;24). The reason for excess nonarrhythmic death remains unclear. This again brings up the issue that implantation of ICDs for primary prevention not only reduces arrhythmogenic risk but also may introduce ICD related co-morbidity such as possible pacing-aggravated heart failure or implantation related complications (18;25;26).

In post-MI patients with hemodynamically tolerated VTs and relatively preserved LV function (EF > 30%), treatment with drugs, ablation, surgery either or not in combination with ICD may be considered (1;4). None of these therapeutic options has yet been proven to be superior to the others.

Idiopathic dilated cardiomyopathy

No specific trials have addressed the issue of secondary prevention in DCM patients. However, ICD implantation is considered a class I indication with level of evidence C.

With respect to primary prevention in dilated cardiomyopathy (DCM), the results of the Cardiomyopathy Trial (CAT) (27) prompted revision of the ICD guidelines in patients with DCM (4). The CAT trial was a relatively small trial of only 104 symptomatic DCM patients with depressed left ventricular function (EF ≤ 30%) of recent onset (≤ 9 months) without or with only nonsustained ventricular arrhythmias (27). The trial was terminated due to futility because the end-point all-cause mortality was much lower than expected (5.6% vs. 30%, respectively).

The AMIOVIRT study (28), published in 2003, compared amiodarone and ICD in 103 patients with nonischemic cardiomyopathy but also failed to show effect.

More recently, other trials evaluating the same patient group were published. In the DEFINITE trial (26), 458 patients with nonischemic cardiomyopathy were followed, with an average EF of 20% and with PVCs or nonsustained VTs on Holter recordings. In these patients with mild heart failure (79% NYHA I and II), drug treatment was optimized and an ICD was implanted in half of the patients. After a follow-up of 29±14 months, no difference in total mortality was observed, since 28 of 229 (12%) patients died in the ICD group, compared to 40 of 229 (17%) patients in the optimal medical treatment group (P=0.08). The relative risk for arrhythmic death was significantly higher (13 of 40 deaths (33%)) in the medically treated group as compared to the ICD group (3 of 28 deaths (11%)) (Hazard ratio 0.2, confidence interval 0.06-0.71, p=0.006)

The COMPANION trial, published in may 2004, addressed 1520 heart failure patients who were eligible for biventricular pacing (NYHA≥III, QRS>120 ms, PR>150 ms, LVEDD>60 mm, EF ≤ 35%) (21). Patients were randomized to optimal medical therapy (OPT), OPT combined with biventricular pacing (CRT) or OPT, biventricular pacing and ICD therapy (CRT-D). It was primarily a heart failure trial, the primary end point being a composite of death from any cause or hospitalization for any cause. There were 26% withdrawals in the OPT group, as compared with 6 % of those in the CRT group and 7 % of those in the CRT-D group. In the nonischemic patients, a 50% reduction in death from any cause was observed. This study was the first to demonstrate mortality reduction in nonischemic cardiomyopathy, be it in the setting of concomitant severe heart failure.

The SCD-Heft study confirmed the findings of the COMPANION study that mortality reduction was achieved regardless of etiology of LV dysfunction (20). In addition, since 70% of SCD-Heft patients was in NYHA class II, the SCD-Heft showed efficacy of device therapy in the absence of sever heart failure. Results did vary according to the NYHA class: mortality reduction in NYHA II patients was greater than in NYHA III patients. This appears at odds with COMPANION, where the poorer NYHA class patients benefited most.

On basis of the CAT study, the use of ICDs for primary prevention in DCM was no longer recommended as class IIa but as class IIb indication with level of evidence B in the Update of the European guidelines as published in 2003 (4). The AMIOVIRT, COMPANION, DEFINITE and SCD-Heft trials were, however, not taken into account. Apparent discrepancies between COMPANION and SCD-Heft still urge further analysis and discussion. Nonetheless, the weight of evidence of SCD-Heft and COMPANION will likely lead to a more liberal implantation policy in primary prevention in nonischemic

cardiomyopathy in the foreseeable future. We propose a class IIa indication with level of evidence B for nonischemic cardiomyopathy.

Recently published ESC and AHA guidelines for the diagnosis and treatment of chronic heart failure already may pre-empt future ESC ICD guidelines in so far that ICD implantation is already mentioned as a class I recommendation for primary prevention of SCD in selected patients with severely reduced LV ejection fraction (29;30). Unfortunately these guidelines did not specify which group of patients is being referred to.

Heart failure and ICD implantation

The COMPANION is as of yet the only study in which patients were randomized to either CRT or CRT-ICD, but no direct comparison between outcome in these groups was made. When compared to OPT, secondary endpoint (death from any cause) at 1 year was not significantly different for CRT only, whereas CRT-ICD reduced 1-year mortality significantly. This lack of beneficial effect of CRT only may have to be contributed to the premature termination and short follow-up in the COMPANION study. This gap was filled by the CARE-HF study, published in March 2005 (31). This study included 813 patients with severe heart failure (NYHA $\geq$ III, LV EF $\leq$ 35% and QRS > 120 with signs of ventricular dyssynchrony) randomized to either OPT or CRT without ICD. Follow-up was considerably longer than in the COMPANION study (2.5 years). Primary endpoint (composite of death from any cause or unplanned cardiovascular hospitalization) was significantly reduced in the CRT group when compared to OPT (39 vs. 55%). The secondary endpoint (death from any cause) was also significantly less in the CRT group (20% vs. 30%). These findings demonstrated unequivocally that CRT reduces the risk of death in patients with class III/IV heart failure. Since 33% of deaths were sudden in both groups, it is attractive to speculate that adding ICD therapy may reduce mortality even further.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a relatively common cardiac disorder (prevalence 1:500) with a relatively benign course in most patients but in which sudden, unexpected death may occur with a frequency of 1-4%, most commonly in the young patients (<30 yrs) (24). The evidence for ICD implantation in HCM is mainly based on retrospective studies, small prospective studies and the opinion of experts.

Generally accepted risk factors in HCM patients for SCD are: prior cardiac arrest, sustained VT, positive family history of SCD, extreme wall thickening (>30 mm), syncope (especially when recurrent and related to exertion), hypotensive blood pressure response to exercise (≥ 25 mmHg systolic blood pressure drop), nonsustained VT and specific malignant genotypes (32-34). Patients are considered to have a "positive" family history of SCD if either a first degree family member suffered from SCD < 40 years of age or multiple family members in different generations died suddenly of cardiac causes < 40 years of age (5). There has been no update on the European guidelines for ICD implantation since publication of the last Task Report of the ESC in 2001 (1). The 2003 AHA/ESC expert consensus document did not result in any change in ICD guidelines (35).

Because no randomized trials have been conducted with respect to secondary prevention in HCM patients, ICD implantation in these patients can be considered class I with level of evidence C. Primary prevention in HCM patients with 2 or more risk factors is strongly recommended, with a class IIa recommendation and level of evidence C (33). In patients with only one risk factor, the positive predictive accuracy for SCD is low and therapy must be individualized.

Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a cardiac disorder, which has a prevalence of 1:1,000-10,000. Autosomal dominant inheritance with incomplete penetrance is present in about 30% of cases. It is probably one of the main causes of SCD in the non-coronary artery disease group. Only limited information is available on risk assessment of SCD in ARVC. The evidence is based on small studies and on the opinion of experts.

SCD may occur more frequently in patients with extensive right ventricular changes and in those with LV involvement (recommendation IIa, level of evidence C) and after previous cardiac arrest or ventricular fibrillation (recommendation class I, level of evidence C) (36). ICD implantation in ARVC patients was associated with a relatively high implantation- and lead related complication rate in a recent study (36), but no further data are available on this subject. Inducibility by itself, adverse family history, syncope, ventricular tachycardia are of uncertain value in risk stratification for SCD (recommendation class IIb, level of evidence C) (37).

Long QT syndrome

This is a familial disease with a prevalence of 1: 5,000, manifesting itself primarily in children or teenagers. LQTS is associated with high risk of SCD. Risk stratification is based on descriptive studies, but include syncope, ventricular arrhythmias, QT interval duration and specific genetic defects (38).

Primary prevention is still mainly based on the use of beta-blockers (39-44). It is conceivable that defibrillator shocks because of the accompanying sympathetic drive may elicit subsequent arrhythmias and repeated ICD discharges. According to the current guidelines, there is in general no indication for ICD therapy as primary prevention. However, if symptomatic recurrences of ventricular arrhythmias occur despite adequate beta-blocker treatment, if serious doubt exists concerning therapy compliance (children or adolescents), ICD implantation should be considered (recommendation class IIa, level of evidence C) (45). Secondary prevention of SCD with ICD and beta-blockers has a class I recommendation (level of evidence C).

Brugada syndrome

Patients with the Brugada syndrome have the typical Brugada ECG in the absence of structural heart disease, associated with malignant ventricular arrhythmias and/or a family history of these arrhythmias or sudden death at young age. The typical Brugada ECG is characterized by right precordial ST segment elevation of ≥ 2 mm with coved ST-segment and negative T-wave (type I ECG). ST segment elevation of the saddle-back type represents type II and III ECG depending on the amount of ST elevation (46).

Risk stratification remains controversial, with conflicting data on the role of programmed electrical stimulation (PES). Data from Priori et al. indicate a very low incidence of arrhythmic events and no predictive value of PES in asymptomatic individuals with a Brugada ECG (47;48). Results of a study by Eckardt et al, with the longest follow-up to date (mean 40 months) of a cohort of individuals with type I Brugada ECG, also fail to identify positive PES as a risk factor for sudden death and demonstrate <1% incidence in asymptomatic individuals (49). In contrast, Brugada et al recently reported an 8% occurrence rate of (aborted) sudden cardiac death in asymptomatic individuals with a type I ECG over only 2 years follow-up, in addition to predictive value of PES in identifying individuals at risk (50;51). The reason for the discrepancy between Brugada's data and others may lie in

selection bias, including patients with a more malignant genotype. The higher incidence of a family history of sudden cardiac death in the Brugada registry and higher incidence of events during follow-up are in accordance with this hypothesis.

At this time, consensus is that patients with a type I ECG and symptoms (syncope or documented ventricular arrhythmia) should receive an ICD (class I, level of evidence B) (46). The indication for asymptomatic patients with positive EPS remains controversial (class IIb, level of evidence B). Longer follow-up and larger studies are awaited. In addition, a recent report by Belhassen on beneficial effects of quinidine in high risk patients (52) invites further discussion on the role of drug therapy in this patient population.

Catecholaminergic polymorphic ventricular tachycardia

This disease has an unknown prevalence and is characterized by adrenergically induced polymorphic ventricular tachycardia in the absence of structural cardiac abnormalities. The usual presentation is syncope and patients have a positive family history of syncope and SCD in approximately one third of cases. There is evidence for an autosomal dominant inheritance in which the ryanodine receptor pathway is implicated (53).

The arrhythmias, VTs with bidirectional QRS morphology, are reproducibly induced during exercise stress testing or during isoproterenol infusion at heart rates above 120 bpm (54). Inducibility with PES is variable. Since no large studies are available, reliable data on risk stratification are missing and all recommendations are based on expert opinion (level of evidence C). The ICD has a role in secondary prevention. Primary prevention of patients with early onset of symptoms of syncope, ventricular tachycardia at Holter recording or positive family history is still primarily based on beta-blockers, whereas the use of ICDs in this patient group is unsure except for those patients with hemodynamically not tolerated VT or VF. Since episodes of polymorphic VT are catecholamine dependent, there is a potential risk of repeated ICD shocks elicited by post-shock adrenergic drive.

Miscellaneous cardiac abnormalities and cardiac arrest

SCD has been described in aortic valve stenosis, mitral valve prolapse, WPW syndrome, myocardial bridging, and anomalous origin of the coronary arteries, infiltrative disorders (such as sarcoidosis with cardiac involvement, amyloidosis and Gaucher disease), neuromuscular disorders and lamin A/C deficiency. Attention is focused on treatment of the underlying disease. ICD implantation plays a role only in secondary prevention.

Arrhythmias in children and (adult) congenital heart disease

The risk of SCD in children is low and mainly concerns congenital heart disease or cardiomyopathy. A high incidence of SCD has been described in patients with surgery for aortic stenosis, transposition of the great arteries, tetralogy of Fallot. Risk factors have been described especially in the last group (55). No new data have become available since publication of the guidelines in 2001 (5).

Extended indications for ICD implantation

The currently presented update on the guidelines covers the indications for implantation of ICD for prevention of SCD. However, ICDs have evolved importantly in the last decade and the current ICD generation has advanced features for the diagnosis, detection and management of well-tolerated ventricular tachycardia. Ventricular antitachycardia pacing either or not in combination with anti-arrhythmic drugs may provide a patient-friendly way of curtailing recurrent ventricular tachycardia, thereby preventing hospitalizations (56).

Table 1. Indications and strength of evidence

Indications
Class I. Conditions for which there is evidence and/or general agreement that a given procedure (or risk stratification parameter) is useful and effective
Class II. Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the procedure or treatment (or risk stratification parameter). IIa. Weight of evidence/opinion in favour of usefulness/efficacy. IIb. Usefulness/efficacy is less well established by evidence/opinion.
Class III. Conditions for which there is evidence or general agreement that the procedure/treatment is not useful/effective.
Strength of evidence
Level of evidence A = data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B = data derived form a single randomized trials or non-randomized studies
Level of evidence C = observational data or consensus opinion of the experts

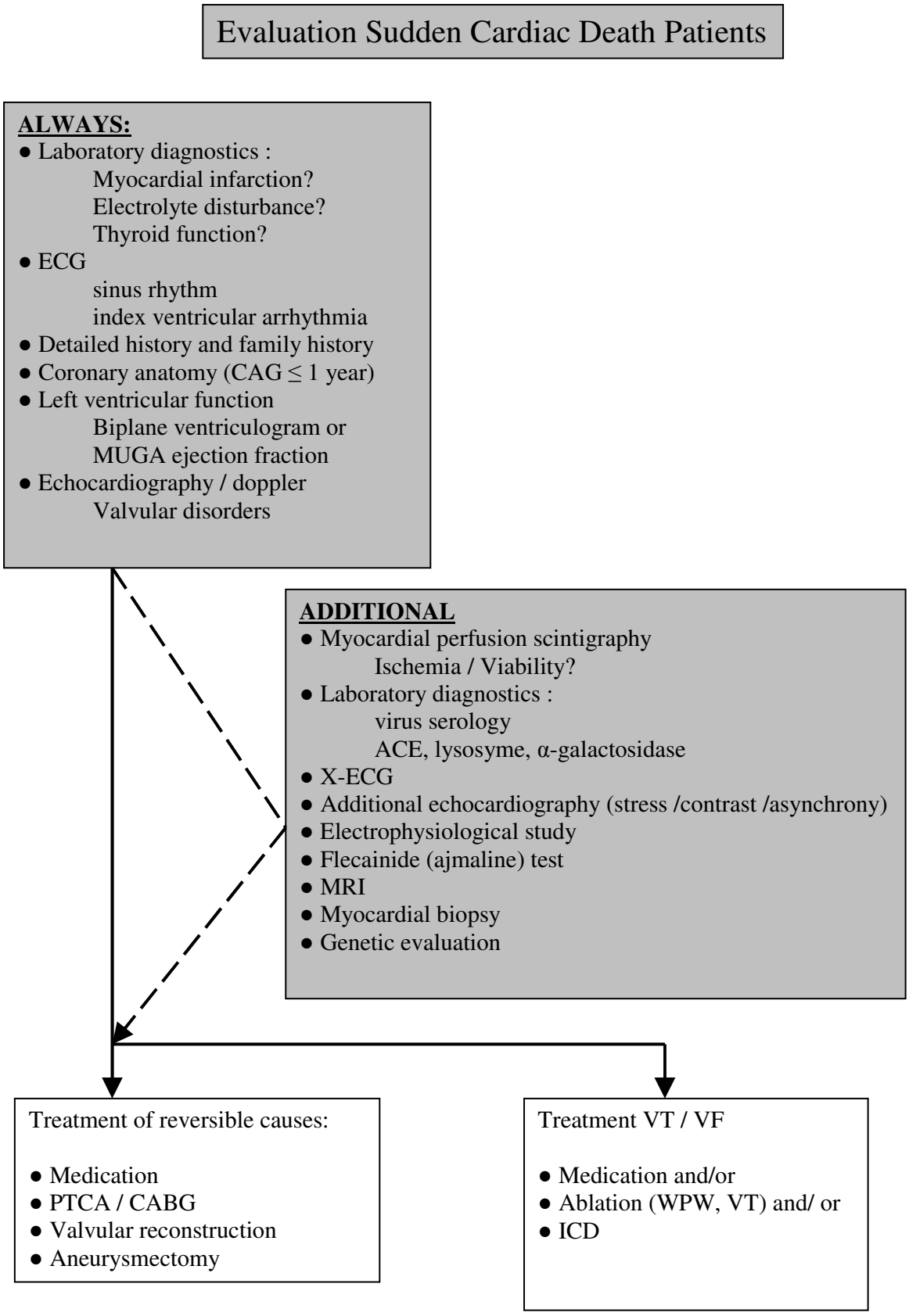

Figure 1. Evaluation of sudden cardiac death patient. PTCA=Percutaneous Transluminal Angioplasty; CABG=Coronary Artery Bypass Grafting; ICD=Implantable Cardioverter/Defibrillator; WPW=Wolff-Parkinson-White Syndrome; VT=Ventricular

Tachycardia; VF=Ventricular Fibrillation; CAG=Coronary Angiography; MRI=Magnetic Resonance Imaging

	Primary prevention		Secondary prevention			
Ischemic cardiomyopathy						
EF ≤ 30%, > 40 days after MI	Class IIa	A				(18;20)
EF ≤ 40% + spont. nsVT (> 3 weeks post MI)	Class IIa	C				(8;9)
Resuscitated VT/VF, spont. hemodyn. non-tolerated sVT			Class I	A		(15;57)
Spont. well tolerated monomorphic VT (EF > 40%)	Class IIb	B				(58)
Dilated cardiomyopathy						
EF < 35%, NYHA III and IV	Class IIa	B				(20;21)
Resuscitated VT/VF, spont. hemodyn. non-tolerated sVT			Class I	C		(1)
Hypertrophic cardiomyopathy						
2 or more risk factors (see text)	Class IIa	C				(35)
Resuscitated VT/VF, spont. hemodyn. non-tolerated sVT			Class I	B		(35)
Arrhythmogenic right ventricular cardiomyopathy						
Extensive RV disease or LV involvement	Class IIa	C				(36)
Resuscitated VT/VF, spont. hemodyn. non-tolerated VT			Class I	C		(36)
Long QT syndrome						
Persistent ventricular arrhythmias / syncope despite beta-blockers	Class IIa	C				(45)
Resuscitated VT/VF, spont. hemodyn. non-tolerated VT			Class I	C		
Brugada syndrome						
Asymptomatic, with or without positive PES	Class IIb	B				(51)
Type I ECG, syncope or	Class I	B				(46)

documented ventricular arrhythmia						
Resuscitated VT/VF, spont. hemodyn. non-tolerated VT,			Class I	B		(46)

Table 2. Indication summary. EF=Ejection fraction; nsVT=non-sustained VT; PES=Programmed Electrical Stimulation;

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